Published in final edited form as:

Clin Psychol Sci. 2019 September ; 7(5): 914–927. doi:10.1177/2167702619834576.

Positive Affect as a Buffer between Chronic Stress and Symptom Severity of Emotional Disorders

Amy R. Sewart, M.A.^a, Tomislav D. Zbozinek, Ph.D.^b, Constance Hammen, Ph.D., Richard E. Zinbarg, Ph.D.^c, Susan Mineka, Ph.D.^c, Michelle G. Craske, Ph.D.^a

^aUniversity of California, Los Angeles, Department of Psychology, 1285 Franz Hall, Los Angeles, CA 90025, USA

^bCalifornia Institute of Technology, Humanities and Social Sciences, 1200 E. California Blvd., MC 228-77, Pasadena, CA 91125, USA

^cNorthwestern University, Department of Psychology, 2029 Sheridan Road, Swift Hall 102, Evanston, IL 60208, USA

Abstract

Research has demonstrated that stressors play a critical role in the development of generalized anxiety disorder (GAD), social anxiety disorder (SAD), and major depressive disorder (MDD). Separately, deficits in positive affect (PA) have been identified in GAD, SAD, and MDD. While previous research has linked the buffering effects of PA in chronic illness, such effects have yet to be investigated for chronic stressors and emotional disorder-related symptom severity. The purpose of the present study was to examine PA as a moderator of chronic interpersonal and non-interpersonal stress on GAD, SAD, and MDD symptom severity. Using a multilevel statistical approach with a sample of adolescents and young adults ($N=463$), PA was found to significantly moderate the relationship between chronic interpersonal stress and symptom severity for MDD and SAD. Findings suggest that in times of chronic interpersonal stress, higher PA may serve as a buffer from development of SAD and MDD symptoms.

Keywords

social anxiety; generalized anxiety; depression; positive affect; chronic stress

Introduction

Stress has long been implicated as a factor in the development of mental health disorders and is predictive of poorer psychological well-being (McGonagle & Kessler, 1990; Monroe & Simons, 1991; Pynoos, Steinberg, & Piacentini, 1999). Based on its time course, stress can be categorized into either acute (episodic) or chronic (ongoing) stress (Hammen, Kim, Eberhart, & Brennan, 2009). While acute stressful life events possess relatively distinct onset and offset, chronic stress captures the “enduring aspects of the social and/or physical

environment which involve deprivation or disadvantage and create a continuous stream of threats and challenges for the individual” (Compas 1987, p. 276) and often slowly develops as a continuing, problematic condition (Wheaton, 1997). Chronic stress is typically categorized as interpersonal or non-interpersonal in nature (Connolly, Eberhart, Hammen, & Brennan, 2010; Hammen, 1991; Hammen et al., 1987). Chronic interpersonal stress arises from sustained relationship discord – wherein a given relationship may be conflictual, untrustworthy, or nonreciprocal – or, alternatively, from the absence of close, confiding relationships (Shih, Eberhart, Hammen, & Brennan, 2006). In contrast, stressors not generated from relationship dynamics are considered non-interpersonal stressors (Shih, Eberhart, Hammen, & Brennan, 2006). Chronic non-interpersonal stressors take a variety of forms, including enduring financial strain, academic or job-related difficulties, neighborhood crime, and poor health (Baum, Garofalo, & Yali, 1999; Dohrenwend et al., 1992; Rudolph et al., 2000; Sheets & Craighead, 2014).

Both acute and chronic stress have been implicated in the development of anxiety and depressive disorders (Arborelius, Owens, Plotsky, & Nemeroff, 1999; Baran, Armstrong, Niren, Hanna, & Conrad, 2009; Faravelli et al., 2012; Hammen, 1991; Heim, Newport, Mletzko, Miller, & Nemeroff, 2008; McWilliams, Cox, & Enns, 2003; Phillips, Hammen, Brennan, Najman, & Bor, 2005; Vyas, Pillai, & Chattarji, 2004). Disorder-specific research has shown that both acute and chronic stress contribute to the onset of generalized anxiety disorder (GAD; Blazer, Hughes, & George, 1987; Kendler, Hettema, Butera, Gardner, & Prescott, 2003; Kessler et al., 2008; Moffitt et al., 2007). For example, in adults (ages 18-44 years), experiencing one or more stressful life events in the past year was found to increase the risk threefold of developing GAD (Blazer, Hughes, & George, 1987). These stressful life events were both interpersonal and non-interpersonal (e.g., personal health, legal matters involving self or family, change in residence, work or marital status), and were coded as “stressful life events” if they were unexpected, negative, and very important. A second study examining adults (ages 18-55 years) showed that experiencing more acute stressors, particularly threat-related (e.g., potential future loss, including potential trauma or threatening/dire outcomes) or loss-related stressors (i.e., reduced sense of connectedness/well-being potentially covering every aspect of life, such as loss of person, material possessions, or health), significantly predicted GAD onset (Kendler, et al., 2003). Furthermore, stressful life events during childhood have also been shown to predict GAD onset (Beesdo, Pine, Lieb, & Wittchen, 2010; Kessler et al., 2008; Moffitt et al., 2007). For example, Kessler, et al. (2008) found that childhood adversity – measured as maltreatment (neglect, physical abuse, sexual abuse) and loss (parental death, parental divorce, other long-term separation) – significantly predicted GAD (age 15-24; Kessler, Davis, and Kendler, 1997). In total, the research seems to suggest that both acute and chronic stress may predict GAD.

Furthermore, the literature suggests that chronic interpersonal stress is associated with social anxiety disorder (SAD; Davila & Beck, 2002; Wittchen, Stein, & Kessler, 1999), though the data is scarcer than that for GAD. For example, Davila and Beck (2002) found that chronic interpersonal stress in the previous six months with close relationships (i.e., friendships, social life, romantic relationships, family relationships, and independence from one’s family) was significantly associated with greater SAD symptomatology for young adults

($M_{age}=18.72$). Similarly, Wittchen, et al. (1999) found that parental separation during childhood or early adolescence was an interpersonal risk factor associated with SAD (ages 14-24 years). These findings are complemented by an experimental animal study investigating chronic interpersonal versus non-interpersonal stress on subsequent anxiety (Barsy, Leveleki, Zelena, & Haller, 2010). The results showed that rodents who were administered chronic psychosocial stressors (i.e., repeated conflict with other rats) but not non-social stressors (i.e., physical restraint) exhibited increased anxiety in a subsequent social interaction test, suggesting that chronic interpersonal stress may increase social anxiety. Thus, in total, it seems that chronic interpersonal stress may be a risk factor for developing SAD.

Finally, both chronic and acute interpersonal and non-interpersonal stress are well-established risk factors for major depressive disorder (MDD; e.g., Hammen, 1991, 2003; Hammen, et al., 2004, 2009; Kessler, 1997; Kessler, et al., 2008; Mazure, 1998; Shih, et al., 2006). For example, Shih, et al. (2006) found that both male and female 15-year-olds experienced increased depressive severity as a result of acute and chronic interpersonal and non-interpersonal stress. In adults, Hammen, et al. (2009) investigated the relationship between chronic stress, acute stress, and major depressive episode onset in women ($M_{age}=41$). Results from this study revealed that chronic stress was positively associated with the occurrence of acute stressors and that major depressive episode onset was positively associated with both chronic and acute stressors. These results were collapsed across both interpersonal and non-interpersonal stress. Thus, it seems that depression is associated with interpersonal, non-interpersonal, acute, and chronic stress.

Positive affect may facilitate resilience in the presence of stressors and reduce vulnerability to mental health disorders. Resilience is defined as the ability of individuals to adapt when faced with acute stress, trauma, or chronic adversity and maintain or rapidly regain psychological well-being (Carver, 1998; Charney, 2004; Fredrickson, 1998; Lazarus, 1993; Tugade & Fredrickson, 2007). Positive affect refers to one's pleasurable engagement with the environment and includes positive emotional states, such as happiness, joy, interest, excitement, confidence, and alertness (Watson & Clark, 1991). Positive affect has been shown to promote flexibility in thinking and problem solving (Billings, Folkman, Acree, & Moskowitz, 2000; Folkman & Moskowitz, 2000b; Fredrickson & Branigan, 2005; Isen, Daubman, & Nowicki, 1987), reduce the physiological effects of negative emotions (Fredrickson, 2004; Fredrickson, Mancuso, Branigan, & Tugade, 2000; Fredrickson & Levenson, 1998; Ong, Bergeman, Bisconti, & Wallace, 2006), build enduring social resources (Keltner & Bonanno, 1997), promote effective coping strategies (Affleck & Tennen, 1996; Folkman & Moskowitz, 2000a, 2000b; Folkman, Moskowitz, Ozer, & Park, 1997; Ong, Bergeman, & Bisconti, 2004; Ong et al., 2006), and create upward spirals of improved emotional well-being (Fredrickson & Joiner, 2002).

The "dynamic model of affect" (Zautra, Smith, Affleck, & Tennen, 2001) predicts that positive and negative affect are relatively independent under ordinary circumstances but may become inversely related during stress. In support, high (but not low) positive affect significantly lowered the effects of chronic health-related stress on negative affect (Zautra et al., 2001). High positive affect also reduced the deleterious effects of pain on negative affect

significantly more during times of high versus low pain (Zautra et al., 2001). Similarly, Zautra, Johnson, and Davis (2005) found that, in a sample of females experiencing chronic health-related stress and pain from osteoarthritis and/or fibromyalgia, higher weekly levels of pain and stress predicted higher negative affect. However, higher weekly levels of positive affect and average overall positive affect resulted in less negative affect, and this association was stronger during weeks of high pain. In addition, individuals with greater positive affect were less likely to demonstrate higher negative affect during weeks of interpersonal conflict when compared to those with low positive affect – though these interpersonal stressors were more acute in nature (e.g., argued with spouse/partner). Similar patterns were reported by Strand et al. (2006), where, during times of pain, individuals high in positive affect experienced less negative affect. Moreover, Tugade and Fredrickson (2007) found that cultivating positive affect promoted resilience through automatic activation of positive emotions during stress. Taken together, these findings suggest that positive affect buffers chronic interpersonal and non-interpersonal stress through reductions of negative affect.

The role of positive affect is especially relevant given evidence that low levels of positive affect are characteristic of depression (Watson & Clark, 1991) and anxiety disorders (Amies, Gelder, & Shaw, 1983; Brown, Chorpita, & Barlow, 1998; Chorpita, Plummer, & Moffitt, 2000; Kashdan, 2007; Watson, Clark, & Carey, 1988). Significant portions of factorial variance in symptoms of anxiety as well as depression are explained by positive affect (Kashdan, 2007; Prenoveau, Zinbarg, Craske, Mineka, Griffith & Epstein, 2010). Effect sizes for cross-sectional and longitudinal relationships between positive affect and anxiety are significant and indistinguishable from corresponding effect sizes for positive affect and depression (Kotov, Gamez, Schmidt & Watson, 2010; Khazanov & Ruscio, 2016). Of the anxiety disorders, the evidence for deficits in positive affect is most conclusive for SAD. Using structural equation modeling, Brown, Chorpita, and Barlow (1998) found no evidence for differential influence of positive affect while examining the paths of positive affect on SAD ($-.28$) and of positive affect on depression ($-.29$) after covarying for negative affect. Converging with these findings, a meta-analysis examining 19 studies which reported either correlations or group difference tests of positive affect and social anxiety also found a stable, moderate, inverse relationship ($r = -.36$; 95% CI: $-.31$ to $-.40$; Kashdan, 2007). Notably, this relationship remained consistent after covarying for depressive symptoms and disorders, which are commonly attributed to low positive affect and often comorbid with SAD ($r = -.21$; 95% CI: $-.16$ to $-.26$). Individuals with GAD also report low positive affect (Chambers, Power, & Durham, 2004; Power & Tarsia, 2007). In addition, an “anxious-misery” factor comprised of positive affect and anhedonic depression items from the Mood and Anxiety Symptom Questionnaire (MASQ; Keogh & Reidy, 2000; Watson & Clark, 1991) and Inventory to Diagnose Depression (IDD; Zimmerman & Coryell, 1987) was found to be associated with GAD, SAD, and MDD severity in a late adolescence to emerging adulthood sample (Prenoveau et al., 2010).

Given the relationship between a) positive affect and SAD, GAD, and MDD and b) positive affect and chronic stress, it is plausible that higher levels of positive affect may mitigate the effects of chronic stress upon these disorders. The goals of the present study were to investigate the role of chronic interpersonal and non-interpersonal stress in the development of SAD, GAD, and MDD symptom distress and impairment (i.e., symptom severity) and to

examine whether positive affect moderated this relationship. The present study uses data from the Youth Emotion Project (e.g., Zinbarg et al., 2010), a longitudinal study aimed at assessing the predictors of the development of mood and anxiety disorders (e.g., neuroticism) from adolescence into young adulthood.

We hypothesized that chronic interpersonal stress would predict increased distress (e.g., relationship discord) and impairment relating to SAD and MDD symptom severity. Furthermore, we hypothesized that chronic non-interpersonal stress (e.g., financial, health, school/work, etc.) would predict increased distress and impairment relating to GAD and MDD symptom severity. Positive affect is associated with greater resilience during stress (Tugade & Fredrickson, 2007), and SAD, GAD, and MDD are associated with lower positive affect (e.g., Chambers, Power, & Durham, 2004; Kashdan, 2007). Thus, we hypothesized an interaction between positive affect and chronic interpersonal stress such that higher positive affect would moderate the effects of stress to predict decreased severity of SAD and MDD. Similarly, we hypothesized an interaction between positive affect and chronic non-interpersonal stress such that higher positive affect would moderate the effects of stress to predict decreased severity of GAD and MDD. Lastly, to examine specificity of the effects of positive affect, we analyzed the effects of negative affect in moderating the relationship between a) chronic interpersonal and non-interpersonal stress and b) GAD, SAD, and MDD symptom severity.

Methods

Participants

Participants enrolled in a larger two-site, 8- to 10-year longitudinal study (total N = 627) who completed a chronic life stress interview, a structured interview for psychiatric disorders, and a self-report measurement of positive affect at successive time points during the study were eligible for inclusion in the analyses. Our multilevel statistical approach allowed us to include multiple observations nested within individuals (see Statistical Approach). The larger study examined risk factors for psychopathology in late adolescence and early adulthood (Youth Emotion Project, YEP; Zinbarg et al., 2010) in three consecutive cohorts of high school juniors from two demographically diverse public high schools in suburban Chicago and suburban Los Angeles. To increase the incidence of psychopathology onset during the longitudinal phase, at-risk students exhibiting high levels of neuroticism (i.e., top tertile) as measured by the Neuroticism subscale of the revised 23-item Eysenck Personality Questionnaire Neuroticism Scale (EPQ-R-N; Eysenck & Eysenck, 1975) were oversampled (Clark, Watson, & Mineka, 1994; Hayward, Killen, Kraemer, & Taylor, 2000). See Zinbarg et al. (2010) for details regarding sampling procedures. Given that risk for mania is associated with abnormal elevations in positive emotion (e.g., Gruber, Johnson, Oveis, & Keltner, 2008), participants who endorsed symptoms of Bipolar I, Bipolar II, or Cyclothymia during the structured interview for psychiatric disorders at any timepoint during the study were excluded from the present analyses (n=15). Using these criteria, 1898 observations nested within 463 individuals were eligible for inclusion in the analyses (*M*_{age} = 16.94). The resulting sample was 68.7% female and 49.0% White, 15.1% Hispanic/Latino, 13.8% African-American, 4.5% Asian, 0.9% Native American/Pacific Islander/Alaskan

Native, 12.1% Multiracial, and 4.5% who identified with another unlisted racial/ethnic group. Based on Hollingshead socioeconomic status scores, the sample on average was upper middle class ($M = 48.96$, $SD = 12.37$; Hollingshead, 1975).

Measures

Chronic interpersonal and non-interpersonal stress.—An annual evaluation of ongoing, typical chronic life stress was conducted using the UCLA Life Stress Interview (LSI; Hammen, 1991; Hammen et al., 1987), a semi-structured interview of ongoing, typical conditions in ten life domains. Each domain of the LSI was rated by trained interviewers on objective circumstances, indicating severity of chronic stress in the given role domain on a 5-point scale ranging from 1 to 5, using half-point increments (1 = minimal stress, 5 = very stressful circumstances). Per interview protocol, raters were trained to consider scores of 1 and 5 to be rare and relatively extreme cases and used general probes to elicit relevant, objective information from participants. To assess chronic interpersonal and non-interpersonal stress, role domains of 1) close friendship, 2) social life, 3) intimate (romantic) relationships, and 4) relationships with family members were categorized as interpersonal, while the domains of 5) neighborhood environment, 6) academic performance, 7) work environment, 8) financial status, 9) personal health, and 10) health of the individual's family were considered non-interpersonal. For example, when evaluating the 'close friendship' role domain raters assessed the degree to which participants felt trust, stability, closeness, reciprocity, conflict within their closest friendship. For this domain, a rating of 1 would indicate a close friendship posing little chronic stress and that the relationship is close, confiding, mutually satisfying, and stable with good conflict resolution, whereas a rating of 5 would indicate an absence of a close, confiding friendship or a highly conflictual relationship (Shih, Eberhart, Hammen, & Brennan, 2006). Similar probes and criteria were used to evaluate other domains. Composite scores for chronic interpersonal and non-interpersonal stress used for analyses were calculated by averaging category-relevant domain scores. To determine baseline reliability of chronic life stress in the present study, intraclass correlation coefficients (ICCs) were calculated using 76 intersite and intrasite rated audiotaped interviews. The ICCs ranged from 0.57 to 0.91 for each domain, and averaged 0.73 across interpersonal and non-interpersonal domains (Doane et al., 2013).

Positive and negative affect.—Positive affect was measured using the Mood and Anxiety Symptom Questionnaire (MASQ; Watson & Clark, 1991), a self-report measure in which participants are asked rate the strength of emotional experiences over the past week using a 5-point Likert scale ranging from 1 (not at all) to 5 (extremely). Using factor analysis, high positive affect items were found to possess higher loadings ($b = 0.73$ - 0.78) on the general positive affect factor than low positive affect items ($b = 0.42$ - 0.46) (see Kendall et al., 2016). This may be due to unintended contamination of negative affect into items constructed to assess low positive affect on this measure (e.g., "Thought of death or suicide") (Kendall et al., 2016). In addition, high positive affect items are considered to be more robust markers of the underlying positive affect factor (see Watson et al., 1988). As a result, we chose the mean of the 14 high positive affect items (e.g., "Felt optimistic", "Felt like I had a lot of interesting things to do") from the Anhedonic Depression subscale of the MASQ (MASQ-AD) to represent positive affect. Using the current sample of adolescents,

the stability of the MASQ-AD was investigated over three years through a series of trait-state-occasion (TSO) models (see Kendall et al., 2016). A trait-only model was found to best represent the data, suggesting there was a significant trait component in positive affect as measured by the MASQ-AD. Furthermore, more than three quarters of the variance (77%) of the MASQ-AD, on average, was found to be stable and attributable to the trait component. As a result, while this measure was designed to assess time-variant experiences, we felt it was appropriate for this measure to represent a more stable measure of positive affect. Negative affect was measured by using the mean score of 15 disorder nonspecific items from the General Distress Mixed subscale (MASQ-GDM) which capture general negative affectivity (e.g., “Felt irritable”) also featured on the MASQ (Watson & Clark, 1991). We included negative affect in our models to 1) appropriately account for negative affect’s influence on disorder-related symptom severity (e.g., Clark, Watson, & Mineka, 1994; Watson & Clark, 1984) and 2) to demonstrate the unique moderating effects of positive affect for chronic interpersonal and non-interpersonal stress on disorder-related symptom severity.

Social anxiety, generalized anxiety, and major depressive disorder symptom severity.—The Structured Clinical Interview for the DSM-IV, non-patient edition (SCID-I/NP; Spitzer, Robert, Gibbon, & Williams, 2002) was administered to assess for Axis I DSM-IV disorders. Participants were administered the SCID-I/NP on an annual basis to evaluate diagnostic symptomatology since the prior interview (see Zinbarg et al., 2010, for details). After administering screening questions to determine which diagnostic sections required further inquiry, interviewers administered endorsed diagnostic sections and rated the clinical severity of each current diagnosis using the 0 to 8 Clinical Severity Rating (CSR) Scale (Di Nardo & Barlow, 1988). A CSR score of 4 or higher for a diagnosis is indicative of clinically significant, distressing or impairing symptoms. All participants received a CSR rating for all possible Axis I diagnoses. Thus, CSR was used as an index for symptom severity for SAD, GAD, and MDD. Inter-rater reliability (Cohen’s κ ; Cohen, 1960) for the present study was found to be acceptable to good for SAD ($\kappa = .65$), GAD ($\kappa = .85$), and MDD ($\kappa = .83$) diagnoses (see Uliaszek et al., 2009 for details). As aforementioned, participants endorsing symptoms of Bipolar I, Bipolar II, or Mania on the SCID-I/NP at any time point were excluded from analyses.

Statistical Approach

Hierarchical multilevel modeling was used to examine the effects of positive affect, chronic interpersonal stress, and chronic non-interpersonal stress on SAD, GAD, and MDD symptom severity. Analyses were performed in HLM 7.0 (Raudenbush, Bryk, Cheong, Congdon, & Du Toit, 2011). Multilevel modeling accounts for nonindependence of repeated measures within individuals and is a superior statistical approach for data with nested sources of variability. Thus, utilizing a multilevel approach allowed us to include multiple observations nested within individuals while remaining robust against violations of homoscedasticity and sphericity (Quené & Van den Bergh, 2004).

Our model included measurements of positive and negative affect at time point_{*t*}, chronic interpersonal and non-interpersonal stress at time point_{*t*+1}, and disorder symptom severity

at time point $t+2$. Each of these time points was separated by approximately one year. This was done to establish temporal precedence and to assess direction of effects. Establishing temporal precedence made certain that 1) any effects of positive or negative affect occurred on subsequent chronic stress and 2) chronic stress was experienced before the occurrence of disorder symptoms.

Covarying for previous symptom severity time point (i.e., lagged symptom severity) was not included in these present analyses due to threats of violation of the assumption of independence of random effects and covariates in mixed models. The hierarchical linear models in the current study include a random intercept which represents the combined effect on y of all unobserved variables that do not change over time. Because the model applies to all time points, the random intercept effect would likely include effects of baseline differences on the outcome that would covary with an observed lagged symptom severity variable. This violation of this independence can bias both the coefficient for the lagged dependent variable and the coefficients for other variables. The present hierarchical linear models control for person-level effects, which in part controls for previous time point symptom severity. Given that the majority of individuals had a disorder symptom severity of '0', or no disorder symptom severity, at any given time point our outcome variables of interest possessed zero-inflated distributions. To appropriately account for this distribution, multilevel Poisson regressions were performed for our moderator analyses. Models were examined separately for interpersonal chronic stress and non-interpersonal stress as predictors due to avoid issues relating to multicollinearity of the two variables. SAD, GAD, and MDD symptom severity were examined separately as outcomes using the following model:

Level-1 Model

$$\begin{aligned} E(\text{Symptom Severity}_{t+2(i)} | \pi_i) &= \lambda_{ti} \\ \log[\lambda_{ti}] &= \eta_{ti} \\ \eta_{ti} &= \beta_{0i} + \beta_{1i}(\text{Positive Affect}_{t(ij)}) + \\ &\quad \beta_{2i}(\text{Negative Affect}_{t(ij)}) + \\ &\quad \beta_{3i}(\text{Chronic Stress}_{t+1(ij)}) + \\ &\quad \beta_{4i}(\text{Positive Affect}_{t(ij)} * \text{Negative Affect}_{t(ij)}) + \\ &\quad B_{5i}(\text{Positive Affect}_{t(ij)} * \text{Chronic Stress}_{t+1(ij)}) + \\ &\quad B_{6i}((\text{Negative Affect}_{t(ij)} * \text{Chronic Stress}_{t+1(ij)})) \end{aligned}$$

Level-2 Model

$$\begin{aligned} \beta_{0j} &= \gamma_{00} + u_{0j} \\ \beta_{1j} &= \gamma_{10} + u_{1j} \\ \beta_{2j} &= \gamma_{20} + u_{2j} \\ \beta_{3j} &= \gamma_{30} + u_{3j} \\ \beta_{4j} &= \gamma_{40} + u_{4j} \\ \beta_{5j} &= \gamma_{50} + u_{5j} \\ \beta_{6j} &= \gamma_{60} + u_{6j} \end{aligned}$$

Results

Descriptive Statistics

Descriptive statistics for all variables of interest are presented in Table 1. The overall mean of chronic interpersonal stress across all time points indicated participants on average experienced mild to moderate levels of chronic interpersonal ($M = 2.29$, $SD = 0.42$) and non-interpersonal stress ($M = 2.23$, $SD = 0.33$). Mean scores on the modified MASQ-GDM ($M = 1.90$, $SD = 0.67$) and MASQ-AD ($M = 2.89$, $SD = 0.83$) were consistent with previously collected undergraduate samples (e.g., Watson et al., 1995). Rates of clinically significant GAD (1.8%; $M = 0.14$, $SD = 0.72$), SAD (3.3%; $M = 0.30$, $SD = 0.95$), and MDD (2.8%; $M = 0.16$, $SD = 0.82$) – indicated by a CSR of 4 or higher – corresponded with prevalence rates found in large-scale adolescent and young adult samples (see Beesdo, Knappe, & Pine, 2009).

Bivariate between-person correlations are presented in Table 2. Chronic interpersonal stress was significantly correlated with SAD ($r = .092$, $p < .01$), GAD ($r = .101$, $p < .01$) and MDD symptom severity ($r = .104$, $p < .01$). Similarly, chronic non-interpersonal stress was significantly correlated with SAD ($r = .046$, $p < .05$), GAD ($r = .122$, $p < .01$) and MDD symptom severity ($r = .107$, $p < .01$). Positive affect was significantly negatively correlated with SAD ($r = -.074$, $p < .01$) and MDD symptom severity ($r = -.060$, $p < .01$) but not with GAD symptom severity ($r = -.041$, $p = .071$). Furthermore, SAD, GAD, and MDD symptom severity were all positively significantly positively correlated with each other. SAD and GAD ($r = .205$, $p < .01$), SAD and MDD ($r = .144$, $p < .01$) GAD and MDD ($r = .211$, $p < .01$). Finally, negative affect was significantly positively correlated with SAD ($r = .118$, $p < .01$), GAD ($r = .127$, $p < .01$), and MDD symptom severity ($r = .133$, $p < .01$).

Symptom Severity, Chronic Interpersonal Stress, and Positive Affect

Main effects of chronic interpersonal and noninterpersonal stress on symptom severity.—To establish whether chronic interpersonal and non-interpersonal stress were predictors of future symptom severity of SAD, GAD, and MDD, we first analyzed the main effects of chronic interpersonal and non-interpersonal stress on disorder-related symptom severity ratings using separate Poisson regression models. Chronic interpersonal stress was a significant predictor of future SAD ($b = .516$, $SE = .123$, $p < .001$), GAD ($b = .479$, $SE = .161$, $p = .003$), and MDD symptom severity ($b = .344$, $SE = .153$, $p = .029$). Chronic non-interpersonal stress was a significant predictor of future GAD ($b = 1.161$, $SE = .346$, $p < .001$) and MDD symptom severity ($b = .719$, $SE = .189$, $p < .001$), but not SAD symptom severity ($b = -.004$, $SE = .239$, $p = .985$).

Moderating effects of positive affect for chronic interpersonal stress on symptom severity.—To examine whether positive affect dampened the effect of chronic interpersonal stress on future SAD, GAD, and MDD symptom severity, we analyzed the main effects of positive affect, negative affect, chronic interpersonal stress, and their two-way interactions using separate multilevel Poisson regression models (Table 3). Supporting our hypothesis, positive affect significantly moderated the effects of chronic interpersonal stress on future SAD symptom severity ($b = -.148$, $SE = .058$, $p = .011$) and MDD symptom

severity ($b = -.657$, $SE = .242$, $p = .007$), but not GAD symptom severity ($b = -.080$, $SE = .081$, $p = .324$). Given the chronological nature of our analyses – in which positive affect preceded chronic stress, which preceded disorder symptom severity – this suggests that increased positive affect reduced the effect of chronic interpersonal stress on SAD and MDD symptom severity, resulting in lower SAD¹ and MDD severity. Simple slopes tests were subsequently conducted to determine at which level positive affect moderates the effects of chronic interpersonal stress on SAD and MDD symptom severity (see Aiken & West, 1991). Poisson analyses at one standard deviation above (i.e., high positive affect) and below (i.e., low positive affect) the mean were executed by mean-shifting the data. For both SAD and MDD symptom severity, the effect of chronic interpersonal stress on symptom severity was significant when positive affect was low (SAD $b = .38$, $SE = .20$, $p = .04$; MDD $b = .32$, $SE = .12$, $p = .01$) but insignificant when positive affect was high (SAD $b = -.10$, $SE = .14$, $p = .46$; MDD $b = -.33$, $SE = .25$, $p = .19$). Thus, it appears positive affect possesses buffering capabilities beginning at high levels for SAD and MDD symptom severity. There were no significant results of negative affect moderating the effects of chronic interpersonal stress on SAD, GAD, or MDD (p s > .170). Thus, overall, positive affect buffered the effects of chronic interpersonal stress on symptom severity of SAD and MDD but not GAD; additionally, negative affect did not significantly moderate with the effects of chronic interpersonal stress in relation to symptom severity with SAD, GAD, and MDD.

Moderating effects of positive affect for chronic non-interpersonal stress on symptom severity.—To examine whether positive affect dampened the effects of chronic non-interpersonal stress on future SAD, GAD, and MDD symptom severity, we paralleled the analyses conducted above (Table 4). Positive affect did not moderate the effects of chronic non-interpersonal stress with SAD, GAD, or MDD (p s > .231). However, negative affect significantly and positively moderated the effects of chronic non-interpersonal stress on future MDD symptom severity ($b = .778$, $SE = .242$, $p = .001$). As negative affect increased, the effect of chronic non-interpersonal stress on distress and impairment of MDD symptoms was amplified. Thus, overall, positive affect did not significantly moderate the effects of non-interpersonal stress on symptom severity of SAD, GAD, and MDD. However, negative affect enhanced the effects of non-interpersonal stress on MDD symptom severity, whereas no effects were observed with SAD and GAD symptom severity.

Discussion

Research has reliably demonstrated that stress plays a critical role in the development of GAD (e.g., Blazer et al., 1987), SAD (e.g., Rosellini, Rutter, Bourgeois, Emmert-Aronson, & Brown, 2013), and MDD (e.g., Hammen, 1991). In a separate line of research, deficits in positive affect have been identified in individuals with GAD (e.g., Power & Tarsia, 2007), SAD (e.g., Brown et al., 1998), and MDD (Brown et al., 1998). The present study serves to both bridge and extend these two bodies of research by examining chronic interpersonal and

¹To determine whether depressive symptoms better accounted for these results, we analyzed the main effects of depressive symptoms, measured by the Inventory to Diagnose Depression (IDD; Zimmerman & Coryell, 1987), chronic interpersonal stress, and their two-way interaction in a supplemental Poisson regression. The interaction between depressive symptoms and chronic interpersonal stress was nonsignificant in predicting future SAD symptom severity ($b = -.037$, $SE = .065$, $p = .564$).

non-interpersonal stress, largely unstudied dimensions of stress within the context of anxiety disorders, as predictors of SAD and GAD symptom severity and – in addition – MDD symptom severity. Furthermore, we examined the potential moderating role of positive affect on the effects of chronic interpersonal and non-interpersonal stress on SAD, GAD, and MDD symptom severity. Whereas previous research has linked the buffering effects of positive affect on negative affect in the context of chronic illness (e.g., Zautra, Johnson, & Davis, 2005), these findings have yet to be investigated for chronic stress and symptom severity of anxiety and depressive disorders. Thus, the present study was an initial investigation into the dynamics of positive affect and chronic stress for anxiety and depression symptom severity.

The present study had several main findings. First, chronic interpersonal stress had a significant main effect in predicting SAD, GAD, or MDD symptom severity. Chronic non-interpersonal stress had a significant main effect in predicting GAD and MDD – but not SAD – symptom severity. Most notably, higher positive affect significantly reduced the effects of chronic interpersonal stress on SAD and MDD – but not GAD – symptom severity. Negative affect did not significantly moderate the effects of chronic interpersonal stress on SAD, GAD, and MDD symptom severity, suggesting specificity of the effects of positive affect. In contrast to our chronic interpersonal stress findings, positive affect did not significantly moderate the effects of chronic non-interpersonal stress on SAD, GAD, and MDD symptom severity. Higher negative affect, however, enhanced the effects of chronic non-interpersonal stress on MDD symptom severity, but had no effects on SAD or GAD symptom severity.

We found that positive affect reduced the effects of chronic interpersonal stress on SAD and MDD symptom severity. This is the first study to implicate positive affect as a buffering agent between a) chronic interpersonal stress and b) SAD and MDD symptom severity. These results were not limited to those with a diagnosis of SAD or MDD, but applied to individuals with symptoms ranging from below clinical severity to extreme clinical severity. Conceivably, for SAD, relationship discord experienced during chronic interpersonal stress may give rise to increased fear of negative evaluation and rejection that may generalize to relationships broadly. These fears may then motivate SAD-related behaviors, such as avoidance of social interactions, and in turn reinforce social fears and increase symptom severity. Similarly, for MDD, relationship discord may give rise to depressed mood, anhedonia, behavioral inactivation, and various maladaptive cognitions (e.g., negative self-statements). Positive affect may protect individuals from these sorts of downstream effects and reduce SAD and MDD symptom severity. As previously noted, individuals with high positive affect have been found to build enduring social resources (Fredrickson & Branigan, 2001; Keltner & Bonanno, 1997). Thus, in times of chronic interpersonal stress, individuals with high positive affect may be more likely to cope partly due to support from their social network. From an associative and instrumental learning perspective, seeking support and feeling supported by others may provide positive social associations that inhibit negative, excitatory associations such as “if I ask for help, I will be rejected,” which are implicated in the development of SAD (see Sewart & Craske, in press). Thus, in times of chronic interpersonal stress, positive affect may provide motivation to seek out social support, which therein provides corrective inhibitory learning on socially-relevant feared outcomes.

Similarly, individuals high in positive affect may engage in social forms of behavioral activation, leading to reward-based learning and experiences (e.g., “if I ask for help, people will support me; if I interact with other people, I will enjoy myself”) and reduction in MDD symptom severity.

These results of the benefits of positive affect converge with recent research evaluating the effects of targeting positive versus negative affect in the treatment of anxiety and depression (Craske, Meuret, Ritz, Treanor, Dour & Rosenfield, in submission). In this study, participants were admitted based on elevated anxiety or depression and were randomized to receive treatment focused on either increasing positive affect or decreasing negative affect (see Craske et al., in submission for details). While each treatment included cognitive-behavioral components, the focus of each treatment differed. For example, in the behavioral component, the negative affect condition engaged in behaviors to disconfirm negative outcomes (e.g., exposures), whereas the positive affect condition engaged in behaviors to increase positive outcomes (e.g., pleasant events scheduling). Similarly, in the cognitive component, the negative affect condition engaged in cognitive restructuring to reduce negative cognitions (e.g., reduce perceived likelihood and magnitude of negative outcomes), whereas the positive affect condition engaged in cognitive restructuring to enhance positive cognitions (e.g., increase anticipation/motivation for future positive effects, increase perceived ownership of positive outcomes). The results showed that the positive affect treatment condition was superior to the negative affect treatment condition in terms of symptoms of depression and anxiety, and for participants higher on pre-treatment levels of stress-related symptoms.

Our results also showed that for individuals with higher negative affect, experiencing later chronic non-interpersonal stress resulted in increases in MDD symptom severity. Given the heterogeneity in non-interpersonal stressors, we are unable to specify which of the many types of non-interpersonal stressors predicted this effect (e.g., finances, health, school/job, neighborhood crime). This suggests that if individuals already have high negative affect, experiencing later chronic non-interpersonal stress will combine with negative affect to increase MDD symptom severity.

Regarding positive affect’s buffering effect on chronic interpersonal stress, a plausible explanation for this specificity lies in a theorized function of positive emotions. According to the ‘broaden-and-build’ theory, positive emotions not only give rise to exploratory and novel thoughts and actions - thus broadening one’s behavioral range (i.e., thought-action repertoire), but also encourage the strengthening of enduring personal resources and relationships (Fredrickson 1998, 2001; Fredrickson, 2004). Given that one of the primary functions of positive affect is theorized to urge individuals to build upon interpersonal relationships to optimize well-being (Fredrickson & Branigan, 2001; Keltner & Bonanno, 1997), positive affect and associated behaviors may, as a result, also serve to directly remediate the psychological consequences chronic interpersonal stress. Although our findings did not support positive affect as a buffer for non-interpersonal chronic stress, this domain still deserves further attention. Given that certain forms of chronic non-interpersonal stress (e.g., financial problems) are likely to be higher for those with low socioeconomic

status, future studies should examine if factors such as education or income may also play a predictive role in this relationship.

Furthermore, the present study found that positive and negative affect had an inverse interaction in predicting SAD, GAD, and MDD symptom severity within the chronic non-interpersonal stress model; a similar effect was observed in the chronic interpersonal stress model with GAD and trended with SAD. One interpretation of this finding is that higher positive affect lowers the effects of negative affect on later symptom severity. This is consistent with findings that positive affect reduces the physiological effects of negative emotions (e.g., Fredrickson, 2004; Fredrickson et al., 2000; Fredrickson & Levenson, 1998; Ong et al., 2006) and creates upward spirals of improved emotional well-being (Fredrickson & Joiner, 2002). Perhaps having high positive affect during times of negative affect protects individuals from later increasing SAD, GAD, or MDD symptom severity.

This study possesses several strengths. Rather than using a dichotomous outcome, the present study considered individuals presenting with a wide range of SAD, GAD, and MDD symptom severity spanning from non-existent to severely distressing and impairing symptomatology. Furthermore, rather than relying on questionnaires, we assessed both symptom severity and chronic stress using semi-structured interviews conducted by trained interviewers rather than relying upon self-report which can be biased by factors such as current mood state. In addition, we were able to establish temporal precedence between our three variables of interest, ensuring that observed disorder-related symptom severity did not precede chronic stress. Our variables were measured over a two-year period (see Statistical Approach), making a strong case for the durability of effects from positive/negative affect and chronic stress on SAD, GAD, and MDD symptom severity.

Additionally, our study possessed some limitations. Our measurement of positive affect was framed “over the past week.” While this specific questionnaire has demonstrated high stability over time (Kendall et al., 2016), future studies may benefit from using measures that are specifically created to capture trait versions of positive affect, such as the Positive and Negative Affect Schedule – Expanded Form (PANAS-X). Psychometric testing of the PANAS-X reflected consistently higher stability of both higher (e.g., positive affect) and lower-order (e.g., sadness) scale coefficients when items were queried using general instructions than when the same items were probed “over the past week” (Watson & Clark, 1999). Second, while a strength of our study was the use of clinical interview to measure symptom severity, our study would have benefited from using a multi-modal approach, including behavioral, physiological, or neurological data to measure positive/negative affect, anxiety, and depression. Third, chronic interpersonal stress and SAD symptom severity may possess a generative relationship, similar to that of stress generation observed in depressive disorders (Hammen, 1991). This hypothesis states that behaviors of depressed individuals – influenced by negative, depressive beliefs and expectations – contribute to the occurrence of negative life events. Chronic interpersonal stress continuation has been demonstrated in anxiety disorders broadly with neuroticism implicated as a partial contributor in this relationship, where continued stress elevation may be attributed to behaviors that correspond with high neuroticism (Uliaszek et al. (2012). However, stress generation has yet to be examined specifically in individuals with SAD. Thus, further statistical analyses employing

longitudinal data, such as cross-lagged analyses or bidirectional associations, is warranted to determine the dynamics of this relationship. Lastly, our analyses were limited to SAD, GAD, and MDD. Future studies could examine the effects on other anxiety disorders.

Overall, this study highlights an exciting line of research that deserves further attention. If positive affect can indeed attenuate the effects of chronic interpersonal stress on SAD and MDD symptom severity, efforts to increase positive affect (e.g., Craske, Meuret, Ritz, Treanor, Dour & Rosenfield, in submission) in cognitive-behavioral therapy may result in further symptom reduction and protection from the deleterious effects of chronic interpersonal stress in the future.

References

- Affleck G, & Tennen H (1996). Construing benefits from adversity: Adaptational significance and dispositional underpinnings. *Journal of Personality*, 64, 899–922. [PubMed: 8956517]
- Aiken LS, & West SG (1991). *Multiple regression: Testing and interpreting interactions*. Newbury park, CA: Sage Publications.
- Amies PL, Gelder M, & Shaw P (1983). Social phobia: A comparative clinical study. *The British Journal of Psychiatry*, 142, 174–179. [PubMed: 6839072]
- Arborelius L, Owens M, Plotsky P, & Nemeroff C (1999). The role of corticotropin-releasing factor in depression and anxiety disorders. *Journal of Endocrinology*, 160, 1–12. [PubMed: 9854171]
- Baran SE, Armstrong CE, Niren DC, Hanna JJ, & Conrad CD (2009). Chronic stress and sex differences on the recall of fear conditioning and extinction. *Neurobiology of Learning and Memory*, 91, 323–332. [PubMed: 19073269]
- Barsy B, Leveleki C, Zelena D, & Haller J (2010). The context specificity of anxiety responses induced by chronic psychosocial stress in rats: A shift from anxiety to social phobia? *Stress*, 13, 230–237. [PubMed: 20392194]
- Baum A, Garofalo JP, & Yali AM (1999). Socioeconomic status and chronic stress: does stress account for SES effects on health?. *Annals of the New York Academy of Sciences*, 896(1), 131–144. [PubMed: 10681894]
- Beesdo K, Knappe S, & Pine DS (2009). Anxiety and anxiety disorders in children and adolescents: Developmental issues and implications for DSM-V. *The Psychiatric Clinics of North America*, 32, 483–524. [PubMed: 19716988]
- Beesdo K, Pine DS, Lieb R, & Wittchen H-U (2010). Incidence and risk patterns of anxiety and depressive disorders and categorization of generalized anxiety disorder. *Archives of General Psychiatry*, 67, 47–57. [PubMed: 20048222]
- Billings DW, Folkman S, Acree M, & Moskowitz JT (2000). Coping and physical health during caregiving: The roles of positive and negative affect. *Journal of Personality and Social Psychology*, 79, 131. [PubMed: 10909883]
- Blazer D, Hughes DC, & George LK (1987). The epidemiology of depression in an elderly community population. *The Gerontologist*, 27, 281–287. [PubMed: 3609795]
- Brown TA, Chorpita BF, & Barlow DH (1998). Structural relationships among dimensions of the DSM-IV anxiety and mood disorders and dimensions of negative affect, positive affect, and autonomic arousal. *Journal of Abnormal Psychology*, 107, 179–192. [PubMed: 9604548]
- Carver CS (1998). Resilience and thriving: Issues, models, and linkages. *Journal of Social Issues*, 54, 245–266.
- Chambers JA, Power KG, & Durham RC (2004). The relationship between trait vulnerability and anxiety and depressive diagnoses at long-term follow-up of generalized anxiety disorder. *Journal of Anxiety Disorders*, 18, 587–607. [PubMed: 15275941]
- Charney DS (2004). Psychobiological mechanisms of resilience and vulnerability: Implications for successful adaptation to extreme stress. *American Journal of Psychiatry*, 161, 195–216. [PubMed: 14754765]

- Chorpita BF, Plummer CM, & Moffitt CE (2000). Relations of tripartite dimensions of emotion to childhood anxiety and mood disorders. *Journal of Abnormal Child Psychology*, 28, 299–310. [PubMed: 10885687]
- Clark LA, Watson D, & Mineka S (1994). Temperament, personality, and the mood and anxiety disorders. *Journal of Abnormal Psychology*, 103, 103. [PubMed: 8040472]
- Cohen J (1960). A coefficient of agreement for nominal scales. *Educational and Psychological Measurement*, 20, 37–46.
- Connolly NP, Eberhart NK, Hammen CL, & Brennan PA (2010). Specificity of stress generation: A comparison of adolescents with depressive, anxiety, and comorbid diagnoses. *International Journal of Cognitive Therapy*, 3, 368–379. [PubMed: 22724042]
- Sheets ES, & Craighead WE (2014). Comparing chronic interpersonal and noninterpersonal stress domains as predictors of depression recurrence in emerging adults. *Behaviour Research and Therapy*, 63, 36–42. [PubMed: 25277497]
- Craske MG, Meuret AE, Ritz T, Treanor M, & Dour HJ (2016). Treatment for anhedonia: A neuroscience driven approach. *Depression and Anxiety*, 33(10), 927–938. [PubMed: 27699943]
- Craske MG, Meuret AE, Ritz T, Treanor M, Dour HJ & Rosenfield D (in submission). Treatment for anhedonia: A randomized clinical trial.
- Davila J, & Beck JG (2002). Is social anxiety associated with impairment in close relationships? A preliminary investigation. *Behavior Therapy*, 33, 427–446.
- DeWit DJ, MacDonald K, & Offord DR (1999). Childhood stress and symptoms of drug dependence in adolescence and early adulthood: Social phobia as a mediator. *American Journal of Orthopsychiatry*, 69, 61–72. [PubMed: 9990437]
- Di Nardo P, & Barlow D (1988). *Anxiety Disorders Interview Schedule for DSM-III-R (ADIS-R)*. Albany, NY: Graywind.
- Doane LD, Mineka S, Zinbarg RE, Craske M, Griffith JW, & Adam EK (2013). Are flatter diurnal cortisol rhythms associated with major depression and anxiety disorders in late adolescence? The role of life stress and daily negative emotion. *Development and Psychopathology*, 25, 629–642. [PubMed: 23880381]
- Dohrenwend BP, Levav I, Shrout PE, Schwartz S, Naveh G, Link BG, ... & Stueve A (1992). Socioeconomic status and psychiatric disorders: the causation-selection issue. *Science*, 255(5047), 946–952. [PubMed: 1546291]
- Eysenck HJ, & Eysenck SBG (1975). *Manual of the Eysenck Personality Questionnaire (junior and adult)*. London, England: Hodder and Stoughton.
- Faravelli C, Lo Sauro C, Lelli L, Pietrini F, Lazzaretto L, Godini L, ... Castellini G, (2012a). The role of life events and HPA axis in anxiety disorders: A review. *Current Pharmaceutical Design*, 18, 5663. [PubMed: 22632471]
- Faravelli C, Sauro CL, Godini L, Lelli L, Benni L, Pietrini F, ... Ricca V, (2012b). Childhood stressful events, HPA axis and anxiety disorders. *World Journal of Psychiatry*, 2, 13. [PubMed: 24175164]
- Folkman S, & Moskowitz JT (2000a). Positive affect and the other side of coping. *American Psychologist*, 55, 647. [PubMed: 10892207]
- Folkman S, & Moskowitz JT (2000b). Stress, positive emotion, and coping. *Current Directions in Psychological Science*, 9, 115–118.
- Folkman S, Moskowitz JT, Ozer EM, & Park CL (1997). Positive meaningful events and coping in the context of HIV/AIDS Coping with chronic stress (pp. 293–314). New York, NY: Springer.
- Fredrickson BL (1998). What good are positive emotions? *Review of General Psychology*, 2, 300. [PubMed: 21850154]
- Fredrickson BL (2004). The broaden-and-build theory of positive emotions. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 359, 1367.
- Fredrickson BL, & Branigan C (2005). Positive emotions broaden the scope of attention and thought-action repertoires. *Cognition & Emotion*, 19, 313–332. [PubMed: 21852891]
- Fredrickson BL, & Joiner T (2002). Positive emotions trigger upward spirals toward emotional well-being. *Psychological Science*, 13, 172–175. [PubMed: 11934003]

- Fredrickson BL, Mancuso RA, Branigan C, & Tugade MM (2000). The undoing effect of positive emotions. *Motivation and Emotion*, 24, 237–258. [PubMed: 21731120]
- Gruber J, Johnson SL, Oveis C, & Keltner D (2008). Risk for mania and positive emotional responding: Too much of a good thing? *Emotion*, 8, 23. [PubMed: 18266513]
- Hammen C (1991). Generation of stress in the course of unipolar depression. *Journal of Abnormal Psychology*, 100, 555. [PubMed: 1757669]
- Hammen C (2003). Interpersonal stress and depression in women. *Journal of Affective Disorders*, 74(1), 49–57. [PubMed: 12646298]
- Hammen C, Adrian C, Gordon D, Burge D, Jaenicke C, & Hiroto D (1987). Children of depressed mothers: Maternal strain and symptom predictors of dysfunction. *Journal of Abnormal Psychology*, 96, 190. [PubMed: 3680756]
- Hammen C, Kim EY, Eberhart NK, & Brennan PA (2009). Chronic and acute stress and the prediction of major depression in women. *Depression and anxiety*, 26, 718–723. [PubMed: 19496077]
- Hammen C, Shih JH, & Brennan PA (2004). Intergenerational transmission of depression: test of an interpersonal stress model in a community sample. *Journal of Consulting and Clinical Psychology*, 72(3), 511. [PubMed: 15279534]
- Hayward C, Killen JD, Kraemer HC, & Taylor CB (2000). Predictors of panic attacks in adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry*, 39, 207–214. [PubMed: 10673832]
- Heim C, Newport DJ, Mletzko T, Miller AH, & Nemeroff CB (2008). The link between childhood trauma and depression: Insights from HPA axis studies in humans. *Psychoneuroendocrinology*, 33, 693–710. [PubMed: 18602762]
- Hollingshead A Four factor index of social status. New Haven, CT: Yale University; 1975 Unpublished manuscript.
- Isen AM, Daubman KA, & Nowicki GP (1987). Positive affect facilitates creative problem solving. *Journal of Personality and Social Psychology*, 52, 1122. [PubMed: 3598858]
- Kashdan TB (2007). Social anxiety spectrum and diminished positive experiences: Theoretical synthesis and meta-analysis. *Clinical Psychology Review*, 27, 348–365. [PubMed: 17222490]
- Keltner D, & Bonanno GA (1997). A study of laughter and dissociation: Distinct correlates of laughter and smiling during bereavement. *Journal of Personality and Social Psychology*, 73, 687. [PubMed: 9325589]
- Kendall AD, Zinbarg RE, Bobova L, Mineka S, Revelle W, Prenoveau JM, & Craske MG (2016). Measuring positive emotion with the mood and anxiety symptom questionnaire: Psychometric properties of the Anhedonic Depression Scale. *Assessment*, 23, 86–95. [PubMed: 25657305]
- Kendler KS, Hettema JM, Butera F, Gardner CO, & Prescott CA (2003). Life event dimensions of loss, humiliation, entrapment, and danger in the prediction of onsets of major depression and generalized anxiety. *Archives of General Psychiatry*, 60, 789–796. [PubMed: 12912762]
- Keogh E, & Reidy J (2000). Exploring the factor structure of the Mood and Anxiety Symptom Questionnaire (MASQ). *Journal of Personality Assessment*, 74, 106–125. [PubMed: 10779936]
- Kessler RC (1997). The effects of stressful life events on depression. *Annual Review of Psychology*, 48(1), 191–214.
- Kessler RC, Davis CG, & Kendler KS (1997). Childhood adversity and adult psychiatric disorder in the US National Comorbidity Survey. *Psychological Medicine*, 27(5), 1101–1119. [PubMed: 9300515]
- Kessler RC, Gruber M, Hettema JM, Hwang I, Sampson N, & Yonkers KA (2008). Co-morbid major depression and generalized anxiety disorders in the National Comorbidity Survey follow-up. *Psychological Medicine*, 38, 365–374. [PubMed: 18047766]
- Khazanov GK, & Ruscio AM (2016). Is low positive emotionality a specific risk factor for depression? A meta-analysis of longitudinal studies. *Psychological Bulletin*, 142(9), 991. [PubMed: 27416140]
- Kotov R, Gamez W, Schmidt F, & Watson D (2010). Linking “big” personality traits to anxiety, depressive, and substance use disorders: a meta-analysis. *Psychological Bulletin*, 136(5), 768. [PubMed: 20804236]
- Fredrickson BL, & Levenson RW (1998). Positive emotions speed recovery from the cardiovascular sequelae of negative emotions. *Cognition & Emotion*, 12, 191–220. [PubMed: 21852890]

- Lazarus RS (1993). From psychological stress to the emotions: A history of changing outlooks. *Annual Review of Psychology*, 44, 1–22.
- Mazure CM (1998). Life stressors as risk factors in depression. *Clinical Psychology: Science and Practice*, 5(3), 291–313.
- McGonagle KA, & Kessler RC (1990). Chronic stress, acute stress, and depressive symptoms. *American Journal of Community Psychology*, 18, 681–706. [PubMed: 2075897]
- McWilliams LA, Cox BJ, & Enns MW (2003). Mood and anxiety disorders associated with chronic pain: An examination in a nationally representative sample. *Pain*, 106, 127–133. [PubMed: 14581119]
- Moffitt TE, Caspi A, Harrington H, Milne BJ, Melchior M, Goldberg D, & Poulton R (2007). Generalized anxiety disorder and depression: Childhood risk factors in a birth cohort followed to age 32. *Psychological Medicine*, 37, 441–452. [PubMed: 17201999]
- Moreno-Peral P, Conejo-Cerón S, Motrico E, Rodríguez-Morejón A, Fernández A, García-Campayo J, ... Bellón JÁ (2014). Risk factors for the onset of panic and generalised anxiety disorders in the general adult population: a systematic review of cohort studies. *Journal of Affective Disorders*, 168, 337–348. [PubMed: 25089514]
- Monroe SM, & Simons AD (1991). Diathesis-stress theories in the context of life stress research: Implications for the depressive disorders. *Psychological Bulletin*, 110, 406. [PubMed: 1758917]
- Ong AD, Bergeman CS, & Bisconti TL (2004). The role of daily positive emotions during conjugal bereavement. *The Journals of Gerontology Series B: Psychological Sciences and Social Sciences*, 59, P168–P176.
- Ong AD, Bergeman CS, Bisconti TL, & Wallace KA (2006). Psychological resilience, positive emotions, and successful adaptation to stress in later life. *Journal of Personality and Social Psychology*, 91, 730. [PubMed: 17014296]
- Phillips NK, Hammen CL, Brennan PA, Najman JM, & Bor W (2005). Early adversity and the prospective prediction of depressive and anxiety disorders in adolescents. *Journal of Abnormal Child Psychology*, 33, 13–24. [PubMed: 15759588]
- Power M, & Tarsia M (2007). Basic and complex emotions in depression and anxiety. *Clinical Psychology & Psychotherapy*, 14, 19–31.
- Prenoveau JM, Zinbarg RE, Craske MG, Mineka S, Griffith JW, & Epstein AM (2010). Testing a hierarchical model of anxiety and depression in adolescents: A tri-level model. *Journal of Anxiety Disorders*, 24, 334–344. [PubMed: 20171054]
- Pynoos RS, Steinberg AM, & Piacentini JC (1999). A developmental psychopathology model of childhood traumatic stress and intersection with anxiety disorders. *Biological Psychiatry*, 46, 1542–1554. [PubMed: 10599482]
- Quene H, & Van den Bergh H (2004). On multi-level modeling of data from repeated measures designs: A tutorial. *Speech Communication*, 43, 103–121.
- Raudenbush SW, Bryk AS, Cheong YF, Congdon R, & Du Toit M (2011). *Hierarchical linear and nonlinear modeling (HLM7)*. Lincolnwood, IL: Scientific Software International, 1112.
- Rosellini AJ, Rutter LA, Bourgeois ML, Emmert-Aronson BO, & Brown TA (2013). The relevance of age of onset to the psychopathology of social phobia. *Journal of Psychopathology and Behavioral Assessment*, 35, 356–365. [PubMed: 23935239]
- Rudolph KD, Hammen C, Burge D, Lindberg N, Herzberg D, & Daley SE (2000). Toward an interpersonal life-stress model of depression: *The developmental context of stress generation*. *Development and psychopathology*, 12(2), 215–234. [PubMed: 10847625]
- Shih JH, Eberhart NK, Hammen CL, & Brennan PA (2006). Differential exposure and reactivity to interpersonal stress predict sex differences in adolescent depression. *Journal of Clinical Child and Adolescent Psychology*, 35(1), 103–115. [PubMed: 16390306]
- Sewart AR, & Craske MG (in press). Inhibitory Learning in Exposure Therapy In Abramowitz JA & Blakey S (Eds.), *Clinical Handbook of Fear and Anxiety: Psychological Processes and Treatment Mechanisms*.
- Spitzer M, Robert L, Gibbon M, & Williams J (2002). Structured clinical interview for DSM-IV-TR axis I disorders, research version, non-patient edition (SCID-I/NP). New York: Biometrics Research, New York State Psychiatric Institute.

- Strand EB, Zautra AJ, Thoresen M, Ødegård S, Uhlig T, & Finset A (2006). Positive affect as a factor of resilience in the pain—negative affect relationship in patients with rheumatoid arthritis. *Journal of Psychosomatic Research*, 60, 477–484. [PubMed: 16650588]
- Tugade MM, & Fredrickson BL (2007). Regulation of positive emotions: Emotion regulation strategies that promote resilience. *Journal of Happiness Studies*, 8, 311–333.
- Uliaszek AA, Hauner KKY, Zinbarg RE, Craske MG, Mineka S, Griffith JW, & Rose RD (2009). An examination of content overlap and disorder-specific predictions in the associations of neuroticism with anxiety and depression. *Journal of Research in Personality*, 43, 785–794. [PubMed: 20161016]
- Uliaszek AA, Zinbarg RE, Mineka S, Craske MG, Griffith JW, Sutton JM, ... Hammen C (2012). A longitudinal examination of stress generation in depressive and anxiety disorders. *Journal of Abnormal Psychology*, 121, 4–15. [PubMed: 22004114]
- Vyas A, Pillai A, & Chattarji S (2004). Recovery after chronic stress fails to reverse amygdaloid neuronal hypertrophy and enhanced anxiety-like behavior. *Neuroscience*, 128, 667–673. [PubMed: 15464275]
- Watson D, & Clark LA (1984). Negative affectivity: the disposition to experience aversive emotional states. *Psychological Bulletin*, 96(3), 465. [PubMed: 6393179]
- Watson D, & Clark LA (1991). The mood and anxiety symptom questionnaire. Unpublished manuscript, University of Iowa, Department of Psychology, Iowa City.
- Watson D, & Clark LA (1999). The PANAS-X: Manual for the positive and negative affect schedule-expanded form.
- Watson D, Clark LA, & Carey G (1988). Positive and negative affectivity and their relation to anxiety and depressive disorders. *Journal of Abnormal Psychology*, 97, 346. [PubMed: 3192830]
- Watson D, Weber K, Assenheimer JS, Clark LA, Strauss ME, & McCormick RA (1995). Testing a tripartite model: I. Evaluating the convergent and discriminant validity of anxiety and depression symptom scales. *Journal of Abnormal Psychology*, 104, 3. [PubMed: 7897050]
- Wheaton B (1997). The nature of chronic stress Coping with chronic stress (pp. 43–73). New York, NY: Springer.
- Wittchen H-U, Stein MB, & Kessler RC (1999). Social fears and social phobia in a community sample of adolescents and young adults: Prevalence, risk factors and comorbidity. *Psychological Medicine*, 29, 309–323. [PubMed: 10218923]
- Zautra A, Smith B, Affleck G, & Tennen H (2001). Examinations of chronic pain and affect relationships: Applications of a dynamic model of affect. *Journal of Consulting and Clinical Psychology*, 69, 786. [PubMed: 11680555]
- Zautra AJ, Johnson LM, & Davis MC (2005). Positive affect as a source of resilience for women in chronic pain. *Journal of Consulting and Clinical Psychology*, 73, 212. [PubMed: 15796628]
- Zimmerman M, & Coryell W (1987). The Inventory to Diagnose Depression (IDD): a self-report scale to diagnose major depressive disorder. *Journal of Consulting and Clinical Psychology*, 55(1), 55–59. [PubMed: 3571659]
- Zinbarg RE, Mineka S, Craske MG, Griffith JW, Sutton J, Rose RD, ... Waters AM. (2010). The Northwestern-UCLA youth emotion project: Associations of cognitive vulnerabilities, neuroticism and gender with past diagnoses of emotional disorders in adolescents. *Behaviour Research and Therapy*, 48, 347–358. [PubMed: 20070951]

Table 1

Means and Standard Deviations of Positive Affect, Chronic Interpersonal Stress, and Disorder Symptom Severity

Measure	<i>M</i>	<i>SD</i>	<i>Min</i>	<i>Max</i>	<i>N</i>
Positive affect ^a	2.89	0.83	1.00	5.00	2626
Negative affect ^a	1.90	0.67	1.00	4.93	2626
Chronic interpersonal stress ^b	2.29	0.42	1.25	4.50	2626
Chronic non-interpersonal stress ^b	2.23	0.33	1.42	3.83	2621
Social anxiety disorder symptom severity ^c	0.30	0.95	0	7.00	1898
Generalized anxiety symptom severity ^c	0.14	0.72	0	6.00	1897
Major depressive disorder symptom severity ^c	0.16	0.82	0	7.00	1898

Note.

^aMood and Anxiety Symptom Questionnaire (MASQ; Watson & Clark, 1991) ranges 1 (not at all) to 5 (extremely);

^bLife Stress Inventory (LSI; Hammen, 1991; Hammen et al., 1987) ranges from 1 (minimal stress) to 5 (very stressful circumstances);

^cClinical Severity Rating (CSR; Di Nardo & Barlow, 1988) ranges from 0 (no distress/interference) to 8 extreme distress/interference).

Table 2

Bivariate Between Group Correlations Among Positive Affect, Negative Affect, Chronic Interpersonal Stress, and Disorder Symptom Severity

Measure	1	2	3	4	5	6	7
1. Positive affect	—						
2. Negative affect	-.259 **	—					
3.. Chronic interpersonal stress	-.216 **	.218 **	—				
4. Chronic non-interpersonal stress	-.168 **	.143 **	.470 **	—			
5. Social anxiety disorder symptom severity	-.074 **	.118 **	.092 **	.046 *	—		
6. Generalized anxiety symptom severity	-.041	.127 **	.101 **	.122 **	.205 **	—	
7. Major depressive disorder symptom severity	-.060 **	.133 **	.104 **	.107 **	.144 **	.211 **	—

Note.

**
 $p < .01$,

*
 $p < .05$, Pairwise deletion

Table 3

Multilevel Poisson Models for Positive Affect, Negative Affect, Chronic Interpersonal Stress, Social Anxiety, Generalized Anxiety, and Major Depressive Disorder Symptom Severity

Measure	Social anxiety disorder symptom severity				Generalized anxiety disorder symptom severity				Major depressive disorder symptom severity			
	β	SE	t-ratio	~ d.f.	β	SE	t-ratio	~ d.f.	β	SE	t-ratio	~ d.f.
Positive affect	-.194	.078	-2.458	462	.014	-.290	.114	-2.545	.011	.182	.580	462
Negative affect	.097	.110	.886	462	.376	-.197	.145	-1.365	.173	.284	3.175	462
Chronic interpersonal stress	.194	.181	1.072	462	.284	.316	.251	1.262	.208	.336	1.383	462
Positive affect $\times$ Negative affect	-.115	.060	-1.932	462	.054	-.027	.077	-2.212	.027	.252	.500	462
Chronic interpersonal stress $\times$ Positive affect	-.148	.058	-2.548	462	.011	-.080	.081	.988	.324	.242	-2.714	462
Chronic interpersonal stress $\times$ Negative affect	.031	.063	.492	462	.623	-.103	.077	-1.374	.170	.217	-.509	462

Table 4

Multilevel Poisson Models for Positive Affect, Negative Affect, Chronic Non-Interpersonal Stress, Social Anxiety, Generalized Anxiety, and Major Depressive Disorder Symptom Severity

Measure	Social anxiety disorder symptom severity					Generalized anxiety disorder symptom severity					Major depressive disorder symptom severity				
	β	SE	t-ratio	$\sim d.f.$	p	β	SE	t-ratio	$\sim d.f.$	p	β	SE	t-ratio	$\sim d.f.$	p
Positive affect	.162	.082	1.955	462	.051	-.154	.115	-1.339	462	.181	-.044	.166	-.264	462	.792
Negative affect	.104	.107	.977	462	.329	-.243	.135	-1.798	462	.073	.305	.154	1.978	462	.049
Chronic non-interpersonal stress	.337	.212	1.591	462	.112	1.287	.302	4.625	462	<.001	-.277	.392	-.706	462	.481
Positive affect $\times$ Negative affect	-.158	.059	-2.657	462	.008	-.231	.082	-2.821	462	.005	-.310	.119	-2.607	462	.009
Chronic non-interpersonal stress $\times$ Positive affect	.068	.059	1.149	462	.251	-.097	.074	-1.199	462	.231	.233	.154	1.978	462	.481
Chronic non-interpersonal stress $\times$ Negative affect	-.084	.054	-1.551	462	.122	.037	.082	.501	462	.617	.778	.242	3.208	462	.001