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Generalized Anxiety Symptoms and Interpersonal Self-Perceptions During Stressors: A Prospective Examination of Psychological and Biological Stress

Jamie A. Lewis
Seattle Pacific University

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Jamie A. Lewis

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Reviewed by:

Thane M. Erickson, Ph.D.
Professor of Clinical Psychology
Dissertation Chair

Amy Mezulis Ph.D.
Chair, Clinical Psychology
Seattle Pacific University

John Thoburn, Ph.D.
Professor of Clinical Psychology
Committee Member

Katy Tangenberg, Ph.D.
Dean, School of Psychology,
Family & Community

Jacob Bentley, Ph.D.
Associate Professor of Clinical Psychology
Committee Member

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Abstract

Individuals with generalized anxiety disorder (GAD) symptoms endorse negative emotionality, psychosocial dysfunction, and biological dysregulation. Interpersonal dominance and affiliation have also been linked to GAD symptoms. Little research has examined individuals with GAD symptoms in terms of naturalistic stressors and chronic use of interpersonal behaviors. GAD symptoms, as well as lower dominance and affiliation, have been linked to hypothalamic-pituitary-adrenal (HPA) dysregulation. However, no studies have examined the unique and interacting contributions of GAD symptoms and interpersonal processes to chronic cortisol levels and distress. College students completed baseline measures of GAD symptoms, measures of interpersonal self-perceptions and distress for five weeks, then a lab visit to collect hair cortisol samples. I hypothesized that higher GAD symptoms and lower dominance and affiliation, would predict higher cortisol and psychological distress during interpersonal stressors. I expected that effects of dominance and affiliation on psychological and biological outcomes would be blunted for individuals higher in GAD symptoms. Results showed that GAD symptoms predicted higher distress and cortisol. Effects of interpersonal variables varied depending on whether they reflected aggregate mean levels or person-centered levels. Person-centered affiliation predicted lower distress, and blunted effects of GAD symptoms on distress. Mean level dominance predicted *higher* levels of both distress and cortisol. A marginally significant three-way interaction suggested the possibility that GAD symptoms combined with high dominance and low affiliation might predict the highest cortisol levels. Understanding the ways chronic use of interpersonal behaviors influence the relationship between GAD and negative outcomes may inform interventions for those with GAD symptoms.

CHAPTER I:

Introduction and Literature Review

Worry, a perseverative form of negative cognition in which one anticipates possible negative future outcomes, is relatively normal, common, and not always pathological. However, worry may become excessive or uncontrollable in the context of diagnosed generalized anxiety disorder (GAD), other emotional disorders, and in subclinical individuals who worry chronically without meeting full criteria for such disorders. GAD is a mental health disorder characterized by excessive and uncontrollable worry and anxiety about multiple topics, which often include occupational, academic, financial, interpersonal, and other domains. Diagnostic criteria for GAD in the Diagnostic and Statistical Manual – fifth edition (DSM-5; American Psychiatric Association, 2013) require that the worry and anxiety are associated with at least three of the following symptoms on at least half of the days of six months or longer: restlessness or feeling keyed up or on edge, becoming easily fatigued, difficulty concentrating, irritability, muscle tension, and sleep disturbance.

Both GAD symptoms and worry have been linked to distress and impairment along a spectrum of subclinical to clinical levels. Taxometric analyses suggest that GAD symptoms (Kertz, McHugh, Lee, & Björgvinsson, 2014; Marcus, Sawaqdeh, & Kwon, 2014) and worry (Kertz et al., 2014) lie along a continuous, latent symptom dimension and therefore are best conceptualized as dimensional rather than as discrete categories. For example, some individuals with pathological worry also suffer from disability, despite not meeting full GAD criteria or failing to meet the six-month criteria (Lee, Vaingankar, Chong, & Subramaniam, 2016). In addition, GAD symptoms and worry have predicted markers of biological stress and dysfunction (Arbel, Shapiro, Timmons, Moss & Margolin, 2017; Fisher, 2015; Steudte et al., 2011). For

adults (Marcus et al., 2014), community participants (Olatunji et al., 2010), and undergraduate participants (Olatunji et al., 2010), the diagnostic thresholds used to identify GAD may be arbitrary - thus, providing support for a dimensional reconceptualization of worry and GAD symptoms. The core symptom of worry is problematic in the context of GAD, subclinical GAD, and independent of GAD, and therefore research must further elucidate the ways that such GAD symptoms contribute to distress in individuals' lives.

In addition, GAD symptoms have been consistently found to associate with reduced psychosocial functioning including interpersonal problems (Przeworski et al., 2011) and marital dissatisfaction (Afifi, Cox, & Enns, 2006). Additionally, the most common worry topic endorsed by individuals with GAD is social concerns. Previous reviews (e.g., Newman & Erickson, 2010) have found that interpersonal factors on the interpersonal circumplex (IPC) dimensions of dominance versus submissiveness and affiliation versus coldness are relevant to GAD and may play a role in how individuals experience their symptoms. However, research has been inconsistent on whether GAD symptoms are associated with one prototypical interpersonal style or instead with heterogeneous interpersonal styles. Moreover, most of the research in this domain has been cross-sectional in nature.

In the present study I aim to examine in a prospective study how dimensional GAD symptoms predict downstream negative emotions and cortisol as a biomarker of stress, as well as how these relationships may depend on participants' chronic use of interpersonal behaviors in social stressors. Prior to discussing the current study, I provide an overview of GAD prevalence and associated problems, links to interpersonal dysfunction, and cortisol and its links to GAD and interpersonal domains.

GAD Prevalence and Course

GAD is one of the most frequent mental health conditions encountered within the general population (Skapinakis et al., 2013). The DSM-5 estimates a prevalence rate of GAD of 2.9% among adults in the United States (APA, 2013), although some estimates are slightly (e.g., 4.1% and 4.5%; Skapinakis et al., 2013) and notably higher (18% in a primary care sample; Bunevicius et al., 2014). GAD is one of the most common mental disorders observed in primary care settings (Bunevicius et al., 2014), approximately 8.3% in one large scale study (Ansseau, Fischler, Dierick, Mignon, & Leyman, 2005). Prevalence in adolescence has been estimated around 3% for threshold GAD, 5% for subthreshold GAD as determined by a reduced duration criterion, and 6% when further relaxing the uncontrollability criterion (Burstein, Beesdo-Baum, He, & Merikangas, 2014). It may be that prevalence in youth populations is actually greater, but is clouded by misdiagnosis of another disorder such as ADHD. In fact, one study found quite a high rate of comorbidity with externalizing disorders, particularly for ADHD (45%; Jarrett et al, 2015). Additionally, it was found that adolescents with threshold GAD most frequently endorsed symptoms including difficulty concentrating, irritability, and restlessness, whereas the least frequently endorsed symptom was muscle tension (Burstein et al., 2014). Consistent with links between female gender and higher anxiety disorder rates (Bunevicius et al., 2014), women are generally more prone to GAD than men (e.g., 5.6% versus 2.5%; Skapinakis et al., 2013). European-American ancestry is also linked to GAD (Kertz & Woodruff-Borden, 2011).

In addition to full-blown GAD, many people experience GAD symptoms without meeting full criteria. Other research has found greater prevalence estimates for subthreshold GAD, relative to clinical GAD, estimated as two times more prevalent than DSM diagnosed populations (Haller, Cramer, Lauche, Gass, & Dobos, 2014). For instance, prevalence rates for subthreshold GAD (as defined by one out of four diagnostic criteria not fulfilled) were

approximately 2.4% and, when further relaxing the criteria to endorsing a single GAD criterion, approximately 3.33% (Hoyer, Becker, & Margraf, 2002). Moreover, even in healthy individuals, an estimated 24.8% reported intense/frequent worry episodes pertaining to a single topic (Hoyer, Becker, & Margraf, 2002), consistent with the idea that GAD symptoms and associated impairments are dimensional in nature.

The onset of GAD can occur across the lifespan (Watterson, Williams, Lavorato, & Patten, 2017). One epidemiological household survey conducted over the course of a year found that the median age of onset for GAD was 20 years (Vaingankar, Rekhi, Subramaniam, Abdin, & Chong, 2013). A national sample survey of Korean adults revealed that the median age of onset was earliest for anxiety disorders (relative to other mood disorders and alcohol use disorders), at age 29 (Cho et al., 2012). In contrast, Muhsen and colleagues found the highest prevalence of GAD in individuals between the ages of 40 and 59 (Muhsen, Lipsitz, Garty-Sandalon, Gross, & Green, 2008), although it is also relatively common in young adults. In fact, some research has indicated a bimodal distribution with peaks for both early- (defined as before age 50) and late (defined as after 50) onset (Le Roux, Gatz, & Wetherell, 2005). Rhebergen and colleagues (2017) also found a bimodal distribution using different age parameters, that is, early onset (defined as 24 years and younger) and late onset (defined as greater than 24 years). The DSM-5 reports a median age of 30 (APA, 2013). Additionally, those with full GAD have reported significantly younger average age (average age of 43) compared to those who endorsed only Criterion A (excessive worry occurring more days than not; average age of 51) but did not differ in age from subthreshold groups endorsing Criterion A and one or two other criteria (average of 49; Kertz & Woodruff-Borden, 2011). Different factors, such as gender, education, physical

illness, and personality factors may contribute to the development of early- vs. late-onset (Rhebergen et al., 2017).

GAD typically runs a chronic and persistent course with waxing and waning of symptoms (American Psychiatric Association, 2013). In fact, in a prospective study, patients with generalized anxiety disorder had low probability of achieving recovery over 12 years of follow-up (probability of 0.58; Bruce et al., 2005). Destoop and colleagues (2013) identified the point prevalence of remission at 13.3% of their clinical sample. Lower remission rates have been associated with lower socioeconomic status, the presence of comorbid symptoms, and less medication use as well (Destoop et al., 2013). Reductions in the likelihood of remission has also been associated with lower overall life satisfaction, poor relationships, and comorbid personality disorders (Yonkers, Dyck, Warshaw, & Keller, 2000). Remission rates may be comparable between psychiatric and primary care settings (Destoop et al., 2013).

Evidence of Impairment Associated with GAD Symptoms

In addition to the prevalence and chronicity of GAD symptoms (whether clinical or subclinical), individuals endorsing such symptoms report diminished well-being and satisfaction with one's life (Stein & Heimberg, 2004). In fact, epidemiological data indicates that reductions in psychosocial functioning for those with subthreshold GAD is comparable to those with fulfilled GAD criteria, and much lower relative to healthy controls (Hoyer, Becker & Margraf, 2002). One meta-analysis found a large effect size for the relationship between anxiety disorder status (relative to controls) and lower quality of life, and this effect was present across all anxiety disorders including GAD (Olatunji, Cisler, Tolin, 2007). Previous research has found that both GAD and non-GAD worriers perceive themselves as impaired across significant life domains (Gentes & Ruscio, 2014). Several lines of evidence that suggest that GAD is associated with

impairment, including research on emotional distress, comorbidity, dysfunctional cognition, role dysfunction, and somatic problems. Representative findings from these literatures are summarized next.

GAD and psychological distress. GAD symptoms and worry predict psychological distress. Psychological distress is a term used to describe the negative affect (NA) and uncomfortable thoughts that impact an individual's level and degree of functioning. Correlational studies have found links of GAD symptoms and negative emotions. For instance, Erickson and Newman (2007) found greater levels of sad affect in a GAD sample relative to non-anxious controls during a social interaction task. In an 8-14-year follow-up, individuals diagnosed and treated for GAD demonstrated elevated scores for trait negative affect, trait anxiety, and trait depression (Chambers, Power, & Durham, 2004). Emotion regulation deficits related to distressing negative emotions, including difficulties with emotional clarity and acceptance of emotions, have been linked with chronic worry and GAD (Salters-Pedneault, Roemer, Tull, Rucker, & Mennin, 2006). In addition to evidence suggesting greater negative emotionality and distress, low trait positive emotionality prospectively predicted initial onset of GAD (Kendall et al., 2015).

Additionally, there is evidence of causal links such that GAD symptoms and worry cause NA. Induction of worry has been associated with eliciting emotional components of both depression and somatic anxiety (Andrews & Borkovec, 1988). In a study that manipulated worry, individuals engaged in 5-minute counterbalanced worry induction while recalling past traumatic events, and worry increased anxious and depressed affect (Behar, Zuellig, & Borkovec, 2005). In a study of individuals with GAD and non-anxious controls randomly assigned to worry, relaxation, and neutral inductions preceding sequential exposure to various emotional

film clips, worry increased negative emotionality from baseline (Llera, & Newman, 2014). This was the case for both individuals with GAD and non-anxious controls.

Psychiatric comorbidities. Comorbidity of GAD with other conditions provides evidence of impairment, with as many as 89% of individuals with GAD endorsing co-occurring psychiatric conditions in primary care settings (Olfson et al., 1997). GAD shares high comorbid rates with other anxiety disorders (Hoyer, Becker, & Margraf, 2002). Individuals with GAD have heightened rates of depressive disorders including major depressive disorder and dysthymia (Ma et al., 2009; Stein & Heimberg, 2004). Moreover, subclinical worry correlates with other forms of anxiety and depressive symptoms (Erickson et al., 2016). GAD and other anxiety disorders are predictive of increases in the likelihood of lifetime substance use disorders (Goodwin & Stein, 2013). Lastly, GAD symptoms have been linked to personality disorders and related traits. More than half of those diagnosed with GAD may meet criteria for a comorbid personality disorder, especially within the cluster C personality disorders (e.g., avoidant and obsessive-compulsive personality disorder; Garyfallos et al., 1999). The presence of at least one personality disorder is positively associated with occurrence of GAD (Dyck et al., 2001; Grant et al., 2005; Mavissakalian et al., 1993). Other personality features including higher neuroticism and lower extraversion have been associated with worry (Yang, Wang, Chen, & Ding, 2015). Consistent with the idea of dimensionality, higher prevalence of comorbid subthreshold disorders (versus those meeting full threshold) is common in patients with GAD (Camuri et al., 2014).

GAD and impairments in cognition. GAD has been linked not only to poor physical health, but also dysfunctional forms of cognition. Endorsement of frequent pathological worry has been linked to disturbances in cognitive control (Pretorius, Walker, & Esterhuyse, 2015), and attention control was relatively poor in GAD (MacNamara & Proudfit, 2014). GAD has also

been linked to biased information processing (toward threats) and low self-efficacy/perceived control (Barlow, 2000). Individuals with GAD may have difficulty with their ability to sustain attention to emotional stimuli (Seeley et al., 2016). It has been suggested that individuals with GAD have abnormalities in the neural tracking or processing of emotional information. Specifically, when shown video with dynamic emotional content, anxious individuals demonstrated less brain-based communication, as measured by fMRI, within emotion-processing brain regions and the default mode network, a functionally connected brain network (Carlson, Rubin & Mujica-Parodi, 2017). In other words, there is a disconnect in terms of emotional cues in the environment and an individual's response to those cues. Moreover, worriers have endorsed problematic metacognitive beliefs about worry itself (Pretorius, Walker, & Esterhuyse, 2015). Lastly, GAD has predicted poor problem orientation (Fergus, Valentiner, Wu, & McGrath, 2015), which may further compromise the individual's ability to employ adaptive strategies to manage anxiety.

GAD and impairment in role functioning. Given the foregoing cognitive impairments, it is unsurprising that GAD is associated with impairments in role functioning. Teachers have reported greater learning problems in children with GAD (Jarrett, Black, Rapport, Grills-Taquechel, & Ollendick, 2015), consistent with links of anxiety disorders (separation, social and/or generalized anxiety disorders) to academic impairment in youth (Nail et al., 2015). Adolescents experiencing GAD symptoms may have difficulty graduating high school and have a lower likelihood of attending college (Mojtabai et al., 2015), in line with findings that GAD has correlated with receiving less education (Ma et al., 2009). With regard to other roles, employment status (Ma et al., 2009; Murcia, Chastang, & Niedhammer, 2015) and income level

(Ma et al., 2009) each suggesting potential interference of GAD symptoms with success in these roles.

One of the most consistent findings is that individuals with GAD experience significant impairment in interpersonal relationships. GAD symptoms have been associated with reductions in psychosocial functioning (Hoyer, Becker, Margraf, 2002), ranging from lack of friends (Whisman, Sheldon, & Goering, 2000), to marital dissatisfaction, separation, divorce, or singlehood (Afifi, Cox, & Enns, 2006; Hunt, Issakidis, & Andrews, 2002; Whisman, Sheldon, & Goering, 2000). Social concerns have been found to be one of the most frequent worry topics in GAD and for chronic worriers (Roemer, Molina, & Borkovec, 1997). Moreover, because personality disorders involve interpersonal deficits, comorbidity of GAD with personality disorders suggests relational problems (Sanderson, Wetzler, Beck, & Betz, 1994). There is also evidence suggesting potential interpersonal causes of GAD. For example, past interpersonal trauma (i.e. physical or emotional assault, or separation due to divorce or death of a parent) (Molina, Roemer, Borkovec, & Posa, 1992; Roemer, Molina, Litz, & Borkovec, 1996; Torgerson, 1986) and family violence predict likelihood of GAD (Priest, 2015). In contrast, positive interpersonal factors such as paternal warmth may constitute protective factors (Moscati, Flint, & Kendler, 2016). However, despite robust links of interpersonal difficulties with GAD symptoms, the ways that interpersonal behaviors interact with GAD symptoms to predict distress remain poorly understood, a point to which we will return shortly.

Somatic and physiological impairments. Lastly, somatic disturbances provide further evidence of the problematic nature of GAD and worry. Health related factors and chronic illness have been noted in GAD including asthma, hypertension, high blood lipids, osteoporosis (Muhsen et al., 2008). GAD status has been commonly associated with obesity (Wiltink et a.,

2011), as well as cardiac problems, such as coronary heart disease (Barger, & Sydeman, 2005). In fact, a review by Tully, Cosh and Baune (2013) suggests that both worry and GAD status predict blood pressure and diagnosed hypertension or related medication use. Moreover, individuals with GAD may experience discomfort from somatic symptoms such as migraine (Dindo, Recober, Haddad, & Calarge, 2016) and both GAD and subthreshold GAD have been associated with headache disorders (Lucchetti et al., 2013). Other common physical symptoms include trembling, feeling shaky, soreness, muscle aches (APA, 2013), and poor sleep (Choueiry, Salamoun, Jabbour, El Osta, Hajj, & Khabbaz, 2016).

Additionally, physical symptoms of autonomic dysregulation have been implicated in GAD and worry. Chronic reductions in heart rate variability (HRV) have been noted in clinical GAD samples, and this link may be greater for GAD samples that have comorbid major depressive disorder (Chang et al., 2013). Worry has been associated with prolonged additional reductions in HRV (Verkuil, Brosschot, Tollenaar, Lane, & Thayer, 2016). In a community sample, GAD was associated with lower HRV during exposure to fearful and sad stimuli, despite comparable subjective ratings, suggesting autonomic inflexibility (Seeley et al., 2016). Following experimental induction of worry, individuals with GAD were more likely to demonstrate decreases in vagal cardiac control, poor coordination of sympathoexcitatory-HPA axis stress reactivity, and rigidity in cardiovascular reactivity (Fisher, 2015). Lastly, hyperarousal (e.g., exaggerated startle response) can suggest poor autonomic nervous system (ANS) modulation; experimental manipulations have demonstrated a significantly larger startle responses for participants with GAD as compared to controls (Ray et al., 2009).

In summary, GAD symptoms, whether at clinical or subclinical levels, are associated with significant distress and impairment across many domains and pose a public health concern

that has major health consequences and associated economic costs (Kujanpää, Ylisaukko-oja, Jokelainen, Linna, & Timonen, 2014). In the present paper, I focused particularly on specific aspects of two of those domains—social/interpersonal behaviors and biological stress responding—for which the links to GAD symptoms are less clearly understood.

GAD and the Interpersonal Domain

The interpersonal circumplex model. An in-depth examination of the interpersonal aspects of GAD symptoms requires a review of the model which most systematically conceptualizes and measures interpersonal functioning - the Interpersonal Circumplex (IPC; Wiggins, 1982). The IPC provides a structural model to assess a set of related constructs of social behavior. Interpersonal styles are enduring and generalizable individual differences of social exchange (Jordan, Masters, Hooker, Ruiz, & Smith, 2014). In other words, interpersonal styles describe how individuals communicate with others and perceive themselves in relation to others (Podubinski, Lee, Hollander, & Daffern, 2014).

The IPC model evaluates interpersonal styles as combinations of two core dimensions – *affiliation* (also referred to as *communion* or *nurturance*) and dominance (also referred to as *agency* or *control*). These dimensions make up the orthogonal horizontal and vertical axes of the circumplex. Affiliation is representative of warmth, friendliness and closeness at one extreme, and cold social behavior or distance at the other. Dominance describes an individual's power, assertion, or mastery of the self and/or the environment (Podubinski et al., 2014). Although different models may utilize different terminology for the dimensions of affiliation and dominance, they typically reflect variations on these two higher-order constructs. Assessments of these axes, depending on the measure, can describe trait-like interpersonal styles, momentary and fluctuating states of social exchange, as well as qualities of relationships (Jordan et al., 2014).

Vectors further divide the circumplex into eight octants (i.e., eight "pie" slices of circular space) displaced 45 degrees around the center. Trait ratings of these octants represent eight combinations of dominance and affiliation around the circumference of the circumplex. The length of the vector represents the magnitude of the interpersonal profile and scores plotted in the periphery are most indicative of maladaptive interpersonal styles (McCartney, Collins, Park, Larkin, & Duggan, 1999). In other words, the vector length is a measure of extremity, with longer vectors indicating a more extreme interpersonal stance and a more well-defined interpersonal profile (Gurtman, 2009). The IPC provides a framework for conceptualization and measurement of the interpersonal domain, though there are many different IPC measures that assess different phenomena in the interpersonal domain (i.e. interpersonal problems, traits, capabilities, behavior, etc.). Resultant scores from these measures are used to calculate an individual's placement on the IPC. For instance, the eight octants of interpersonal styles on the Inventory of Interpersonal Problems Circumplex Scales (IIP-C; Horowitz et al., 2000) assess dysfunctional interpersonal behaviors including being domineering, vindictive, cold, socially inhibited, nonassertive, overly accommodating, self-sacrificing, and intrusive. These scales blend dominance and affiliation dimensions, varying based on location around the circle and angular coordinates (Gurtman, 2009). From the circular patterns of correlates, the circumplex was developed such that variables sharing close conceptual content are located closely on the circumplex by angular degree (higher correlations among closer octants and lower correlations between scales as one moves around the circle up to 180°).

The IPC model additionally provides a way to conceptualize interaction dynamics. In other words, the framework models the ways in which one behaves interpersonally and the interpersonal response evoked in an interaction partner. Specifically, *complementarity* is a

concept used to describe ways in which individuals invite complementary responses from others (Locke & Sadler, 2007). Complementarity involves a pattern of similarity along the horizontal axis (i.e., warm and friendly behavior tends to invite others to be warm and friendly, whereas cold behavior pulls for cold responses). Additionally, complementarity entails opposing behaviors along the vertical axis (Locke & Sadler, 2007). For example, one's dominant behavior invites others to act submissive, and vice versa. Thus one's interpersonal behavior can elicit others' behavior and may thereby shape the stability of one's social interactions, as well as how one perceives oneself following such interactions. Dominance and affiliation serve regulatory purposes though it is unclear whether this regulation has any downstream effect on psychological and biological outcomes in individuals with GAD.

Links of GAD to specific behaviors on the IPC. In addition to the aforementioned links to interpersonal dysfunction in general, subclinical GAD (Eng & Heimberg, 2006) and clinical GAD symptoms (Przeworski et al., 2011; Salzer, Pincus, Winkelbach, Leichsenring, & Leibing, 2011) have demonstrated strong links to self-reported interpersonal problems related to dominance and affiliation dimensions on the IPC. However, the nature of the links between GAD and IPC dimensions have been inconsistent across studies.

Interpersonal problems and worry may be linked to the IPC in two different ways. The first, the interpersonal *pathoplasticity* model (Przeworski et al., 2011), suggests that various types of interpersonal problems are linked to GAD and worry. GAD symptoms may coexist with any form of interpersonal behavior (i.e., individuals with GAD symptoms have heterogenous interpersonal presentations), and each may reciprocally shape the expression of the other. It is possible that individuals with GAD have similar worry symptoms and vary in the way those symptoms manifest in interpersonal interactions (Erickson et al., 2016; Przeworski et al., 2011).

Studies supporting this perspective have found multiple interpersonal subtypes linked to GAD (Przeworski et al., 2011) as measured by the Inventory of Interpersonal Problems (IIP-C; Alden, Wiggins, & Pincus, 1990), including four prototypical interpersonal clusters: primarily nonassertive (i.e., submissive), exploitable (i.e., affiliative-submissive), cold, or intrusive (affiliation-dominant) interpersonal problems. Salzer and colleagues (2011) also found four interpersonal subtypes in GAD: overly nurturant (affiliative), intrusive, socially avoidant (cold-submissive), and nonassertive (submissive). These studies support the notion that many different interpersonal styles or problems may coexist with GAD symptoms, implying that clinicians should understand an individual's symptoms *and* social behaviors to obtain the fullest picture (and should consider how interpersonal tendencies might interact with GAD symptoms).

In contrast, the idea of *interpersonal specificity* in the context of GAD would suggest that these symptoms have a prototypical interpersonal prototype, seemingly in contrast to an idea of heterogeneity in IP subtypes. Erickson and colleagues (2016) found that after controlling for commonly coexisting symptoms of social anxiety and depression, dimensional worry predicted self-reported affiliative or warm-submissive tendencies across measures of interpersonal traits, problems, goals, and social behaviors in daily life (although significant others associated worry with cold behavior, paradoxically). Other research has found that clients with GAD demonstrated more exploitable (affiliative-submissive) and nonassertive interpersonal problems relative to those in a general clinical sample (Gomez Penedo, Constantino, Coyne, Westra, & Antony, 2017). Other relevant processes such as empathy and theory of mind allow an individual to understand the feelings and thoughts of others, implying affiliative tendencies. In an experimental study, theory of mind reasoning was more accurate among those with GAD relative to controls; moreover, compared to relaxation, worry led to higher theory of mind reasoning

accuracy among the GAD group (Zainal & Newman, 2018). Similarly, higher trait affective empathy was associated with greater anxiety, as measured by autonomic arousal, skeletal muscle effects, situational anxiety, and subjective experience of anxious affect (Powell, 2018). These effects suggest the possibility of unique links of GAD and affiliative tendencies.

Thus, research suggests both that individuals high in worry/GAD symptoms endorse a range of different types of interpersonal difficulties, but also that there may exist some specific unique links to affiliative or warm-submissive regions of the IPC. If dominant or affiliative behaviors in daily life interact with (i.e., amplify the effect of) GAD symptoms on distress, it may further elucidate whether particular IPC regions are important for understanding GAD. Conversely, if neither IPC dimension moderates the effects of GAD on subsequent experiences of stress, then it may argue against interpersonal specificity. Research linking IP domains to biological stress has the capacity to inform these issues as well, such that interpersonal behavior in daily life, should it interact with GAD symptoms on biological stress, may suggest that there are specific regions of the IPC that are particularly relevant to individuals with GAD symptoms. The present study aims to examine these questions in the context of daily interpersonal stressors, with regard to how they pertain to subjective reporting of distress as well as a biomarker of stress (i.e., cortisol).

Biological Stress Responses

The second major aim I examined was the relationship of GAD symptoms to biological stress responses. Considering the links of GAD symptoms to subjective experiences of stress (i.e., psychological distress), they also are likely to predict biological aspects of stress responses. However, despite robust associations of GAD symptoms with arousal and self-reported somatic

symptoms, the effects of GAD on biological stress responses via the body's primary stress-response system—the hypothalamic-pituitary-adrenal (HPA) axis—have been less consistent.

The various divisions and subdivisions of the nervous system, as well as other functionally related brain systems, contribute to the stress response. Once a threat is perceived, the fight-or-flight response is activated as part of the sympathetic nervous system (SNS). These changes are immediate and can occur within seconds. A host of changes occur in the body including vasodilation of blood vessels leading to parts of the brain and skeletal muscles. Heart rate and pulse rate increase, blood is sent to the muscles and vital organs, the muscles are warmed, pupils dilate, and increases in oxygen are provided to the brain. These changes facilitate behaviors such as running away from a threat or seeking shelter, and are useful for escaping threat and danger. The autonomic nervous system (ANS), controlling vegetative and reproductive functions, has inhibitory control and will withdraw its inhibitory influence (via the vagus nerve) in the presence of a perceived stressor. Brain systems related to fear- and anxiety-related behaviors include the limbic system, prefrontal cortex, ANS, SNS and the hypothalamic-pituitary-adrenal (HPA) axis. However, the following review focuses exclusively on the HPA axis and cortisol as its output, the biological variable of interest. Discussion of this point first warrants an overview of the HPA axis.

The HPA axis. The HPA axis is the primary stress response system and involves the nervous system and the endocrine system. First, a stressor is perceived (threat detection). Psychological factors related to threat, such as uncontrollable situations and social evaluative threats, trigger the HPA system (Dickerson & Kemeny, 2004). Once triggered, these networks rely on a series of signals from stress hormones. There are three main components that comprise the HPA axis. First, the hypothalamus, is a diencephalic structure located under the thalamus

(Pinel & Edwards, 1998). The perception of a stressful stimulus is linked to an integrated response in the hypothalamus, resulting in the release of corticotropin-releasing hormone, though chronic stress can influence the production of neurotransmitter in this brain structure. In fact, in an experimental study of mice, those mice exposed to chronic social stress demonstrated fewer cells positive for serotonin release, a neurotransmitter involved in regulating HPA functions, in the hypothalamus (Florez, Solano, & Cardenas Parra, 2017). The second major component of the HPA is the pituitary gland, which is suspended from the hypothalamus and is composed of two separate glands; the anterior pituitary and posterior pituitary (Pinel & Edwards, 1998). CRH, stimulated by the hypothalamus, travels to the pituitary, which triggers the release of adrenocorticotropic hormone (ACTH). The anterior pituitary releases ACTH, which travels to the adrenal glands, the third component of the HPA axis, which prompts the release of cortisol. The adrenal glands are located on top of each kidney. These modified neurons are comprised of the adrenal cortex and adrenal medulla. Under sympathetic activation, various hormones, including norepinephrine, epinephrine, cortisol, and aldosterone, amongst others, are released from the adrenal gland. Those hormones then go into the bloodstream to coordinate a stress response. To maintain hormone levels within an optimal physiological range, negative feedback regulation of hormones occurs. This results in the inhibition of tropic hormone secretion (CRH and ACTH). This cascade of hormonal signals allows for an individual to react to threat (i.e. stress or fear) however, several specific pathways and endocrine products are most central in the HPA system.

Cortisol. Cortisol is an important hormone with characteristic temporal patterns and functions. Cortisol normally follows a circadian pattern where levels are highest before awakening, slowly decline throughout the day, and are lowest near bedtime. This is a regular,

healthy pattern. However, under stress, more cortisol will be produced. There are both positive and negative effects of the stress hormone cortisol. In the short term, after a perceived stressor, cortisol takes approximately 10 -20 minutes to peak (Goodin et al., 2012; Hernandez, et al., 2014) and facilitates the individuals adapting to stressors. In fact, acute and tolerable stressors may enhance cognitive processes such as working memory in animals (Lindau, Almkvist, & Mohammed, 2016; Luo et al., 2018). Furthermore, corticosterone, at low levels, facilitates spatial learning (Lindau, Almkvist, & Mohammed, 2016; Meaney, Aitken, Van Berkel, Bhatnagar, & Sapolsky, 1988). One review (Juszczak & Stankiewicz, 2018) found links between cortisol and a series of metabolic processes that regulate energy expenditure and efficiency during a stress response. However, with continual exposure to stressors, high levels of cortisol can become problematic and result in chronic HPA activation/secretion.

Cortisol dysregulation. Evidence of cortisol dysregulation takes different forms and includes basal hypercortisolism (chronic high cortisol secretion; Andreescu et al., 2017), basal hypocortisolism (low cortisol; Heim, Ehlert, & Hellhammer, 2000), as well as problematic cortisol response to stressors (i.e. hyper-reactivity and hypo-reactivity; Skoranski et al, 2018). A dysregulation of this system in any form can have negative side effects for both mental and physical health.

Physical health. The HPA is thought to be a mediator of the effects of stressors on physical health and illness and it appears that cortisol may negatively impact various bodily systems. In fact, prolonged elevations in cortisol may be a risk factor for cardiovascular disease (CVD; Manenschijn et al., 2013). Elevated cortisol has additionally been positively associated with obesity (as measured by urine and saliva; Abraham, Rubino, Sinaii, Ramsey, & Nieman,

2013) and metabolic syndromes (as measured with urine; Vogelzangs et al, 2007). Low cortisol concentrations have been implicated in fatigue (Sudhaus, et al., 2009).

Mental health. It appears that high levels of cortisol have been implicated in panic disorder (Roy-Byrne et al., 1986), obsessive-compulsive personality disorder (Kanehisa et al., 2017), depression (Herane Vives, et al., 2015; Lamers et al., 2013; Strawbridge & Young, 2016), anorexia nervosa (Gold et al., 1986), high-arousal negative affect (Castonguay, Wrosch, & Sabiston, 2017), and cognitive dysfunction in those with Cushing's syndrome (Forget, Lacroix, Bourdeau, & Cohen, 2016). Diminished cortisol activity has been implicated in posttraumatic stress disorder (Herane Vives, et al., 2015), depressive mood (Sudhaus, et al., 2009), and vital exhaustion (Strahler & Fischer, 2018). Furthermore, cortisol responses may be blunted in anxious individuals in some cases (Skoranski et al, 2018).

Measurement of cortisol. Historically, cortisol has been measured using several methods (i.e., saliva, blood, urine, feces, and hair) and at different times of the cortisol circadian rhythm (e.g., cortisol response to awakening [CAR], diurnal cortisol, basal cortisol, free cortisol, "area under the curve" or total cortisol secretion in response to a stressor). Salivary cortisol, used for assessing adrenal functioning, is often used in psychobiological studies given its relatively non-intrusive collection. For this reason, saliva samples are useful for repeated collections, where an individual might be subjected to multiple sampling procedures. However, salivary sample concentrations are much smaller (approximately 0.1%) than those gathered from plasma samples (Hammond & Langley, 1986) and may be vulnerable to contamination. Plasma in blood samples on the other hand, requires penetration of the skin to gather the sample, which can be difficult and/or cause additional stress. Urine and feces have also been used to assess cortisol concentrations though the optimal time-frame suggested for collecting samples is within one day

of the potential stressor (Chen, Yao, Yang, Fan, & Xiang, 2017). In addition, these methods are appropriate for estimating reactivity in cortisol but do not easily provide estimates of long-term cortisol secretion unless they are aggregated across long periods of time, which is rare.

Measurement of cortisol through *hair* samples provides a novel biomarker for estimates of chronic HPA activity. Hair sampling is cost-effective, relatively non-intrusive, and generates a retrospective indicator of cortisol levels over an extended period of time. Hair cortisol levels correlate with cortisol in 24-hour urine (Sauvé, Koren, Walsh, Tokmakejian, & Van Uum, 2007). Limitations of hair cortisol are minor and include the need for cutting hair to obtain samples (which may hold cultural or spiritual meaning), and risk of decreased representation of cortisol levels in artificially colored hair (Sauvé, Koren, Walsh, Tokmakejian, & Van Uum, 2007). Though hair sampling for cortisol has its shortcomings, research procedures can account and accommodate for such influences. Previous studies have utilized hair cortisol concentrations to examine HPA activity in other disorders characterized by negative affect, including depression, PTSD (Luo et al., 2012), and other anxiety disorders. However, relatively little research has examined effects of GAD symptoms on hair cortisol levels, an aim of the proposed research. Hair cortisol measures aggregated (averaged) to assess chronic cortisol levels parallels the idea of GAD as chronic worry and chronic perceived uncontrollability. Given my interest in chronic stress and the long-term GAD symptoms (i.e., six months or longer) rather than on reactivity to particular stressors, hair cortisol represents a stress biomarker appropriate for the present study.

GAD Symptoms and Cortisol

Research on cortisol secretion in individuals with GAD has yielded inconsistent results. There is evidence that individuals with GAD and worriers experience general cortisol dysregulation (Steudte et al., 2011), while other research has yet to find such differences

(Steudte-Schmiedgen et al., 2017). Studies disagree on whether individuals with GAD symptoms producing elevated levels of cortisol (hypercortisolism) or chronically low levels of cortisol (hypocortisolism) cortisol, a point that I turn to next.

Hypocortisolism and hypercortisolism. Evidence of hypercortisolism has been noted in several reviews (Lenze et al., 2011). There is evidence that acute psychological stressors (Cinque, et al., 2017; Dickerson & Kemeny, 2004), pathological anxiety (Vreeburg et al., 2010), and GAD predicted higher salivary cortisol levels (Mantella et al., 2008). Pathological anxiety has predicted larger salivary CAR (Vreeburg et al., 2010). Additionally, compared to non-anxious controls, those with GAD demonstrated elevated basal salivary cortisol levels and greater peak cortisol levels (Mantella et al., 2008). Symptom improvement in those with GAD has been linked to changes in cortisol, such that greater improvements predicted greater decreases in salivary cortisol from the morning to the rest of the day (Keefe, Guo, Li, Amsterdam, & Mao, 2018). Other stressors one might plausibly link to GAD, such as job strain (high job demands and low job control; Steptoe, Cropley, Griffith, & Kirschbaum, 2000), job instability (Harris, Cox, Brett, Deary, & MacLullich, 2017), a training-related stress (i.e. medical internship; Mayer, Lopez-Duran, Sen, & Abelson, 2018), have been linked with elevated cortisol levels. Generally, it appears that hypercortisolism is representative of acute stress and reactivity to stressors.

Conversely, evidence of hypocortisolism has also been evaluated in the literature. For instance, in a naturalistic setting, individuals with GAD demonstrated significant hypocortisolism (i.e. 50-60% lower cortisol levels) as measured by hair cortisol (Steudte et al., 2011). In fact, this study was the only of its kind found to link GAD with cortisol levels as assessed with hair. Additionally, an inverse relationship has been indicated between GAD

symptoms and salivary cortisol in cancer patients, a population commonly experiencing chronic and profound stress (Sharpley et al., 2017). Hypocortisolism has been reported for populations experiencing chronic stress, such as older adults with chronic anxiety (Hek et al., 2013), those exposed to chronic environmental stressors (Karb, Elliott, Dowd, & Morenoff, 2012), those exposed to early life adversity (Koss, Mliner, Donzella, & Gunnar, 2016), and those with chronic fatigue syndrome (Tak et al., 2011) and fibromyalgia (Demitrak et al., 1991; Riva, Mork, Westgaard, Rø, & Lundberg, 2010).

Overall, anxiety disorders relate to dysregulated HPA-axis functioning, however findings are inconsistent in regard to hypo- or hyper-activation. A variety of factors are thought to influence HPA regulation including; gender, age (Mantella et al., 2008; Piazza, Charles, Stawski, & Almeida, 2013), the presence of a comorbid depressive disorder (Phillips et al., 2011), and stress duration. A meta-analysis (Miller, Chen & Zhou, 2007) suggests that there may be an influence of timing on the HPA response and that differences found in terms of hypercortisolism and hypocortisolism may be reflective of different time points in the stress process. Specifically, it was found that studies focusing on recent and current stress time-points found evidence of hypercortisolism, whereas those focusing on chronic stress and distant traumas documented hypocortisolism. In both acute and chronic stress experiences, individuals must learn to cope in response to external and internal demands. One explanation may involve the concept of allostasis.

Allostasis and allostatic load. By definition, allostasis refers to stability through change. McEwen's model (2004) suggests that in response to a stressor, an allostatic response increases arousal (via biological stress systems) and when the threat has been eliminated, the allostatic response is halted to reduce overload of the system. Though this accommodation is adaptive and

necessary for survival, it can also lead to allostatic load - damage and exhaustion of the system. This model posits that both overactivity and inactivity of related physiological systems effectively lead to allostatic load on those regulatory systems. Some research suggests including a curvilinear association may be most appropriate for analyses of allostatic load (i.e., testing for both low and high levels of stress biomarkers such as cortisol, as linked to the variable in question; Bush, Obradović, Adler, & Boyce, 2011). However, some studies testing for quadratic relationships with cortisol have found better fit for linear relationships (Bellingrath, Weigl, & Kudielka, 2009).

Altogether, GAD symptoms may predict high or low hair cortisol. Though links of GAD to cortisol are inconsistent, one possible explanation may be the interpersonal heterogeneity of those with GAD symptoms, given that IPC dimensions of dominance and affiliation are of relevance to HPA responses/cortisol, a point to which I turn next.

Interpersonal Behavior and Cortisol

Interpersonal constructs have been linked to not only psychological outcomes (as discussed above), but also biological stress markers. Interpersonal behaviors and their ability to elicit emotions and interpersonal behaviors from others are likely implicated in HPA activity and cortisol concentrations.

Dominance. It has been suggested that dominance provides an individual the allowances necessary to establish security and stability in the animal hierarchy. Furthermore, the idea that *low* dominance predicts *elevated* cortisol secretion aligns nicely with the previous review suggesting dominance serves adaptive purposes. It may be that submissive IP behaviors hinder resource acquisition/perception, and in turn, reduce an individuals' ability to survive or succeed. In fact, dominance appears to be closely correlated to social skills displays (Burgoon & Dunbar,

2000), which may facilitate resource acquisition. Submissive IP behaviors have been linked to internalizing problems in adolescents (Powers, Battle, Dorta, & Welsh, 2010), brooding, an interpersonal component reflecting submissive IP behaviors, and future psychopathology, such as depression (Pearson, Watkins, & Mullan, 2010). Moreover, there are poor adaptive outcomes for individuals that are victimized (Ladd, Kochenderfer, & Coleman, 1997), a construct related to submission. Given these links, it would not be surprising for submissive individuals to demonstrate high cortisol levels and likewise, for dominant individuals to demonstrate low levels. Dominance in non-human primates has most often been associated with lower levels of cortisol compared to subordinates (Golub, Sassenrath, & Goo, 1979) and subordinate cynomolgus monkeys hypersecreted cortisol in an experimental study (Shively, Laber-Laird, & Anton, 1997). In humans, dominant IP styles might include cognitive or emotional states such as pride (Williams & DeSteno, 2009), effective persuasion (Hare, Kritzer, & Blumberg, 1979), mastery, and confidence. Mastery, a construct related to dominance, was negatively associated with diurnal cortisol slope as measured by salivary cortisol (Cohen et al., 2006). Additionally, a dominant and confident interpersonal orientation may attenuate anticipatory cortisol responses as measured by saliva (Turan, 2015). There is also evidence that dominance is most pronounced in individuals with low baseline salivary cortisol levels (and high baseline testosterone; Mehta & Josephs, 2010).

Other research has found that opposite relationship —that greater dominance sometimes predicts greater cortisol levels. For instance, in male cynomolgus macaques, cortisol was higher in dominant compared to intermediate and subordinate monkeys in low stress conditions (Jimenez, Allen, McClintick, & Grant, 2017). Individuals in positions of higher social rank, such as executives, demonstrated a higher salivary response relative to those at lower hierarchical

positions (Guedes, Gonçalves, Gonçalves, & Patel, 2017). Moreover, those executives lower in the hierarchy who had a higher sense of control had higher cortisol levels. Furthermore, dominant children displayed higher cortisol levels (compared to more submissive children) at the beginning of the academic school year (Gunnar, 1994). There is also evidence that cortisol and testosterone, a hormone that can influence socially dominant behaviors (Schaal, Tremblay, Soussignan, & Susman, 1996; Tremblay et al., 1998), are positively associated (Deuter, Schächinger, Best, & Neumann, 2016; Turan, Tackett, Lechtreck, & Browning, 2015). It may be that having resources and mastery is protective, but that potential competitive *threats to one's dominance* can elevate cortisol. For instance, competitive tennis players demonstrated increased cortisol levels on competition day relative to the resting day (Lautenbach, Laborde, Klämpfl, & Achtzehn, 2015). Furthermore, elite basketball players demonstrated increased cortisol levels (measured via saliva) compared to those playing easy and medium opponent teams (Arruda, Aoki, Paludo, & Moreira, 2017). Many of these studies examine cortisol responses to specific time-limited stressors, in which higher cortisol reflects adaptive responses. However, many of the studies in non-human primates and humans suggests that individuals who *chronically* employ dominant social behaviors are likely to have lower cortisol levels, implying lower perceived threat to one's social status and resources.

Affiliation. Many studies have examined cortisol levels as they relate to constructs of relevance to the horizontal axis of affiliation, such as social support, closeness, acceptance, and compassion. Most often, perceived affiliation predicts lower cortisol levels. It appears that closeness is a protective factor for individuals. High levels of affiliation are significantly related to the provision of emotional support (Fritz, Nagurney & Helgeson, 2003) and social support has been linked to lower HPA activity (as measured by salivary cortisol) during exposure to social

stressors (Sladek, Doane, Jewell, & Luecken, 2017). In fact, affiliative motives assessed pre-and-post exposure to acute stressors were linked to lower salivary cortisol responses to psychosocial stressors, but not physical stressors (Wegner, Schüler, & Budde, 2014). Intimacy has been associated with significantly lower levels of salivary cortisol secretion in adults (Ditzen, Hoppmann, & Klumb, 2008) and adaptive coping aimed at support seeking was associated with lower overall levels of salivary cortisol in older adults (O'Donnell, Badrick, Kumari, & Steptoe, 2008). Compassion (Cosley, McCoy, Saslow, & Epel, 2010) and connectedness (Papp, Pendry, Simon, & Adam, 2013) have also been associated with lower salivary cortisol reactivity. There may be gender differences in the relationship between affiliation and cortisol levels. In fact, in an interaction task, male participants with high cortisol responses to a stressor showed significantly higher ratings of psychological closeness to their interaction partner (Berger, Heinrichs, von Dawans, Way & Chen, 2016). Given that high levels of unmitigated affiliation (e.g., intrusiveness) predict greater negative interactions (Fritz, Nagurney & Helgeson, 2003), it is possible that higher affiliation may sometimes predict higher cortisol levels or responses to stressors, but overall the extant findings suggest that affiliative social processes more often predict lower chronic cortisol levels.

Additionally, interpersonal tendencies that blend dominance and affiliation have been linked to cortisol. For instance, aggressive and hostile behaviors often involve both high dominance and low affiliation. A negative relationship between hostility and cortisol output following a stress task has been indicated in populations with diabetes (Hackett, Lazzarino, Carvalho, Hamer, & Steptoe, 2015). In another study, greater self-reported aggression predicted less cortisol levels (Sariñana-González, Romero-Martínez, & Moya-Albiol, 2015). Other mental health disorders and traits that are often characterized by cold-dominant behaviors have also

been inversely related to cortisol output, including oppositional-defiant disorder (ODD; Schoorl, van Rijn, de Wied, van Goozen, & Swaab, 2016), psychopathic traits (Johnson, Mikolajewski, Shirtcliff, Eckel, & Taylor, 2015; O'Leary, Taylor, & Eckel, 2010), callous-unemotional traits (Loney, Butler, Lima, Counts, & Eckel, 2006), conduct disorder (Oosterlaan, Geurts, Knol, & Sergeant, 2005), and antisocial personality (Fishbein, Dax, Lozovsky, & Jaffe, 1992). It is important to note that many of these characteristics are a blend of the two IPC dimensions (i.e., higher dominance and lower affiliation), suggesting the possibility that these two dimensions might interact in predicting cortisol outcomes.

The Present Study and Hypotheses

This present study tested the prospective effects of GAD symptoms on both psychological and biological stress, and the effects of interpersonal behaviors on these pathways. Given the literature on cortisol levels in the context of interpersonal behavior, dominance and affiliation may represent adaptive strengths in that those who regularly rely on them to cope with stressors might experience more adaptive psychological and physical outcomes. In fact, affiliation and dominance have predicted better relationships and health outcomes (Helgeson, & Palladino, 2012) and those with high psychological well-being demonstrate lower total cortisol output (Lindfors & Lundberg, 2002). Previously reviewed work demonstrates that interpersonal styles are linked to psychological and biological distress, but the ways in which GAD symptoms and interpersonal behavior in response to stressors might interact to predict stress-related outcomes remains an unresolved question. The present study examined these questions in a prospective study over five weeks of interpersonal stressors.

Main Effects Hypotheses Predicting Emotional Distress

Given previous literature suggesting individuals with GAD and worry have greater negative emotionality (Chambers, Power, & Durham, 2004; Erickson & Newman, 2007; Salters-

Pedneault et al., 2007), I hypothesized that GAD symptoms at baseline would prospectively predict higher chronic emotional distress in the context of psychosocial stressors in daily life (hypothesis 1). Additionally, based on research suggesting that dominant (Burgoon & Dunbar, 2000) and affiliative (Fritz, Nagurney & Helgeson, 2003) IP behaviors serve adaptive purposes, I expected that chronically higher (aggregated across situations), as well as higher person-centered (situational fluctuations in) dominance and affiliation in daily life would predict lower emotional distress in daily life (hypothesis 2a & 2b).

Hypothesis 1. GAD symptoms at baseline will prospectively predict higher chronic psychological distress in daily life.

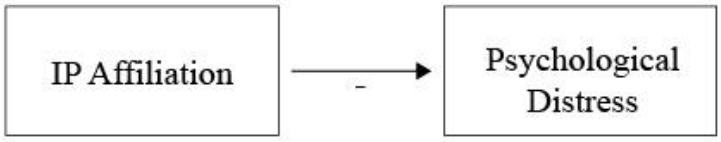

Figure 1. Conceptual model of hypothesized relationship between GAD symptoms and psychological distress.

Hypothesis 2 (a & b). The IP style of (a) dominance will predict lower chronic distress (Figure 2) in daily life and the IP style of (b) affiliation will predict lower chronic distress in daily life (Figure 3). I assessed both mean/chronic levels of dominance and affiliation, as well as within-person fluctuations. With few exceptions (e.g., Erickson et al., 2016), previous studies have not used within-person methodology to explore interpersonal processes associated with GAD symptoms. I included both because they represent unique sources of variance (e.g., between- and within-person) in interpersonal dimensions.

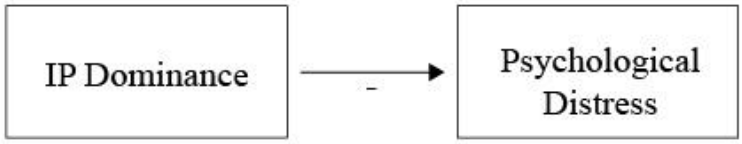

Figure 2. Conceptual model of hypothesized relationship between interpersonal dominance and psychological distress.

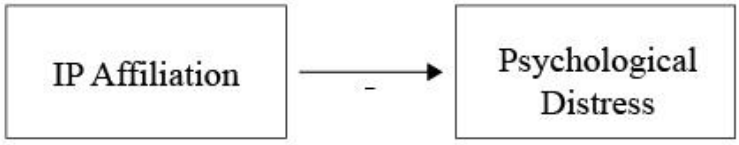

Figure 3. Conceptual model of hypothesized relationship between interpersonal affiliation and psychological distress.

Main Effects of Hypotheses Predicting Biological Stress

In parallel, I hypothesized that GAD symptoms at baseline would prospectively predict a higher chronic cortisol secreted in hair (assessed 4-5 weeks after baseline; hypothesis 3), given the many studies linking perceived stress and anxiety to higher cortisol. However, because GAD symptoms have been conceptualized as a marker of chronic stress, and given one small study finding low hair cortisol in GAD (Steudte et al., 2011), hypocortisolism in those with GAD symptoms is possible. Therefore, I tested for curvilinear effects given previous findings suggesting that GAD is associated with both chronically low and high cortisol levels (Keefe, Guo, Li, Amsterdam, & Mao, 2018; Sharpley et al., 2017; Steudte et al., 2011; Vreeburg et al., 2010). Additionally, based on research suggesting that dominant (Burgoon & Dunbar, 2000) and affiliative (Fritz, Nagurney & Helgeson, 2003) IP behaviors serve adaptive purposes, I expected

that chronically higher (aggregated across situations) dominance and affiliation in daily life would predict low level cortisol secretion (hypothesis 4 a & b).

Hypothesis 3. GAD symptoms at baseline will prospectively predict high cortisol output.

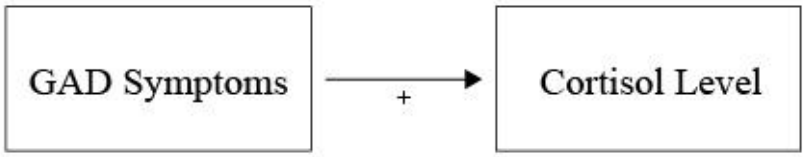

Figure 4. Conceptual model of hypothesized relationship between GAD symptoms and cortisol level.

Hypothesis 4 (a & b). The IP style of (a) dominance will predict low levels of cortisol secretion (Figure 5) and IP style of (b) affiliation will predict low levels of cortisol (Figure 6).

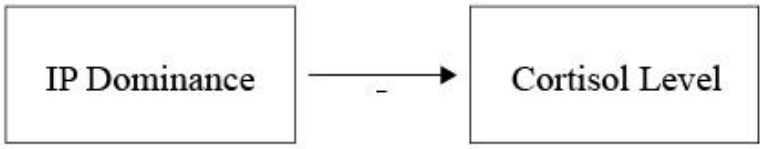

Figure 5. Conceptual model of hypothesized relationship between interpersonal dominance and cortisol level.

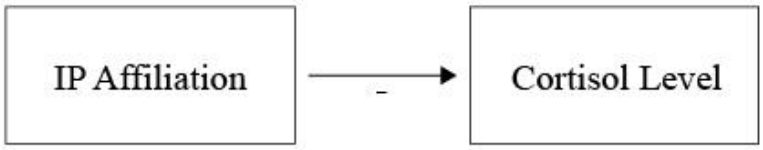

Figure 6. Conceptual model of hypothesized relationship between interpersonal affiliation and cortisol level.

Interaction Hypotheses

The question remains whether that relationship depends on IP styles. In chronically anxious people, it may be that interpersonal styles amplify (or blunt) negative affect (hypothesis 5 a & b) and cortisol secretion (hypothesis 6 a & b). Given the adaptive importance of dominance and affiliation, it is expected that the relationship between GAD symptoms and distress will be blunted by use of these adaptive interpersonal behaviors. Specifically, higher levels of dominance are expected to buffer against the positive effect of GAD symptoms on distress. Additionally, higher levels of affiliation are predicted to buffer the effect of GAD symptoms on distress. Based on evidence that dominant (Burgoon & Dunbar, 2000) and affiliative (Fritz, Nagurney & Helgeson, 2003) social behaviors serve adaptive purposes, I hypothesized that higher dominance and affiliation, as well as the combination (hypothesis 7 a & b) of high dominance and affiliation, would moderate (buffer or decrease) the extent to which baseline GAD symptoms predict higher distress and cortisol dysregulation.

Hypothesis 5 (a & b). High levels of the IP style of (a) dominance and (b) affiliation (at both mean levels and situational fluctuations) will blunt the positive relationship between GAD symptoms and psychological distress (Figure 7 & Figure 8).

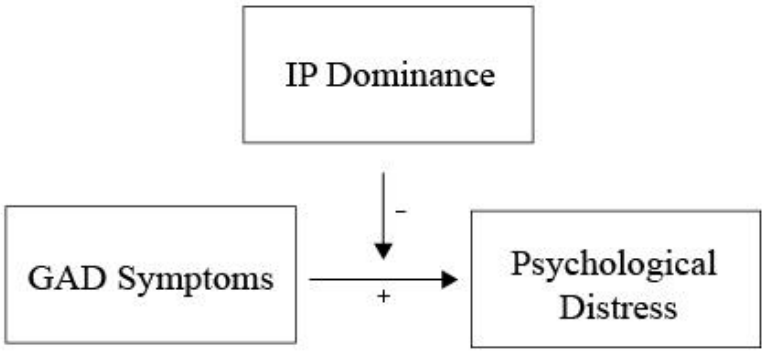

Figure 7. Conceptual model of hypothesized relationship between interpersonal dominance, GAD symptoms and psychological distress.

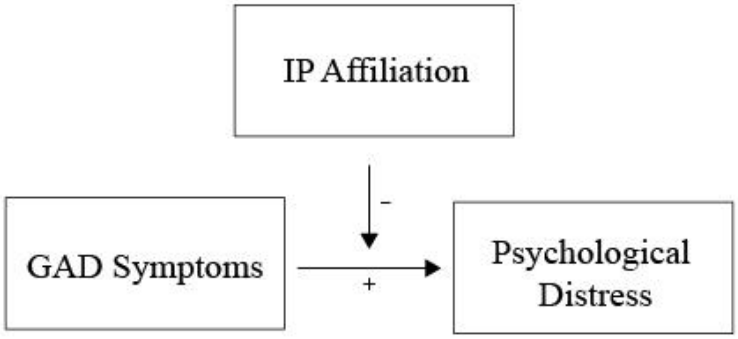

Figure 8. Conceptual model of hypothesized relationship between interpersonal affiliation, GAD symptoms and psychological distress.

Hypothesis 6 (a & b). High levels of the IP style of (a) dominance will blunt the positive relationship between GAD symptoms and cortisol level (Figure 9) and higher levels of the IP style of (b) affiliation will buffer against the relationship between GAD symptoms and cortisol level (Figure 10).

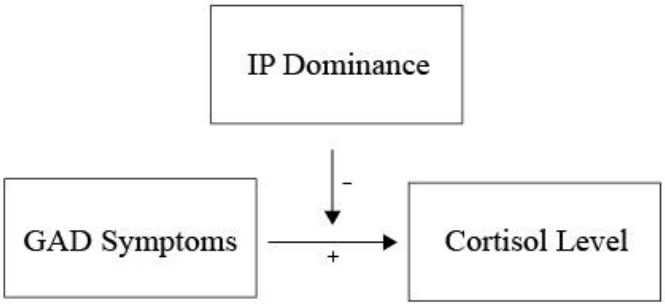

Figure 9. Conceptual model of hypothesized relationship between interpersonal dominance, GAD symptoms and cortisol level.

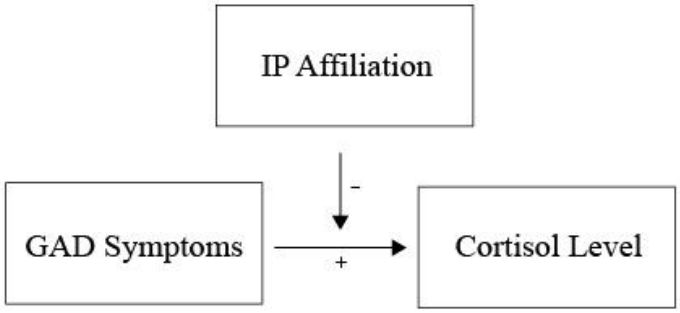

Figure 10. Conceptual model of hypothesized relationship between interpersonal affiliation, GAD symptoms and cortisol level.

Hypothesis 7 (a & b). The combination of both higher dominance and higher affiliation will blunt the relationship between (a) GAD symptoms and emotional distress (Figure 11) and (b) GAD symptoms and cortisol level (Figure 12).

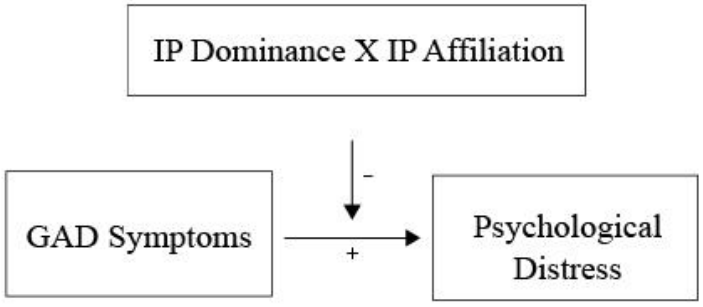

Figure 11. Conceptual model of hypothesized relationship between IP affiliation, IP dominance, GAD symptoms and psychological distress.

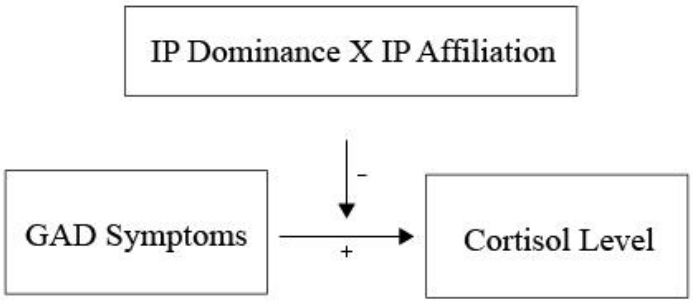

Figure 12. Conceptual model of hypothesized relationship between IP affiliation, IP dominance, GAD symptoms and cortisol level.

CHAPTER II

Method

Participants

Participants included 152 undergraduate students (116 females, 36 males) enrolled in general psychology courses at a private university in the Pacific Northwest. Phase two included 77 women and 13 men that completed the lab visit to collect hair sample for assaying cortisol. Individuals participating in this study ranged in age from 18 - 31 (M = 19.51, SD = 2.09). *A priori*, I retained data from participants who completed at least three diaries (excluding 10 participants). The average number of diaries completed was 13.54 (SD = 2.55). Students described themselves as Caucasian (50%), Asian American (24.3%), or African American (3.9%), Latinx/Hispanic (9.2%) multiracial (7.9%), or other (4.6%). Students recruited via the psychology research subject pool were invited to participate in the study and earned course credit, as well as $15 gift card for providing a hair sample from which cortisol was extracted by the end of the quarter.

Procedure and Design

I utilized a prospective diary method over five weeks of interpersonal stressors. Following recruitment procedures, consenting participants were directed to the online survey to complete baseline measures. All participants completing the baseline assessment were eligible for Phase 2, consisting of experience sampling surveys in which participants were sent (via email) links to daily brief online surveys about stressors and responses to them. These brief surveys were administered via Qualtrics for three days per week (Tuesday, Thursdays, Saturdays) for 5 weeks (5 minutes x 15 days). After completing Phase 2, participants were invited to the lab space (Watson building, B53) to provide a hair sample and participate in an interview about anxiety and depression symptoms (unrelated to the present study). Students were excluded from the last phase of the study if the hair provided did not meet the length requirement (2-3 cm).

Measures

Self-report measures assessed at baseline (phase 1).

Generalized anxiety symptoms. The Generalized Anxiety Disorder Questionnaire IV (GAD-Q-IV; Newman et al., 2002) is a 9-item self-report instrument commonly used to screen for the presence of GAD, as delineated in the Diagnostic and Statistical Manual of Mental Disorders (DSM-IV; American Psychiatric Association, 1994). The measure consists of five 1 (*yes*) and 0 (*no*) items used to assess the presence of frequent, excessive, and uncontrollable worry causing respondent distress. Additionally, six dichotomous items are used to evaluate associated anxiety symptoms (restlessness, fatigue, sleep disturbance, irritability, muscle tension, and difficulty concentrating). Finally, one free-response item prompts respondents to identify frequent worry topics (1 point awarded per worry, with a maximum of 6 points). Levels of distress and impairment are evaluated using two items, each completed on a 9-point Likert scale

from 0 (*none*) to 8 (*very severe*). A dimensional composite was generated by coding, weighting, and summing item responses. Specifically, scoring instructions require that those dichotomous items are either coded 1 or 0; the number of worries is divided by 3; the number of anxiety symptoms is divided by 2; and responses to impairment and distress items are divided by 4. The GAD-Q-IV has demonstrated internal consistency ($\alpha = .75$ in our sample), convergent and divergent validity, and good sensitivity and specificity in predicting GAD diagnosis (Newman et al., 2002; Turk, Heimberg, Luterek, Mennin & Fresco, 2005). Additionally, the GAD-Q-IV has demonstrated utility as both a dichotomous screening measure to assess risk of GAD, as well as a continuous measure (Moore, Anderson, Barnes, Haigh, & Fresco, 2014; Newman et al., 2002) with unifactorial structure (Rodebaugh, Holaway, & Heimberg, 2008).

Repeated measures assessed over 5 weeks (phase 2).

Interpersonal self-perceptions. Interpersonal self-perceptions were measured along dimensions of affiliation and dominance. Participants were asked about their worst interpersonal stressor in the past two days and instructed to report on their perceptions of the other person (this was not examined for the current analysis). Participants were prompted to rate how they viewed themselves in response to the other individuals' behavior during the social interaction. In response, participants rated single items to assess each interpersonal dimension. Items included: "As a result of how you responded in the situation, to what extent did you see yourself as assertive, dominant, or powerful" to represent dominance and "As a result of how you responded in the situation, to what extent did you see yourself as social, outgoing, or close to others" to assess affiliation. Each item was ranked on a 5-item Likert scale from 0 (*not at all*) to 4 (*very much*) to evaluate the degree an individual viewed themselves as interpersonally dominant and

interpersonally affiliative. Guttman split-half correlations across the diary ratings suggested reliability for self-perceptions of dominance (.87) and affiliation (.91).

Psychological stress. A shortened Profile of Mood States (POMS; Lorr & McNair, 1971) was used to assess emotional distress, including 6 items that evaluate levels of anxiety and sadness. Items for anxiety included, “on edge,” “anxious,” and “nervous.” Items for sadness included, "sad," "discouraged," and “hopeless.” Participants ranked their affect during a stressful social interaction using a 5-point Likert scale from 0 (*not at all)* to 4 (*extremely*). We averaged all items to create a composite measure of distress, given that individuals at risk for GAD commonly experience not only anxiety but also dysphoria. Previous research suggests that the POMS short form has demonstrated good internal consistency (i.e., $\alpha = .84$ for both anxiety and depressive states; Gawrysiak et al., 2016). In the current sample, the mean and standard deviation for Cronbach’s alpha across time points were .83 and .03, respectively.

Lab visit to collect hair sample for assaying cortisol (phase 3).

Cortisol. Human hair was collected during the final phase within a designated lab space. Researchers practiced cutting hair samples on a mannequin head before taking hair samples from live participants. Researchers used gloves while handling sample. Hair samples were up to a “coffee-straw” width in diameter and 2-3 cm long. The sampling area was from the posterior vertex of the skull. The hair to be sampled was first secured with a rubber band or clip. The hair was cut, using sterilized scissors, as close to the scalp as possible without nicking the skin. Once cut, the hair sample was placed in an aluminum foil pouch and a clean paper envelope, per instructions of the University of Washington nursing research laboratory, where assays were processed. When placing the sample in the foil and envelope, researchers marked which cut end was proximal to the scalp. Hair samples are much more stable than saliva or plasma, and

therefore remain viable for over one week without refrigeration. We delivered samples within several days to the assay lab, where they were kept at 50 degrees Fahrenheit until processed. Ninety individuals completed this phase of the study.

Sample Size, Power, and Precision

Based upon existing literature linking GAD symptoms to distress and cortisol, I expected medium-sized effects (~.30) for the paths between GAD symptoms, interpersonal styles, psychological distress, and hair cortisol, suggesting the need for around 90 participants if power of .80 and alpha of .05 was assumed.

Analysis Plan

Preliminary analyses. Prior to analyzing data, data were screened to assess assumptions about skew, kurtosis, and linearity. Additionally, I planned to screen for nonlinear relationships between predictors and outcome variables (e.g., it is possible that high GAD symptoms might predict both low and high levels of cortisol). I planned to screen for covariates that may influence cortisol output (e.g., smoking, alcohol, medication use, oral contraceptives in women, and women's menstrual phase).

Core analyses. Both predictors (GAD symptoms, interpersonal dimensions) and outcomes (psychological distress and cortisol level) were modeled as continuous variables. Multilevel modeling (MLM) and multiple regression were utilized to test core hypotheses. MLM was used to test effects of GAD and interpersonal self-perceptions on downstream distress. MLM simultaneously assesses relationships within- and between- hierarchical levels and accounts for the shared variance in hierarchically structured data (e.g., with "level 1" repeated measures such as distress nested within participants at "level 2"). I first tested an unconditional model to ensure that MLM was appropriate (i.e., significant variance in intercepts, justifying

random intercepts). I planned to use MLM for testing effects of GAD symptoms (level 2) predicting distress (level 1), with interpersonal self-perceptions as predictor and moderator as well (level 1). Baseline GAD was grand mean centered (rescored as deviation from the total sample, because I am interested in differences between people). Interpersonal self-perceptions of dominance and affiliation were split into aggregated mean levels (i.e., chronic levels) as well as person-centered predictors (deviation of each diary score from the person's mean level across time) to distinguish between-person from within-person variability. I also tested the three-way interaction of GAD times aggregated interpersonal dimensions and GAD times person-centered interpersonal dimensions. Also, MLM analyses assumed an autoregressive (AR1) covariance structure for our time series data; this assumes that observations closest in time (within each person) are most highly correlated, as is most often the case in this type of data. All effects predicting distress were presented simultaneously in a single model.

For analyses with cortisol as the outcome variable, I used multiple regression (PROCESS macro) because MLM does not typically handle level 2 variables as outcomes (and cortisol was not a repeated measure). Here I tested baseline GAD symptoms, aggregated (average) dominance and affiliation, and their interactions with GAD symptoms as predictors of cortisol levels. All effects predicting cortisol were presented simultaneously in a single model.

CHAPTER III

Results

Preliminary Analyses

Data was entered and analyzed using the Statistical Package for the Social Sciences (SPSS) Version 26. Prior to analysis, all data were examined for validity concerns including

patterns of missingness and outliers, as well as assumptions of normality (e.g., skew and kurtosis). Univariate skew ranges fell within normal range (-0.02 to 1.35, and -0.91 to 1.40, respectively). Variable scatterplots did not indicate any nonlinear relationships. Table 2 provides means and standard deviations for relevant variables. Missing data screening indicated 8.52% of missing data; analyses were conducted on raw data without imputation. Tests of unconditional models suggested that, for all diary variables, there was significant variance in intercepts and slopes, suggesting the appropriateness of modeling random effects in the model. I note that women scored lower than men on affiliation after stressful interactions (see Table 1); however, analyses were conducted with gender as a covariate but because this did not substantially change the pattern of results, I report results here without gender controlled. Incidentally, I also ran analyses to determine if there was a curvilinear relationship between GAD and cortisol level and found no evidence of this effect; therefore, models presented assumed linear relationships.

Table 1.
Correlations Between Covariates

Variable	1	2	3	4	5	6
1. Female Gender	--					
2. IP Dominance	-.15	--				
3. IP Affiliation	-.31**	.50**	--			
4. GAD-Q-IV	.12	-.05	.07	--		
5. Cortisol	.10	.22*	.02	.18†	--	
6. Psychological Distress (POMS)	.03	.08	.05	.42**	.15	--

Note. Correlations for aggregated, or mean/chronic scores, for IP variables and distress. †$p < .10$, *$p < .05$, ** $p < .01$.

Table 2.
Means, Standard Deviations and Reliability Estimates

Variable	Range		*M*	*SD*
	Min	Max		
GAD-Q-IV	0	12.75	6.27	2.98
Interpersonal Dominance	0	4	1.12	1.13
Interpersonal Affiliation	0	4	1.34	1.23
Psychological Distress (POMS)	0	4	1.71	.93
Hair Cortisol	2.25	111.49	28.71	23.87

Note. GAD-Q-IV = Generalized Anxiety Disorder Questionnaire for DSM-IV; POMS = Profile of Mood States; Hair Cortisol in picograms per milligram (pg/mg)

Multilevel Modeling for Psychological Distress Outcome. I had hypothesized that GAD symptoms at baseline would prospectively predict higher chronic distress in daily life (hypothesis 1). As expected, multilevel modeling analyses indicated that GAD symptoms predicted higher chronic psychological distress in the context of stressors (see Table 3). I hypothesized that self-perceived dominance (hypothesis 2a) and affiliation (hypothesis 2b) would predict lower chronic distress in daily life. In this step of the model, I conducted multilevel modeling analyses for main effects of interpersonal dimensions predicting chronic distress, examining both person means (chronic levels) and within-person state fluctuations above those mean levels (see Table 3). High mean dominance significantly predicted higher distress in daily life, counter to expectations. However person-centered dominance, or within-person fluctuations, did not. As hypothesized, lower person-centered affiliation predicted distress in daily life, although lower mean affiliation did not. In other words, chronic levels of affiliation were not related to distress but in situations in which participants perceived themselves as more affiliative relative to their own average levels, they endorsed lower distress.

I also had hypothesized that high levels of dominance (hypothesis 5a) and affiliation (hypothesis 5b) would blunt or buffer the positive relationship between GAD symptoms and subsequent psychological distress. However, multilevel modeling analyses yielded no significant interactions for dominance (both within person and mean levels) interacting with GAD symptoms to predict emotional distress (see Table 3), contrary to hypotheses. For affiliation, mean levels of affiliation did not interact with GAD symptoms. However, within person levels of affiliation interacted significantly with GAD symptoms in predicting emotional distress, as expected. Specifically, the negative relationship between situational affiliation and distress was even lower for individuals with high GAD symptoms (see Table 3). Simple slope tests showed that GAD symptoms positively predicted downstream situational distress when participants left interactions feeling less affiliative than their typical levels (b = .13, SE = .04, p = .002), but this effect was slightly weaker when individuals felt more affiliative than usual (b = .10, SE = .02, p < .001), as expected.

Lastly, I had hypothesized that the combination of both higher dominance and higher affiliation would blunt the relationship between GAD symptoms and emotional distress (hypothesis 7a). I ran the multilevel model with the three-way interaction; however, the three-way interaction of dominance, affiliation, and GAD symptoms did not obtain statistical significance (for mean-level and person-centered interpersonal variables), therefore it was dropped from the model. The final model included only two-way interactions.

Table 3.
Parameter Estimates for Moderated Multiple Regression Model Predicting Psychological Distress

Outcome Variable	Predictor Variable	*B*	*SE*	95% CI	*p*	*pr*
Distress (POMS)	GAD-Q-IV	.13	.03	.07, .19	**<.001**	**.31**
	Mean IP Dom	.18	.08	.01, .34	**.038**	**.17**
	Mean IP Affil	.02	.07	-.13, .17	.788	.02
	Person-Centered IP Dom	-.04	.03	-.09, .01	.144	-.14
	Person-Centered IP Affil	-.08	.02	-.13, -.03	**.001**	**-.27**
	GAD-Q-IV*Mean IP Dom	-.01	.03	-.07, .04	.710	-.03
	GAD-Q-IV*Mean IP Affil	-.01	.03	-.06, .04	.678	-.03
	GAD-Q-IV*Person-Centered IP Dom	.00	.01	-.02, .02	.923	.01
	GAD-Q-IV*Person-Centered IP Affil	-.02	.01	-.03, -.00	**.035**	**-.18**

Note. GAD-Q-IV = Generalized Anxiety Disorder Questionnaire for DSM-IV; IP Dom= Interpersonal Dominance; IP Affil= Interpersonal Affiliation; POMS= Profile of Mood States; Cortisol= hair cortisol level. *pr* = partial correlation

Regression Analyses for Biological Stress Outcome. GAD symptoms at baseline were hypothesized to prospectively predict high levels of cortisol (hypothesis 3; see Table 4). GAD symptoms did predict higher cortisol as expected, when controlling for other variables in the model, such that for every one unit increase in GAD symptoms, there was a 2.05 unit increase in cortisol. I had hypothesized that the IP style of dominance (hypothesis 4a) and affiliation (hypothesis 4b) would predict low levels of cortisol. Contrary to hypotheses, regression analyses indicated that viewing oneself as dominant predicted higher cortisol levels (see Table 4) such that, for every one unit increase in viewing oneself as dominant following stressors, there was a 11.19 unit increase in cortisol. Viewing oneself as affiliative was not a significant predictor of cortisol release.

Several effects tested the hypotheses that high levels of dominance (hypothesis 6a) and affiliation (hypothesis 6b) would separately blunt the relationship between GAD symptoms and cortisol level. Analyses indicated that the interaction of GAD symptoms and viewing oneself as dominant was marginally significant in predicting cortisol, with the positive coefficient meaning that higher chronic dominance amplified or strengthened the effect of GAD symptoms on cortisol (see Table 4; we did not unpack this effect, given the marginal three-way interaction described below). The interaction of GAD symptoms and viewing oneself as affiliative was not significant, counter to hypotheses.

Lastly, I had hypothesized that the combination of both higher dominance and higher affiliation would blunt the relationship between GAD symptoms and cortisol level (i.e., 3-way interaction; hypothesis 7b). Analyses yielded marginally significant results for the interaction of GAD symptoms and the combination of viewing oneself as dominant and viewing oneself as affiliative predicting cortisol (see Table 4), suggesting a pattern of findings departing from expectations. Simple slopes analysis (to better understand this trend) revealed that at high levels of viewing oneself as dominant and low levels of viewing oneself as affiliative, every unit increase in GAD symptoms increased cortisol by 5.88 (b = 5.88, SE = 2.50, p = .021). In contrast, GAD symptoms did not predict hair cortisol in individuals chronically low in dominance and low in affiliation (b = -1.12, SE = 1.46, p = .445), low in dominance and high in affiliation (b = 1.54, SE = 2.83, p = .588), or high in dominance and high in affiliation (b = 1.91, SE = 1.61, p = .112). This suggests that although GAD symptoms predicted higher cortisol overall, those with the combination of high GAD symptoms and chronically leaving stressful interactions seeing themselves as dominant and cold were most prone to high cortisol.

Table 4.
Parameter Estimates for Model Predicting Biological Stress (Hair Cortisol)

Outcome Variable	Predictor Variable	*B*	*SE*	95% CI	*p*
Hair Cortisol	GAD-Q-IV	2.05	1.01	.04, 4.06	**.045**
	IP Dom	11.19	4.76	1.72, 20.66	**.021**
	IP Affil	-2.77	4.18	-11.08, 5.54	.509
	GAD-Q-IV*IP Dom	3.01	1.72	-.40, 6.43	.083
	GAD-Q-IV*IP Affil	-.47	1.69	-3.84, 2.89	.781
	IP Dom*IP Affil	-6.06	6.56	-19.10, 6.99	.358
	GAD-Q-IV*IP Affil*IP Dom	-3.88	2.25	-8.36, .60	.089

Note. GAD-Q-IV = Generalized Anxiety Disorder Questionnaire for DSM-IV; IP Dom= Interpersonal Dominance; IP Affil= Interpersonal Affiliation; both interpersonal variables reflected aggregated or person mean levels across stressors.

CHAPTER V

Discussion

This study tested the unique and interactive effects of GAD symptoms and interpersonal perceptions in predicting psychological and biological markers of stress (hair cortisol specifically). The hypotheses of the present study were partially supported.

Interpretation of Findings

Predicting emotional distress. Results indicating that GAD symptoms predicted higher chronic emotional distress were in line with expectations. GAD sample populations were found to have greater levels of negative emotional outcomes, including sad affect (Erickson & Newman, 2007), trait depression (Chambers et al., 2004), and emotion regulation deficits (Salters-Pedneault et al., 2006). However, whereas many studies of GAD symptoms have involved cross-sectional data or one-time laboratory visits, this study utilized a prospective

design, showing that GAD symptoms at baseline predicted downstream risk for distress experienced in the context of naturalistic psychosocial stressors. This fits with other experience-sampling studies (Crouch, Lewis, Erickson, & Newman, 2017; Newman et al., 2019).

I conceptualized affiliative tendencies as a protective factor against distress in daily life. In fact, high levels of affiliation are related to the provision of emotional support (Fritz, Nagurney & Helgeson, 2003) and social supports have been found to be protective factors for depression (Kaufman et al., 2006) and anxiety (Roohafza et al., 2014). Additionally, greater parental warmth reduced an adolescents' psychopathology symptoms, specifically anxiety and depression symptoms (Quach et al., 2015). In the present study, person-centered affiliation predicted lower distress in daily life, however mean levels of affiliation did not. In other words, those day-to-day affiliative fluctuations that deviate from one's average levels of affiliation were significantly related to lower levels of self-reported distress. It may be that those day-to-day fluctuations are indicative of interpersonal flexibility, which in turn may be associated with interpersonal and/or psychological benefits. In fact, interpersonal flexibility (the propensity to manage interpersonal events contingently and flexibly) has been associated with perceived social support (Liu & Xia, 2018). Interpersonal flexibility has also been linked to self-esteem (Miller, Davis, & Hayes, 1993). In a study of interpersonal behaviors, Leary (1957) found that rigid and inflexible behaviors were linked to degree of psychopathology. In other words, because individuals naturally operate within various contexts, rigid interactional problems can disable individuals from demonstrating the appropriate behavior according to the given situation. In any case, the finding of hypothesized effects for person-centered but not mean levels of affiliation suggests that chronically viewing oneself as affiliative following stressful interactions was not

important for understanding distress; it also underscores the need to distinguish between- and within-person variability when studying psychological phenomena.

I expected to find that dominance would also be a protective factor given links suggesting advantages of dominance and disadvantages of submissiveness. For instance, dominance is to closely correlated to social skills displays (Burgoon & Dunbar, 2000) and submissive IP behaviors have been linked to internalizing problems in adolescents (Powers, Battle, Dorta, & Welsh, 2010), brooding, and future psychopathology, such as depression (Pearson, Watkins, & Mullan, 2010). The current study found that person centered dominance or those idiosyncratic fluctuations in dominance, were not significantly linked to distress in daily life; however higher mean dominance was significantly associated with higher distress in daily life, contrary to the hypothesized relationship. Leary (1957) hypothesized two motives that govern interpersonal behavior (minimization of anxiety and maximization of self-esteem) and that these threats tend to evoke the individual's dominant interpersonal style. Therefore, it may be that daily fluctuations of dominance are indicative of a recent threat of anxiety or self-esteem. It may be that viewing oneself as dominant *following social stressors or conflicts* represents a different phenomenon than viewing oneself as dominant *in general.* Namely, much of the literature on psychological benefits of dominance and status has emphasized cross-sectional designs, traits, or general effects; in contrast, feeling chronically dominant after stressful social interactions may reflect having perceived events as threats and competitive contests which one could "win" or "lose," such that evening "winning" may come with the cost of higher distress.

Predicting cortisol levels. Various studies have found hypercortisolism in individuals with GAD (Cinque, et al., 2017; Dickerson & Kemeny, 2004; Vreeburg et al., 2010; Mantella et al., 2008), providing a context in which to make sense of the present findings that GAD

symptoms positively predicted cortisol symptoms. Interestingly, other studies have found the opposite – hypocortisolism. A previous evaluation of chronic stress on hair cortisol secretion (Steudte et al., 2011) found low cortisol in a GAD group relative to healthy controls. Differences in findings may be due to methodological differences. Steudte and colleagues (2011) had a smaller sample size (N=15) and had a clinical sample using diagnostic categories. In the present study, no evidence emerged of curvilinear effects, suggesting that at least in the case of dimensional GAD symptoms in a subclinical, broad sample (and a notably larger sample than the Steudte et al. study), a linear positive relationship of GAD symptoms to hair cortisol was most likely.

The current study found that viewing oneself as affiliative in stressful interactions did not directly predict cortisol levels, contrary to hypotheses. However, viewing oneself as dominant in stressful interactions positively predicted cortisol levels. It was expected that dominance would be a protective factor against this biomarker of stress as it can facilitate an individual's ability to move up a social hierarchical and obtain necessary resources. One possibility is that because the IP measure assessed the participants' perception of their dominance in response to a specific social interaction, it may be that the item was measuring a reactive dominance, or a dominance that was evoked due to, as Leary (1957) suggested, a threat of anxiety or a threat to self-esteem. This would fit with some past studies finding positive links of dominance phenomena to cortisol (Guedes et al., 2017; Gunnar, 1994; Jimenez et al., 2017) and of competition to cortisol (Arruda et al., 2017; Lautenbach et al., 2015). Moreover, it is intriguing that higher chronic dominance predicted *both* higher distress and higher cortisol, painting a relatively consistent picture. It is possible that individuals may be affiliative in response to a threat to the self as well (e.g., acting

excessively warm to avoid a conflict), but it appears that chronic feelings of dominance evoked in response to social threats was most consistently linked to high hair cortisol in this study.

The interaction of interpersonal styles and emotional distress. I conceptualized dominant and affiliative interpersonal styles as strengths that buffer against downstream emotional distress. The present study sought to evaluate whether interpersonal styles interact with GAD symptoms in predicting emotional distress and found that only person-centered affiliation interacted significantly with GAD in predicting distress. Nonsignificant interaction effects of GAD symptoms were found for mean IP affiliation and dominance, as well as person centered dominance. It appears that only person-centered affiliation played a protective role buffering against emotional distress in individuals high in GAD symptoms. Therefore, for individuals with greater GAD symptoms, those who leave a social interaction feeling that they were affiliative may experience less psychological distress. This is consistent with previous literature indicating the benefits of social support in individuals with GAD symptoms (Sangalang & Gee, 2012). This may be particularly beneficial for those with GAD symptoms considering that those with worry, subthreshold GAD, and GAD often report lower levels of social support (Kertz & Woodruff-Borden, 2011). The lack of an interaction of mean affiliation with GAD symptoms suggests that chronic warm styles in or following social conflicts were neither protective nor likely to amplify risks of distress for individuals with higher GAD symptoms, contrary to expectations and past studies finding unique links of worry to affiliative tendencies (e.g., Erickson et al., 2016).

The interaction of interpersonal styles and cortisol levels. Previously discussed research regarding dominance and cortisol is mixed. For example, research on social behavior in non-human primates and humans has indicated that dominance has most often been associated

with lower levels of cortisol compared to subordinates (Golub, Sassenrath, & Goo, 1979; Shively, Laber-Laird, & Anton, 1997). In specific time-limited stressors, such as competitive sports (Arruda et al., 2017; Lautenbach et al., 2015), results indicated increased cortisol levels. This conflicts with other lines of research that has found higher cortisol in dominant male cynomolgus macaques (Jimenez et al., 2017), as well as in individuals in positions of higher social rank, such as executives (Guedes et al., 2017). In the present study, marginally significant findings were obtained for the interaction of IP dominance on the relationship between GAD symptoms and cortisol, with chronic dominance following stressors to amplify the positive effect of GAD symptoms.

However, a fuller picture requires attending to the marginally significant three-way interaction of GAD symptoms with chronic dominance and affiliation. Namely, individuals who had high GAD symptoms and were chronically dominant and cold (unaffiliative) were most prone to high levels of hair cortisol. This contrasts with previous findings of low cortisol output or reactivity in individuals prone to hostility (Hackett et al., 2015), aggression (Sariñana-González et al., 2015), and psychopathic traits (Johnson et al., 2015), for instance. However, again, the dynamic and prospective nature of the present study, and focus upon stressor contexts, provides methodological contrast to previous cross-sectional studies. It appears that individuals who chronically worried and came away from stressors feeling “on top” but “distant” (i.e., competitive and unsupported) were most likely to produce hair samples implying chronic biological stress. However, future studies must replicate this finding, given the marginally significant nature of the effect and unexpected direction. Nonetheless, the finding highlights the need to examine how dominance and affiliation dimensions may sometimes exert combined effects beyond single dimensions alone.

Clinical Applications

Previous studies have conceptualized interpersonal types in individuals with GAD with some indications of interpersonal specificity (Erickson et al., 2016; Gomez Penedo et al., 2017) and other indications of pathoplasticity (Przeworski et al., 2011; Salzer et al., 2011). The current study does not support the idea that there is one interpersonal type that most exacerbates GAD symptoms, however for individuals with GAD symptoms, it may be beneficial to practice skills that promote affiliative behaviors (e.g., seeking and giving social support) in coping with particularly salient stressors as a means to reduce psychological stress. Such interventions may include seeking social support, empathy, appropriate conflict resolution and practicing interpersonal reparations (e.g., apologies).

Biological stress as measured by hair cortisol levels yielded results that may be clinically beneficial. The use of hair samples to assess cortisol levels is a novel and relatively non-invasive method to evaluating cortisol levels. The current study demonstrates that interpersonal perceptions following an interpersonal conflict are interacting with GAD symptoms. It may be important to address interpersonal behaviors to reduce physiological distress and its correlations to physical and mental health concerns. If high levels of cortisol (reflecting accumulations of the hormone over months) is a reliable biomarker of stress, we can infer that the "body is telling us" that chronic worry and striving for dominance may not be adaptive in the long run. Many clients find biological examples and metaphors useful for legitimating their struggles or destigmatizing them, and so the present study add to the literature linking mind and body in terms of stress responding.

Limitations and Directions for Future Research

The present study included several limitations related to data collection, measurement, and statistical analysis which require that the findings be interpreted with caution. Future research which seeks to address these methodological limitations would be beneficial. First, the data for the present study was collected from undergraduate students at a private Protestant school in a large city, requiring replication. Second, there were substantially more female subjects than male ones, and no students reported other gender identities. This fact limits generalizability to a more diverse population. Although the present study had somewhat of a racially diverse sample, future studies should examine whether these phenomena have similar effects in non-white samples. Dominance, affiliation, and interpersonal stressors may 'look' different in the context of different racial groups or in the context of protests, advocacy or asserting resistance. Third, the variables were measured over a relatively small time period, five weeks. Although the cortisol samples speak to roughly two months of cortisol secretion, the results of this study cannot be extended to long-term effects of interpersonal behaviors. Moreover, the results cannot extend to participants who were excluded due to short hair or being unwilling to provide a sample.

A limitation related to the measurement of study variables was the small number of items to measure interpersonal behaviors. As previously described, the measure of interpersonal problems included two dimensional ratings to assess how an individual perceived themselves interpersonally (one for each domain – dominant and affiliative). Future studies may yield more reliability and compelling results by using more items that map onto interpersonal styles. It may also be advantageous for future studies to include both self and informant report as research has found discrepancies between these two methodologies and that GAD symptoms predict over-endorsing IP affiliation (Shin & Newman, 2019).

The results of the current study highlight important areas for future research. Understanding how individuals with GAD symptoms behave interpersonally can shed light on appropriate treatments to address those aspects of interpersonal styles that contribute to psychological distress. For instance, research has found that motivational interviewing (MI) in addition to cognitive behavioral therapy (CBT) can lead to long-term interpersonal changes (Muir, Constantino, Coyne, Westra & Antony, 2019). An extension of this research found that for individuals with more problematic *low* agency (i.e., dominance), CBT vs. MI-CBT facilitated greater friendly submissiveness, which in turn, was associated with increased worry. Conversely, for individuals with more problematic *high* agency, CBT's facilitation of greater friendly submissiveness reduced worry (Gómez Penedo et al., 2019). Treatment matching based on GAD subgroups of interpersonal characteristics may be promising.

Future research may also evaluate different 'types' of dominance. For instance, assessing blends of dominance as conceptualized on the IPC (e.g., cold-dominance vs. affiliative dominance), or dominance in stable vs. unstable hierarchies and differences in social organizations (e.g., opportunities to move up or down in rank), may reveal important differences. Closer evaluation of various forms of dominance may better clarify under what conditions dominance is buffering vs. amplifying stress.

Conclusion

The primary goal of this study was to examine the associations among generalized anxiety symptoms, interpersonal behaviors, psychological distress and cortisol. Even when controlling for interpersonal dimensions, GAD was a powerful predictor of stress in daily life, and also predicted a relatively novel biomarker of stress. Overall, the study contributes to the interpersonal literature, specifically in individuals with GAD symptoms suggesting further

warrant for investigating IPC dimensions and their interactions with GAD. The current study also underscores the need to study interpersonal phenomena using both person-centered methods and mean/chronic methods, because the two sources of variability did not behave identically. In other words, the study identifies two sources of variability that should be considered within the interpersonal and GAD literature. Further research is warranted to understand the daily experiences and biological processes of individuals at risk for chronic and uncontrollable worry, with the ultimate goal of reducing their suffering.

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