Intolerance of Uncertainty:

A Vulnerability Factor for Internalizing Psychopathologies

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THESIS

Submitted as partial fulfillment of the requirements
for the degree of Master of Arts in Psychology
in the Graduate College of the
University of Illinois at Chicago, 2018

Chicago, Illinois

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LIST OF ABBREVIATIONS

AUD	Alcohol Use Disorder
DASS	Depression, Anxiety, and Stress Scale
DSM-5	Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition
GAD	Generalized Anxiety Disorder
HLMs	Hierarchical Linear Models
ICC	Intraclass Correlation Coefficient
IU	Intolerance of Uncertainty
IUS-12	The Intolerance of Uncertainty Scale, Short Form
N/NA	Neuroticism or Negative Affectivity
OCD	Obsessive-Compulsive Disorder
PD	Panic Disorder
PID-5	Personality Inventory for Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition
PTSD	Posttraumatic stress disorder
RDoC	Research Domain Criteria
SAD	Social Anxiety Disorder
SCID	The Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders
SUD	Substance Use Disorder

SUMMARY

Intolerance of uncertainty (IU) is a putative vulnerability factor for internalizing psychopathologies. However, it is unclear if IU and its subscales (prospective and inhibitory IU) meet criteria for a vulnerability factor. It is also unknown if IU is a vulnerability factor specifically for internalizing (vs. externalizing) psychopathology and whether it is a vulnerability factor independent of neuroticism/negative affectivity (N/NA). Four hundred and ninety-four adult sibling pairs (n = 233) with a wide range of psychopathologies, as well as healthy controls, completed a diagnostic interview and self-report measures of IU and N/NA. Results indicated that, independent of N/NA, IU is not a vulnerability factor for all internalizing psychopathologies, but may be a vulnerability factor for Major Depression, Post-traumatic Stress Disorder, Social Anxiety Disorder, and was trending for Generalized Anxiety Disorder. Furthermore, IU may not specific to internalizing psychopathologies as findings indicated that it may be a vulnerability factor for substance use disorders. In terms of subscales, Inhibitory IU, and not total or prospective IU, exhibited the strongest evidence for being a vulnerability factor.

1. INTRODUCTION

Psychopathologies involving anxiety and depression (i.e. internalizing psychopathologies; Kendler et al. 2003; Krueger et al. 1998; Vollebergh et al. 2001) are serious, prevalent, and costly public health burdens. Internalizing psychopathologies are among the top 10 leading disabilities in the United States and carry an economic burden of hundreds of billions of dollars (Baxter, Vos, Scott, Ferrari, & Whiteford, 2014; Greenberg, Fournier, Sisitsky, Pike, & Kessler, 2015). Identifying vulnerability markers for internalizing psychopathologies could reduce this burden by providing novel clinical targets for early identification and preventative treatments. Intolerance of uncertainty (IU), a personality trait, may be an important vulnerability marker for internalizing psychopathology.

Intolerance of Uncertainty is the tendency to respond to uncertainty with negative cognitive, emotional, and behavioral reactions (Carleton, Norton, & Asmundson, 2007; Freeston, Rheaume, Letarte, Dugas, & Ladouceur, 1994). Early research on IU focused on its proposed specific and integral role in the maintenance of generalized anxiety disorder (GAD; Dugas, Gagnon, Ladouceur, & Freeston, 1998). However, more recent evidence that has shown an association between obsessive-compulsive disorder and heightened levels of IU (Holaway, Heimberg, & Coles, 2006; Tolin, Abramowitz, Brigidi, & Foa, 2003) raised the possibility that IU is not specific to GAD and may relate to anxiety disorders more broadly. In fact, IU has been found to be positively associated with symptoms of panic disorder (Carleton et al., 2014; Carleton, Fetzner, Hackl, & McEvoy, 2013), social anxiety disorder (SAD; Boelen & Reijntjes, 2009; Carleton, Collimore, & Asmundson, 2010; Whiting et al., 2014), and posttraumatic stress disorder (Bardeen, Fergus, & Wu, 2013; Fetzner, Horswill, Boelen, & Carleton, 2013). Intolerance of Uncertainty is also positively correlated with depression symptoms (Yook, Kim,

Suh, & Lee, 2010), even when controlling for neuroticism or negative affectivity (N/NA; McEvoy & Mahoney, 2011, 2012). This association between depression and increased levels of IU is comparable to the associations observed in obsessive-compulsive disorder (OCD), GAD, panic disorder (PD), and SAD (Carleton et al., 2012; Gentes & Ruscio, 2011). Therefore, IU not only relates to anxiety disorders, but is a transdiagnostic trait within internalizing psychopathologies in general.

While internalizing psychopathologies share several other vulnerability factors in addition to IU, IU appears to be particularly important. A recent meta-analysis of cognitive vulnerabilities related to anxiety or depression showed that IU accounted for the most variance among a wide range of cognitive vulnerabilities (Hong & Cheung, 2015). However, while IU is generally considered to be a vulnerability factor (rather than simply a characteristic of internalizing psychopathology), it is unclear whether IU meets all of the necessary criteria of a vulnerability factor.

In their classic study, Zubin & Spring (1977) proposed that a vulnerability marker should exist (a) before, (b) during, and (c) after an episode and (d) that the vulnerability marker must also be familial (i.e., correlated within families). The presence of the trait before the onset of psychopathology ensures that this trait is not merely a "scar" or byproduct of psychopathology. The vulnerability marker must also be present during an episode of psychopathology to ensure that it is, in fact, related to the disorder or disorders of interest. The marker's presence after an episode of psychopathology further demonstrates that it is an enduring and stable trait and not just a symptom of the disorder. Lastly, establishing that a vulnerability marker is familial suggests that this trait is endogenous and heritable.

Aside from the numerous studies that have reported elevated IU in those with current internalizing psychopathology (Carleton et al., 2012, 2010; Holaway et al., 2006; Nelson, Liu, Sarapas, & Shankman, 2016; Nelson, Shankman, & Proudfit, 2014; Tolin et al., 2003; Whiting et al., 2014), few studies have tested whether IU meets the other criteria for a vulnerability factor. A small handful of studies have investigated whether individuals who later develop an internalizing psychopathology show elevated levels of IU before the onset of the disorder. Oglesby, Boffa, Short, Raines, and Schmidt (2016) assessed undergraduates levels of IU before and after exposure to a university campus shooting. Results indicated that, even when controlling for pre-trauma anxiety sensitivity (a construct associated with both anxiety and IU; Hong & Cheung, 2015), pre-trauma IU predicted posttraumatic stress symptoms following the shooting. Boelen, Reijntjes & Smid (2016) also found baseline IU to be predictive of internalizing psychopathology symptom severity following the loss of a loved one. These results suggest that high IU is present before the onset of an internalizing psychopathology. However, it is unknown whether high IU is predictive of other internalizing psychopathologies or whether this relationship is specific to traumatic or other stressful life events. Even fewer studies have assessed IU when participants are in remission. Treatment studies have found that psychotherapy can reduce the severity of IU in those with GAD, OCD, SAD, and PD (Boswell, Thompson-Hollands, Farchione, & Barlow, 2013; Dugas & Ladouceur, 2000; Ladouceur et al., 2000), but have not evaluated whether those in remission still report elevated levels of IU in comparison to those without a history of an internalizing psychopathology. To our knowledge, no prior studies have investigated whether IU runs in families.

1.1. Aims

The aims of the current study are therefore to assess whether IU is (1) elevated in individuals currently experiencing an episode of an internalizing psychopathology (2) elevated in individuals who are in remission from an internalizing psychopathology and (3) familial. It is important to assess these criteria before evaluating whether IU is elevated before the development of an internalizing psychopathology because the latter necessitates the use of a longitudinal high-risk design and significantly more resources. The results of the current study can therefore provide preliminary data to address the question of whether the employment of a longitudinal high-risk design is warranted.

1.1.1. Neuroticism and Negative Affectivity

It is also important to assess whether IU meets the previously stated criteria for vulnerability factors while controlling for trait N/NA. The strength of the association as well as the conceptual overlap between IU and N/NA bring into question the independence of these possible vulnerability factors (McEvoy & Mahoney, 2012; Sexton, Norton, Walker, & Norton, 2003). Indeed, numerous studies have shown that N/NA is a vulnerability factor for depression. Neuroticism or negative affectivity is familial (Farmer et al., 2002) and is elevated before (Kendler, Neale, Kessler, Heath, & Eaves, 1993; Ormel, Oldehinkel, & Vollebergh, 2004), during (Kendler et al., 1993; Ormel et al., 2004), and after (Kendler et al., 1993; Ormel et al., 2004) a major depressive episode. Neuroticism or negative affectivity is also highly correlated with internalizing psychopathology (Griffith et al., 2010). It has also been shown N/NA is not specific to depression, but is a vulnerability factor for all internalizing psychopathologies (Clark, Watson, & Mineka, 1994) and numerous other conditions as well (Lahey, 2009). In order to

examine whether IU is a separate vulnerability factor from N/NA, an additional set of analyses for the first and second aims of the study will control for individuals' levels of N/NA.

1.1.2. Subfactors of Intolerance of Uncertainty

While it is hypothesized that IU will be elevated during and after episodes of internalizing psychopathology as well as be familial, this might not be true for all aspects of IU. Factor analytic studies have demonstrated that IU is made up of two separate, but related factors - prospective IU and inhibitory IU (Carleton et al., 2007; McEvoy & Mahoney, 2011). Prospective IU is characterized by future-oriented cognitive and emotional distress to uncertainty whereas inhibitory IU is described as a behavioral inhibition in response to uncertainty. While these factors are correlated with each other, they have been shown to have differential predictive validity such that prospective IU is associated with heightened startle to unpredictable threat whereas inhibitory IU is related to attenuated startle to unpredictable threat (Nelson et al., 2016). It is possible that internalizing psychopathologies may be elevated or familial for one of these factors and not the other. Furthermore, the relationships with prospective and inhibitory IU may differ by the type of internalizing psychopathology. Therefore, the current study will also explore whether prospective and inhibitory IU are familial as well as elevated across current and remitted GAD, OCD, PD, SAD, MDD, PTSD (Posttraumatic Stress Disorder), and specific phobia or are specific to certain internalizing diagnoses.

1.1.3. Specificity to Internalizing Psychopathologies

It is also possible that IU may not be specific to internalizing psychopathologies and could be a transdiagnostic trait for a much wider range of psychopathologies than previously thought. Alcohol dependence (which is typically characterized as an externalizing, not internalizing, disorder; (Kendler et al., 2003; Krueger et al., 1998; Vollebergh et al., 2001) has

been shown to be related to increased startle to unpredictable threat (Gorka & Shankman, 2017; Gorka, Lieberman, Phan, & Shankman, 2016; Gorka, Nelson, & Shankman, 2013), a key psychophysiological correlate of IU (Nelson et al., 2016). Moreover, startle potentiation to unpredictable, but not predictable, threat is positively correlated with a family history of alcohol use disorder (AUD; Gorka et al., 2016). The increased reactivity to unpredictable threat evidenced in individuals with AUD may be a behavioral correlate of IU. If so, IU may also be a vulnerability factor for AUD – and perhaps other externalizing psychopathologies as well. Consequently, an additional exploratory aim will be to assess whether IU is specific to internalizing psychopathologies or if it is also a vulnerability factor for externalizing psychopathologies such as AUD.

2. METHODS

2.1. Participants

A total of 494 participants were drawn from an ongoing NIMH-funded family study (see Gorka et al., 2016 for additional details). Participants were nested within 261 families and included 233 sibling pairs. Advertisements (fliers, internet postings, etc.) were used to recruit participants from the community and from mental health clinics. Participants were 18 to 30 years old (M = 22.387, SD = 3.158) with a wide range of psychopathologies, as well as healthy controls. A RDoC (Research Domain Criteria) approach was taken to participant recruitment such that recruitment screening was agnostic to Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-5) diagnostic categories (beyond the exclusion criteria listed below). However, participants with severe internalizing psychopathology were oversampled to ensure that the sample was clinically relevant. Specifically, the Depression, Anxiety, and Stress Scale (DASS; Lovibond & Lovibond, 1995) was administered during the initial phone screen to ensure that the severity of internalizing symptomology within the sample was normally distributed, but also had a higher average general psychological distress score (M = 10.35, SD = 10.07) than the general population (M =8.3 , SD = 9.83; Crawford, Cayley, Lovibond, Wilson, & Hartley, 2011).

Inclusion criteria specified that participants had at least one full biological sibling that was also willing to participate in the study. Both siblings participated in the study. Other first-degree relatives of the siblings (e.g., parents) were contacted and asked if they too were willing to participate in an interview either in person or over the phone. Exclusion criteria included personal or family history of psychosis or mania, inability to read or write in English, history of serious head trauma, and left-handedness. Exclusion criteria were chosen to ensure that

participants could provide consent and to protect against confounds with the physiological data collected for the main aims of the larger study.

2.2. Measures

2.2.1. The Intolerance of Uncertainty Scale, short form

The Intolerance of Uncertainty Scale, short form (Carleton et al., 2007) is a 12-item self-report scale that assesses reactions to uncertainty or ambiguity. It has demonstrated better psychometric properties than the original 27-item Intolerance of Uncertainty Scale (Carleton et al., 2007; Freeston et al., 1994). Factor analysis has shown that the IUS-12 consists of two subscales: prospective and inhibitory IU. The prospective IU subscale consists of 7 items (e.g., "One should always look ahead so as to avoid surprises") and the inhibitory IU subscale consists of 5 items (e.g., "The smallest doubt can stop me from acting"). Scores are measured by a 5-point Likert scale ranging from 1 (not at all characteristic of me) to 5 (entirely characteristic of me).

2.2.2. Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders 5

The Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders 5 (First, Williams, Karg, & Spitzer, 2015) is a semi-structured clinical interview used to assess whether an individual meets criteria for any diagnoses as defined by the DSM-5. The following modules were administered in the current study: MDD, AUD, Substance Use Disorder (SUD), Post-Traumatic Stress Disorder (PTSD), PD, Agoraphobia, SAD, Specific Phobia, OCD, GAD, Anorexia, Bulimia, Binge Eating Disorder, and the bipolar and psychotic screening modules. Doctoral students and bachelor's level research assistants were trained to criterion on the SCID and were supervised by a licensed clinical psychologist. Interrater agreement was in

the fair to substantial ranges for lifetime diagnoses (k's = .46 - .87) and in the fair to moderate ranges for current diagnoses (k's = .54 - .74) with the exception of lifetime (k = .18) and current (k = .29) social anxiety disorder diagnoses with interrater agreement in the slight range (Shankman et al., n.d.; Shrout, 1998).

2.2.3. **The Personality Inventory for Diagnostic and Statistical Manual of Mental Disorders 5**

The Negative Affect domain of the The Personality Inventory for DSM-5 (Krueger, Derringer, Markon, Watson, & Skodol, 2012) was used to assess N/NA. The PID-5 was designed to assess the personality traits of the alternative model of personality disorders in DSM-5 and is a 220-item self-report scale that measures five broad pathological personality domains (Negative Affect, Detachment, Antagonism, Disinhibition, and Psychoticism) and 25 underlying facets of these domains. Each item on the PID-5 is rated on a 4-point Likert scale ranging from 0 (very false or often false) to 3 (very true or often true). The Negative Affect domain has been shown to be strongly correlated with Neuroticism (Watson, Stasik, Ro, & Clark, 2013). Three facets or subscales comprise the Negative Affect domain: Emotional Lability (e.g. "My emotions are unpredictable"), Anxiousness (e.g. "I worry about almost everything"), and Separation Insecurity (e.g. "I'd rather be in a bad relationship than be alone").

2.3. **Statistical Analyses**

As current psychiatric medication status (yes vs. no), N/NA, gender, and age have all been shown to be related to internalizing psychopathology status or severity (Clark et al., 1994; Fournier et al., 2010; Griffith et al., 2010; Kessler et al., 2005, 2007), the associations between these variables and the dependent variables (total, prospective, and inhibitory IU scores) were examined. Variables found to be associated with dependent measures were included as

covariates in analyses where appropriate. Hierarchical linear models (HLMs) were conducted to assess study aims 1 and 2 since the sample consists of individuals that are nested within families. For aim 1, a HLM was conducted to assess whether IU is elevated in those currently experiencing an internalizing psychopathology. Current internalizing psychopathology status (any diagnosis of current GAD, OCD, PD, SAD, MDD, PTSD, or specific phobia vs. no current GAD, OCD, PD, SAD, MDD, PTSD, and specific phobia) served as the independent variable and total IU score as the dependent variable. Given the heterogeneity of internalizing psychopathologies, additional analyses were conducted for each of the seven internalizing psychopathologies (GAD, OCD, PD, SAD, MDD, PTSD, and specific phobia) examined in the current study. The status of each current internalizing psychopathology (e.g. current GAD vs. no lifetime history of psychopathology) served as the independent variables and total IU scores served as the dependent variable. These analyses enabled an exploration of whether IU may be related to certain internalizing psychopathologies and not others.

Aim 2 was analyzed in a similar manner, but with remission of internalizing psychopathology status (any diagnosis of remitted GAD, OCD, PD, SAD, MDD, PTSD, or specific phobia vs. no lifetime history of GAD, OCD, PD, SAD, MDD, PTSD, and specific phobia) as the independent variable. This examined whether those who have had an episode of internalizing psychopathology in the past, but are not currently in episode, have elevated levels of IU in comparison to those who have never had an episode of internalizing psychopathology. Similar to the analyses for aim 1, additional analyses were conducted for each of the seven internalizing psychopathologies (GAD, OCD, PD, SAD, MDD, PTSD, and specific phobia) examined in the current study. The status of each remitted internalizing psychopathology (e.g.

remitted GAD vs. no lifetime history of psychopathology) served as the independent variables and total IU scores served as the dependent variable.

To investigate aim 3, a one-way random effects intraclass correlation coefficient (ICC) model was conducted on total IU scores. These analyses tested whether the trait runs in families by assessing the within sibling pair correlations between total IU scores.

For the exploratory aims related to the IU subscales, both HLMs and ICCs were utilized to examine whether prospective and inhibitory IU are elevated across internalizing psychopathologies and familial. These analyses were similar in structure to the analyses outlined above, only with prospective IU and inhibitory IU scores as the dependent variables.

To explore whether IU is specific to internalizing psychopathologies or if it is also a vulnerability factor for externalizing psychopathologies, HLMs were conducted with current and remitted externalizing psychopathology as independent variables and total, prospective, and inhibitory IU scores as dependent variables. The first set of these analyses examined whether IU is elevated in those currently experiencing an externalizing psychopathology (any diagnosis of current AUD or SUD vs. no current AUD and SUD). Like the analyses for aim 1, additional analyses were conducted to explore whether IU may be differentially related to AUD and SUD. The status of current AUD and SUD (e.g. current AUD vs. no lifetime history of psychopathology and current SUD vs. no lifetime history of psychopathology) served as the independent variables. The second set of analyses explored whether IU is elevated in those in remission from an externalizing psychopathology (any diagnosis of remitted AUD or SUD, vs. no lifetime history of AUD and SUD). Levels of IU in remitted AUD and SUD were also explored separately. The status of remitted AUD and SUD (e.g. remitted AUD vs. no lifetime

history of psychopathology and remitted SUD vs. no lifetime history of psychopathology) served as the independent variables.

Lastly, analyses were conducted to compare levels of IU between internalizing and externalizing psychopathologies. The first set of these analyses tested the interaction between current internalizing (any diagnosis of current GAD, OCD, PD, SAD, MDD, PTSD, or specific phobia vs. no lifetime history of psychopathology) and externalizing diagnosis (any diagnosis of current AUD or SUD vs. no lifetime psychopathology). The final set of analyses examined the interaction between remitted internalizing (any diagnosis of remitted GAD, OCD, PD, SAD, MDD, PTSD, or specific phobia vs. no lifetime history of psychopathology) and externalizing psychopathology (any diagnosis of remitted AUD or SUD vs. no lifetime psychopathology).

3. RESULTS

3.1. Covariates

Individuals that were currently taking psychiatric medications (β = 5.220, p = .0002), were high in N/NA (β = 5.838, p < .001), and older in age (β = .881, p = .039) displayed elevated total IU scores, while gender (β = -.854, p = .330) had no effect on levels of total IU. Similarly, individuals that were currently taking psychiatric medications (β = 2.814, p = .001), were high in N/NA (β = 3.173, p < .001), and older in age (β = .536, p = .043) showed higher levels of prospective IU, but gender (β = -.498, p = .360) did not have an effect on levels of prospective IU. For inhibitory IU, individuals that were currently taking psychiatric medications (β = 2.414, p = .0001) and were high in N/NA (β = 2.664, p < .0001) demonstrated elevated levels of inhibitory IU, but age (β = .351, p = .069) and gender (β = -.350, p = .380) had no effect on levels of inhibitory IU. Consequently, medication status, N/NA, and age were entered as covariates for models with total or prospective IU scores as the dependent variables. For models with inhibitory IU as the dependent variable, only medication status and N/NA were entered in as covariates. Furthermore, given the strength of the relationship between IU and N/NA, models both with and without N/NA are reported below.

3.2. Current Psychopathology

3.2.1. Internalizing Psychopathology

Individuals with a current internalizing psychopathology exhibited significantly higher levels of total (β = 2.530, p = .001), prospective (β = 1.442, p = .005), and inhibitory (β = 1.142, p = .001) IU in comparison to those with no lifetime history of internalizing psychopathology. Similar results were found when N/NA was removed from the models. When levels of IU were examined for each diagnosis individually, those with current GAD, MDD, SAD, specific phobia,

and PTSD displayed elevated levels of total IU. For prospective IU, only individuals with SAD and specific phobia displayed significantly higher levels than those with no lifetime history of psychopathology (scores for those with GAD were approaching a significant difference from controls). Inhibitory IU was significantly higher in all internalizing psychopathologies, except for PD. When N/NA scores were removed from the individual diagnosis models, total, prospective, and inhibitory IU scores were all significantly elevated in all current internalizing psychopathologies. The subscale results suggest that significant differences in levels of total IU scores were largely driven by elevated levels of inhibitory, but not prospective, IU. See Table 1 for results on all models regarding main effects of current internalizing psychopathology.

3.2.2. **Externalizing Psychopathology**

Individuals currently experiencing an externalizing psychopathology did not show significantly elevated levels of total ($\beta = -1.330$, $p = .220$) or inhibitory ($\beta = .203$, $p = .682$) IU compared to those with no lifetime history of externalizing psychopathology. In fact, the significant difference between groups on prospective IU ($\beta = -1.531$, $p = .033$) indicated that individuals with current externalizing psychopathology displayed *lower* levels of prospective IU than those with no lifetime history of externalizing psychopathology. When N/NA was removed from the models, there was no longer a significant difference between groups on prospective IU, but those with current externalizing psychopathology showed higher levels of inhibitory IU than those with no lifetime history of externalizing psychopathology.

Current AUD and SUD were also examined separately. Only levels of inhibitory IU in individuals with current SUD significantly differed and were higher than in individuals with no lifetime history of any psychopathology. With N/NA removed from the models, individuals with current SUD displayed significantly higher total, prospective, and inhibitory IU scores than those

with no lifetime history of psychopathology. Individuals with current AUD also showed significantly higher levels of total and inhibitory, but not prospective, IU. See Table 1 for results on all models regarding current externalizing psychopathology.

3.3. **Remitted Psychopathology**

3.3.1. **Internalizing Psychopathology**

Individuals in remission from an internalizing psychopathology did not significantly differ from those with no lifetime history of internalizing psychopathology on levels of total (β = .767, p = .354) or prospective (β = .293, p = .608) IU. However, elevated inhibitory IU scores in those in remission from an internalizing psychopathology, as compared to those with no lifetime history of internalizing psychopathology, were approaching significance, β = .595, p = .089. When N/NA was removed from the models, levels of total (β = 3.140, p = .001), prospective (β = 1.652, p = .007), and inhibitory (β = 1.584, p < .001) IU were significantly higher in individuals in remission from an internalizing psychopathology than those with no lifetime history of internalizing psychopathology.

For individual internalizing diagnoses, only individuals with remitted SAD were found to have significantly higher total IU scores than individuals with no lifetime history of psychopathology. For the subscales, no significant differences were found between groups on prospective IU scores. Although, individuals with remitted MDD, SAD, PD, and PTSD all displayed significantly elevated levels of inhibitory IU (the differences between groups for GAD was approaching significance) in comparison to those with no lifetime history of internalizing psychopathology. When N/NA was removed from the models, total IU scores were significantly higher in individuals with all internalizing psychopathologies except for remitted OCD, which was only approaching significance. Prospective IU scores for all remitted internalizing

psychopathologies, except for OCD, and inhibitory IU scores for all remitted internalizing psychopathologies were significantly elevated in comparison to those with no lifetime history of psychopathology. See Table 2 for results on all models regarding remitted internalizing psychopathology.

3.3.2. **Externalizing Psychopathology**

Individuals in remission from an externalizing psychopathology did not significantly differ on levels of total (β = .164, p = .834), prospective (β = -.008, p = .987), and inhibitory (β = .320, p = .349) IU from those with no lifetime history of externalizing psychopathology. However, when N/NA was removed from the models, those with remitted externalizing psychopathologies showed significantly higher inhibitory IU scores (total IU scores approached significance) than those with no lifetime history of externalizing psychopathology. When AUD and SUD were inspected separately, only levels of inhibitory IU in individuals in remission from SUD were significantly higher than in those with no lifetime history of psychopathology. Levels of inhibitory IU in individuals in remission from AUD were elevated as well, however, this only approached, but did not reach, significance. When N/NA was removed from these models, individuals in remission from AUD displayed significantly higher levels of total, prospective, and inhibitory IU as compared to those with no lifetime history of psychopathology. A similar pattern of results was shown for those with remitted SUD, however, differences in prospective IU only approached significance. See Table 2 for results on all models regarding remitted externalizing psychopathology.

3.4. **Specificity of Intolerance of Uncertainty**

We further explored the specificity of IU to internalizing psychopathology by examining the effect of the interaction of internalizing and externalizing psychopathology on levels of IU.

No significant interaction was found between current internalizing and externalizing psychopathologies for total IU scores (β = 3.251, *p* = .135) or prospective IU scores, β = 1.265, *p* = .381. For inhibitory IU scores, the interaction between current internalizing and externalizing psychopathologies was approaching significance, β = 1.827, *p* = .065. Follow-up models indicated that individuals with current comorbid internalizing and externalizing psychopathology had significantly elevated levels of inhibitory IU in comparison to those with no lifetime history of psychopathology (β = 3.104, *p* = .005). Those with only current internalizing (without current externalizing) and only current externalizing (without current internalizing) psychopathology displayed increased (β = .679, *p* = .057) and decreased (β = -1.243, *p* = .075) inhibitory IU, respectively, that was approaching a significant difference from individuals with no lifetime history of psychopathology. Individuals with only current internalizing (without current externalizing; β = 1.922, *p* = .008) and those with comorbid current internalizing and externalizing (β = 4.347, *p* = .001) both had significantly higher levels of inhibitory IU compared to individuals with only current externalizing (without current internalizing). Finally, individuals with current comorbid internalizing and externalizing psychopathology had significantly higher levels of inhibitory IU compared to those with only current internalizing (without current externalizing) psychopathology, β = 2.425, *p* = .029.

When N/NA was removed from the models, none of the interactions reached or approached significance. Furthermore, none of the interactions between remission of internalizing and externalizing psychopathologies (both with and without N/NA) reached significance. These results suggest that elevated levels of IU are specific to internalizing and not externalizing psychopathologies

3.5. **Correlation within Sibling Pairs**

Intolerance of Uncertainty was correlated within siblings. Significant correlations were found between siblings' scores on the total (ICC = .261, 95% CI .038-.432, F(223, 224) = 1.353, p = .012) and prospective (ICC = .302, 95% CI .093-.464, F(223,224) = 1.434, p = .004) IU scales. Correlations between siblings on the inhibitory IU approached significance, ICC = .189, 95% CI -.055-.377, F(223,224) = 1.233, p = .059.

4. DISCUSSION

Intolerance of uncertainty may be a key, transdiagnostic, vulnerability factor for internalizing psychopathologies. However, it is unknown whether IU meets criteria for a vulnerability factor and if it does so independently of N/NA. Furthermore, it is unclear whether IU is only a vulnerability factor for certain internalizing disorders or if it is even specific to internalizing psychopathologies (vs. externalizing psychopathologies). Results indicated that IU is not a vulnerability factor for all internalizing psychopathologies, but may be a vulnerability factor for several specific disorders. Intolerance of uncertainty was found to be familial and, even when controlling for N/NA, IU was shown to be elevated both during and after episodes of 5 out of the 9 psychopathologies examined. In terms of which specific aspects of IU were vulnerability factors, after controlling for N/NA, prospective IU was elevated in individuals for 3 of the 9 current psychopathologies but was not elevated in individuals in remission from any of the psychopathologies studied. In contrast, inhibitory IU was elevated in individuals for 7 of the 9 current psychopathologies and 7 of the 9 remitted psychopathologies, even when controlling for N/NA. Thus, the role of inhibitory IU in psychopathology was significantly stronger than that of prospective IU and is therefore more likely to be a greater vulnerability factor for psychopathology.

4.1. Neuroticism and Negative Affectivity

Notably, these findings are independent of, and cannot be explained by N/NA. While N/NA is correlated with IU (McEvoy & Mahoney, 2012; Sexton et al., 2003), it is a broader trait than IU that is quite multifaceted (Ormel, Rosmalen, & Farmer, 2004). Indeed, the measure of N/NA used in the present study (PID-5) includes three subscales or facets of N/NA - Emotional Lability, Anxiousness, and Separation Insecurity – that assess different constructs than IU does.

Moreover, measures of N/NA often contain similar items as assessments of internalizing symptoms (Ormel et al., 2013), which increases its collinearity with internalizing psychopathology. Consequently, it is a "riskier test" (Meehl, 1978) to assess whether IU meets criteria for a vulnerability factor independent of N/NA. Despite this risk and similar to previous findings (Boelen & Reijntjes, 2009; McEvoy & Mahoney, 2011, 2012; Yook et al., 2010), results showed that IU added incremental validity over and above N/NA in its associations with internalizing psychopathologies.

Intolerance of uncertainty may also provide greater specificity than N/NA in predicting certain psychopathologies. Indeed, N/NA has been shown to play a role in all internalizing psychopathologies, substance use disorders, and has even been shown to predict the onset of psychotic symptoms (Clark et al., 1994; Griffith et al., 2010; Ormel et al., 2013; Ormel, Rosmalen, et al., 2004). Ormel, Rosmalen, and Farmer (2004) argued that N/NA is a vulnerability factor for many disorders because measures of N/NA assess for fairly stable levels of distress that therefore predict future and past levels of such distress. Assessing for levels of IU may, therefore, increase our specificity in predicting outcomes.

4.2. **Specificity to Internalizing Psychopathologies**

Results suggest that IU may only be a vulnerability factor for MDD, SAD, PTSD, and SUD, but not internalizing psychopathology as a whole. Intolerance of uncertainty may also possibly be a vulnerability factor for GAD; individuals with current GAD showed significantly elevated levels of IU, and higher levels of IU during remission were trending towards significance. Even though IU was originally thought to be a key factor in the maintenance of anxiety disorders (Dugas et al., 1998; Holaway et al., 2006; Tolin et al., 2003), results indicated that, in addition to anxiety-related disorders (SAD, PTSD, and GAD), IU is likely a vulnerability

factor for depression as well. Furthermore, Hong and Cheung (2015) demonstrated that cognitive vulnerabilities to anxiety and depression are not specific to distress disorders (e.g., MDD and GAD) or fear-based disorders (e.g., SAD, OCD, PD, etc.). Instead, their findings suggest that these cognitive vulnerabilities share a common etiological factor for all internalizing psychopathologies, with IU explaining the largest variance in said factor. In fact, IU has previously been shown to be elevated during uncertain rewarding contexts (Gorka, Nelson, Phan, & Shankman, 2016; Luhmann, Ishida, & Hajcak, 2011; Nelson et al., 2014). Therefore, IU may not be specific to uncertain threatening contexts, but may be an aversion to uncertainty itself, regardless of the possible outcomes.

Although IU is likely a vulnerability factor for several internalizing psychopathologies, it may not be specific to internalizing psychopathology. Findings indicated that IU was significantly elevated in individuals with current and remitted SUD. However, these results were likely driven by high rates of comorbidity between SUD and internalizing psychopathologies. When exploring the interaction between internalizing and externalizing psychopathologies, those with current comorbid internalizing and externalizing psychopathologies displayed significantly higher levels of IU than those with no lifetime history of psychopathology. In contrast, those with only current externalizing psychopathology (no current internalizing psychopathology) showed a trend for *lower* levels of IU in comparison to individuals with no lifetime history of psychopathology. These findings should be interpreted with caution, however, as they no longer reached or approached significance when N/NA was removed from the models and were not significant for remitted psychopathologies. Furthermore, 81% of those with current or remitted SUD had a lifetime history of internalizing psychopathology. Analyses of levels of IU for individuals with externalizing psychopathology and no lifetime history of internalizing

psychopathology may consequently have been underpowered. In other words, the present study cannot elucidate how IU functions in SUD independent of the effects of internalizing psychopathologies.

Conversely, IU did not meet criteria to be a vulnerability factor for AUD. If heightened reactivity to unpredictable threat is a psychophysiological correlate of IU (McEvoy, Carleton, Correa, Shankman, & Shihata, n.d.), the present findings are inconsistent with results from prior research. Several studies have shown that individuals with AUD display a heightened reactivity to unpredictable threat (Gorka & Shankman, 2017; Gorka, Lieberman, Phan, & Shankman, 2016; Gorka, Nelson, & Shankman, 2013). Recently, Gorka & Shankman (2017) found that heightened reactivity to unpredictable threat may even be an endophenotype for AUD. This physiological reaction was, amongst other criteria for an endophenotype, found to be elevated in individuals with both current and remitted AUD. It is possible that the discrepancy between the present findings and prior research may be due to an underreporting bias of IU in those with AUD (Johnson & Fendrich, 2005; Morral, McCaffrey, & Iguchi, 2000), especially since the IUS was developed with internalizing psychopathology in mind. However, IU was elevated in SUD suggesting that individuals with SUD did not display such an underreporting bias. It is also possible that heightened reactivity to unpredictable threat may only be a psychophysiological correlate of IU during threatening contexts and not during uncertainty in general.

4.3. **Subfactors of Intolerance of Uncertainty**

In the current study, not all aspects of IU met the vulnerability criteria outlined above. Results indicated that prospective IU was not a vulnerability factor for any of the disorders studied. On the other hand, Inhibitory IU exhibited evidence of being a vulnerability factor for MDD, PTSD, SAD, SUD, and trend effects for GAD as well as for any internalizing

psychopathology. These results are consistent with findings suggesting that inhibitory, and not prospective, IU is specific to symptoms of MDD, PTSD, and SAD (Carleton et al., 2010; Mahoney & Mcevoy, 2012a, 2012b; McEvoy & Mahoney, 2011). Findings have been mixed regarding the association of prospective and inhibitory IU with GAD, PD, and OCD (Mahoney & Mcevoy, 2012a, 2012b; McEvoy & Mahoney, 2011). The lack of consistency in the associations between the IU subscales and GAD, PD, and OCD evidenced in prior studies as well as the present null findings may suggest that these relationships are weak or possibly even non-significant. While prospective IU may be a key maintaining factor in internalizing psychopathologies, inhibitory IU appears to be more important for understanding vulnerability for internalizing psychopathologies.

4.4. **Limitations and Future Direction**

The current study had several limitations which point to future directions of further research. While three of the four criteria for a vulnerability factor were assessed, the current study did not evaluate the most definitive criteria for vulnerability factors - whether it longitudinally predicts the onset of psychopathology. A longitudinal high-risk design is needed to verify whether inhibitory IU is in fact a vulnerability factor for MDD, PTSD, SAD, SUD, and GAD. However, such a study would require a substantial amount of time and a large number of participants with high levels of IU. Such individuals with elevated levels of IU could not have already developed psychopathology and, given the early age of onset of internalizing psychopathologies (Kessler et al., 2005), this would require the sample to be comprised of young children. While this high IU group would likely be at a higher risk for developing the aforementioned psychopathologies, only a portion of the group would go on to develop psychopathology and it may take years of follow-ups to assess and measure this occurrence.

Additionally, the current study had high rates of comorbidity between SUD and internalizing psychopathologies. In order to further explore whether inhibitory IU is in fact a vulnerability factor for SUD, future studies should evaluate if inhibitory IU is elevated before, during, and after episodes of SUD in individuals with no lifetime history of internalizing psychopathology. Finally, the results of this study provide further evidence that IU is a transdiagnostic vulnerability factor. Nolen-Hoeksema & Watkins (2011) argue that a weakness in transdiagnostic models of psychopathology is the lack of explanation of divergent trajectories, for example, why individuals that are high in IU develop certain internalizing psychopathologies and not others. Research is needed to determine what mechanisms link high levels of IU to specific internalizing psychopathologies. For instance, in the context of Nolen-Hoeksema and Watkins' (2011) heuristic for transdiagnostic models, heightened reactivity to unpredictable threat may be a moderator by which IU, a proximal risk factor, leads to fear-based disorders as opposed to distress disorders.

4.5. **Conclusion**

In summary, results suggested that IU is likely a transdiagnostic vulnerability factor for multiple psychopathologies including several internalizing psychopathologies (MDD, PTSD, SAD and trends for GAD) and possibly SUD (an externalizing psychopathology) as well. Additionally, results showed that inhibitory IU, and not total or prospective IU, exhibited stronger (but not definitive) evidence of being a vulnerability factor. Importantly, these effects cannot be explained by levels of N/NA, which is highly correlated with both psychopathology and IU. While these findings provide preliminary evidence suggesting that IU is a transdiagnostic vulnerability factor, further research is needed to evaluate whether IU meets all

criteria for a vulnerability factor and is therefore elevated in individuals before the onset of psychopathology.

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APPENDIX

TABLE I

IU LEVELS IN THOSE WITH CURRENT PSYCHOPATHOLOGY COMPARED TO THOSE WITH NO LIFETIME HISTORY OF PSYCHOPATHOLOGY

Diagnosis	Total	Prospective	Inhibitory
GAD (n=21)	4.800* (15.323***)	2.575† (8.699***)	2.347* (6.600***)
MDD (n=26)	5.686** (12.652***)	2.065 (6.275***)	3.700*** (6.433***)
OCD (n=28)	1.767 (7.325***)	.235 (3.520**)	1.617* (3.799***)
SAD (n=51)	3.875** (11.436***)	2.053* (6.294***)	1.997** (5.234***)
PD (n=12)	1.112 (7.058**)	.315 (3.573*)	.955 (3.550***)
Specific Phobia (n=75)	3.004** (7.508***)	2.042** (4.502***)	1.086* (3.067***)
PTSD (n=6)	6.260* (12.543***)	3.133 (6.682**)	2.974* (5.682***)
AUD (n=29)	-1.261 (4.201*)	-1.482 (1.750)	.418 (2.690***)
SUD (n=31)	2.399 (7.425***)	.415 (3.267**)	1.941** (4.018***)
Internalizing (n=150)	2.530** (6.708***)	1.442** (3.683***)	1.142** (3.067***)
Externalizing (n=51)	-1.330 (1.563)	-1.531* (.062)	.203 (1.506*)

Note. Beta coefficients outside of the parentheses are from models that included N/NA. Beta coefficients inside of the parentheses are from models that did not include N/NA. Values reflect differences between the means of each group. For example, the average total IU score for individuals with current GAD was 4.8 points higher than the average for those with no lifetime history of psychopathology when N/NA was included in the model.

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$

TABLE II

INTERACTION OF CURRENT INTERNALIZING AND EXTERNALIZING STATUS ON IU LEVELS

Diagnosis	Total	Prospective	Inhibitory
Current Internalizing (n=150)	2.146** (6.414***)	1.294* (3.611***)	.916* (2.854***)
Current Externalizing (n=51)	-2.671† (-.018)	-2.049* (-.577)	-.562 (.647)
Current Internalizing X Current Externalizing	3.251 (2.205)	1.265 (.729)	1.827† (1.328)

Note. Beta coefficients outside of the parentheses are from models that included N/NA. Beta coefficients inside of the parentheses are from models that did not include N/NA. Values reflect differences between the means of each group. For example, the average total IU score for individuals with current internalizing psychopathology was 2.146 points higher than the average for those with no lifetime history of internalizing psychopathology when N/NA was included in the model.

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$

TABLE III

IU LEVELS IN THOSE WITH REMITTED PSYCHOPATHOLOGY COMPARED TO THOSE WITH NO LIFETIME HISTORY OF PSYCHOPATHOLOGY

Diagnosis	Total	Prospective	Inhibitory
GAD (n=30)	1.462 (6.722***)	.411 (3.697**)	1.122† (3.070***)
MDD (n=147)	1.069 (5.700***)	.363 (2.899***)	.969* (2.940***)
OCD (n=15)	-.857 (3.997†)	-.918 (2.004)	.115 (2.016*)
SAD (n=49)	3.266* (6.875***)	1.345 (3.490***)	2.070*** (3.480***)
PD (n=29)	2.458 (7.324***)	1.241 (4.077**)	1.500* (3.364***)
Specific Phobia (n=29)	.295 (4.383**)	-.010 (2.296*)	.370 (2.093**)
PTSD (n=29)	2.625 (9.129***)	.829 (4.606***)	2.044** (4.612***)
AUD (n=121)	.616 (4.549***)	.203 (2.485**)	.763† (2.192***)
SUD (n=80)	.908 (3.762**)	-.195 (1.383†)	1.316** (2.467***)
Internalizing (n=137)	.767 (3.140***)	.293 (1.652**)	.595† (1.584***)
Externalizing (n=143)	.164 (1.817†)	-.008 (.902)	.320 (.982*)

Note. Beta coefficients outside of the parentheses are from models that included N/NA. Beta coefficients inside of the parentheses are from models that did not include N/NA. Values reflect differences between the means of each group. For example, the average total IU score for individuals in remission from GAD was 1.462 points higher than the average for those with no lifetime history of psychopathology when N/NA was included in the model.

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$

TABLE IV

INTERACTION OF REMITTED INTERNALIZING AND EXTERNALIZING STATUS ON IU LEVELS

Diagnosis	Total	Prospective	Inhibitory
Remitted Internalizing (n=137)	1.173 (3.616**)	.621 (1.987*)	.599 (1.657**)
Remitted Externalizing (n=143)	.733 (1.102)	.180 (.305)	.729 (.885)
Remitted Internalizing X Remitted Externalizing	-1.707 (-1.167)	-.822 (-.410)	-.805 (-.722)

Note. Beta coefficients outside of the parentheses are from models that included N/NA. Beta coefficients inside of the parentheses are from models that did not include N/NA. Values reflect differences between the means of each group. For example, the average total IU score for individuals in remission from an internalizing psychopathology was 1.173 points higher than the average for those with no lifetime history of internalizing psychopathology when N/NA was included in the model.

† $p < .10$, * $p < .05$, ** $p < .01$, *** $p < .001$

Kelly Correa

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Education

2016-Present **University of Illinois at Chicago**
M.A. Clinical Psychology (2018)
Ph.D. Clinical Psychology (in progress)
Advisor: Dr. Stewart Shankman

2008-2012 **University of San Diego, B.A., Psychology – Honors Program**
Honors Program Senior Thesis: Differences in marijuana use, using marijuana to cope, and stress in perfectionists

Awards

1. UIC Graduate College Abraham Lincoln Fellowship for Underrepresented Minorities ($22,000)
2. Best Paper Award of the 9th International Conference on Augmented Cognition 2015: EEG Coherence within Tutoring Dyads: A Novel Approach for Pedagogical Efficiency.
3. USD Student Research Grant: Correa, K. (November, 2011). Marijuana Use and Coping with Stress in Perfectionists ($500)
4. USD Student Research Grant: Correa, K. & Gutierrez de Velazco, C. (March, 2011). The effects of different types of conversations on long lasting stress hormones ($540)
5. USD Student Research Grant: Correa, K. & Gutierrez de Velazco, C. (May, 2010). Long lasting stress hormone associated with different types of stressors and conversations ($560)

Peer-Reviewed Publications

1. **Correa, K. A.,** Liu, H. & Shankman, S.A. (in preparation). Intolerance of uncertainty: A vulnerability factor for internalizing psychopathologies.
2. Stevens, E. S., Lieberman, L., Funkhouser, C., & **Correa, K. A.**, Shankman, S. A. (revised and resubmitted). Function follows form: Startle during threat longitudinally predicts functional impairment independent of DSM diagnoses. *Journal of Abnormal Psychology*.
3. Stone, B. T., **Correa, K. A.**, Brown, T. L., Spurgin, A. L., Stikic, M., Johnson, R. R., & Berka, C. (2015). Behavioral and neurophysiological signatures of benzodiazepine-related driving impairments. *Frontiers in Psychology*, 6, 1799.
4. **Correa, K. A.**, Stone, B. T., Stikic, M., Johnson, R. R., & Berka, C. (2015). Characterizing donation behavior from psychophysiological indices of narrative experience. *Frontiers in Neuroscience*, 9.
5. Stone, B., **Correa, K.**, Thor, N., & Johnson, R. (2015). EEG Coherence Within Tutoring Dyads: A Novel Approach for Pedagogical Efficiency. In *Foundations of Augmented Cognition* (pp. 697-706). Springer International Publishing.

Book Chapters

1. McEvoy, P. M., Carleton, R. N., **Correa, K.**, Shankman, S., & Shihata, S. (in press). Intolerance of uncertainty: diagnoses to dimensions. In B. Olatunji's (Ed.), Handbook of Anxiety and Related Disorders. Cambridge Press.
2. **Correa, K**. and Taylor, A. (2013). Memory in Movies. In *Encyclopedia of human memory*. (Vol. 2, pp. 742-755). Santa Barbara, CA: Greenwood Publishers.

Conference Presentations

1. **Correa, K. A.,** Lieberman, L., Stevens, E. S. & Shankman, S.A. (October 2017). Do ERP Measures of Predictable and Unpredictable Threat Responding Run in Families?. Poster presented at the annual meeting of the Society for Psychophysiological Research, Vienna, Austria.
2. Funkhouser, C., Carrillo, V., **Correa, K. A.** & Shankman, S.A. (October 2017). Physiological Habituation to Aversiveness and Non-Suicidal Self-Injury: A Test of Acquired Capability for Suicide. Poster presented at the annual meeting of the Society for Psychophysiological Research, Vienna, Austria.
3. **Correa, K. A.**, Lieberman, L., Liu, H. & Shankman, S.A. (April 2017). Does Intolerance of Uncertainty Run in Families?. Poster presented at the annual meeting of the Anxiety and Depression Association of America, San Francisco, CA.
4. Tan, V., Ankrom, A., **Correa, K.**, Crystal, M., & Berka, C. (October 2015). Event-related potential assessment of cognitive tasks in PTSD. Poster presented at the 45th annual meeting of the Society for Neuroscience, Chicago, IL.
5. **Correa, K.**, Kuckertz, J.M., & Amir, N. (November 2012). The role of intolerance of uncertainty in generalized anxiety disorder. Poster presented at the 46th annual meeting of the Association for Behavioral & Cognitive Therapies, National Harbor, MD.

Research Experience

2016-Present **Graduate Research Assistant, Chicago Laboratory of Emotion and Physiology**

- Assist with the collection and processing of psychophysiological data (EMG, EEG, EKG), perform statistical analyses using SPSS and R, and develop software tools to streamline data processing for NIMH-funded grants.

2015-2016 **EEG Analyst, Brain Treatment Center**

- Provided reports on EEG data for patients seeking treatment at a neuropsychiatric outpatient clinic that provides Magnetic Resonance Therapy, a TMS-based treatment, for various disorders and medical conditions such as ASD, PTSD and other anxiety disorders, depression, Alzheimer's, substance abuse, TBI's, strokes, and chemotherapy-related cognitive impairment.
- Selected clean, artifact free segments of eyes closed EEG's to be processed with internal software as well as Persyst's Insight II.
- Screened for any epileptiform activity present in patients' EEGs.

- Provided baseline and follow-up reports tracking the presence of EEG characteristics that may contribute to a patient's symptomology.
- Developed software tools to automate comparisons of EEG-based metrics.

2013-2015 **Research Associate, Advanced Brain Monitoring Inc.**

- Analyzed psychophysiological data using SAS and IBM SPSS and reported the results in scientific papers and monthly reports.
- Assisted in project management for multiple projects by designing study protocols, tasks, and measures; ensuring that the statement of work and deliverables were being met in accordance with funding milestones; and conducting and participating in project meetings and phone conferences for study design and work flow.
- Served as IRB coordinator for multiple projects by creating, editing, and submitting IRB documents (protocols, consents, questionnaires, etc.); managing existing IRB protocols; and closing out protocols upon completion of studies.
- Assisted in the management of Research Technicians and EEG data acquisition internally as well as off-site.
- Performed sLORETA analyses using NeuroGuide software and provided individualized reports based on these analyses.

2012-2013 **Research Technician, Advanced Brain Monitoring Inc.**

- Dramatically reduced data processing time by writing macros in Microsoft's VBA that enabled project-specific batch data processing of EEG (Event-Related Potentials and Power Spectral Density), GSR, and ECG (HR and HRV) data.
- Collected, managed, organized, and prepared data for statistical analysis.
- Developed and maintained a participant scheduling and tracking system.
- Designed protocol templates for human participant studies.
- Edited and contributed to grant submissions and scientific papers for journal submission.

2011-2012 **Research Assistant, Center for Understanding and Treating Anxiety**

- Designed a research study investigating intolerance of uncertainty in GAD.
- Programmed a computer-based behavioral measure of intolerance of uncertainty.
- Phone screened potential participants for likelihood of meeting DSM-IV criteria for GAD.
- Ran GAD, Social Phobia, and non-anxious participants through various study protocols including those involving TMS and EMG.

2009-2012 **Research Assistant, University of San Diego - Biopsychology Lab**

- Collected and analyzed saliva samples for levels of cortisol and alpha-amylase.
- Wrote research grant proposals and conference posters.
- Ran participants through various study protocols.
- Entered and analyzed data using IBM SPSS.

Clinical Experience

2016-Present **Office of Applied Psychological Services**

- Conduct diagnostic assessments
- Administer neuropsychological assessments and interpret, document, and communicate the results of said assessments to clients, supervisors, and colleagues.
- Provide empirically-supported psychological treatments for clients

Teaching Experience

2017-Present **Teaching Assistant, University of Illinois at Chicago**

- Field Work in Applied Psychology (Fall 2017)
- Behavioral Neuroscience (Spring 2018)