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Understanding Comorbid Depression in the Context of PTSD Through Underlying Dimensions

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Abstract

Depression co-occurs with Posttraumatic Stress Disorder at a high rate, resulting in numerous complications for pathology and treatment. More research is needed regarding variables that may explain this common comorbidity. One possible variable is that of underlying dimensions, latent factors that give rise to these manifestations of psychopathology. This study explored potential underlying mechanisms of comorbid PTSD and depression, including negative affect, rumination, emotion dysregulation, neuroticism, and behavioral inhibition. While previous studies have investigated these dimensions individually, there is a dearth of research that simultaneously investigates multiple dimensions or determines the relative contributions of underlying dimensions to psychopathology. Thus, the current study aimed to analyze more comprehensively how underlying psychological constructs predict PTSD and depression. A sample or 717 adults answered an online questionnaire battery surveying symptoms of PTSD and depression as well as the proposed underlying constructs. The relationships between the underlying dimensions and psychopathology were analyzed using a number of statistical methods. Overall, these psychological and behavioral constructs were shown to be related to PTSD and depression, though the exact nature and strength of the relationship varied depending on psychological test. Negative affect, emotion dysregulation, and neuroticism emerged as the most significant predictors. Limitations and clinical implications are discussed.

Depression is the most frequently coexisting disorder of PTSD. Some studies cite that as high as 85% of individuals with PTSD also have depression (Spinhoven, Pennix, van Hemert, Rooij, & Elzinga, 2014). There are several consequences of having comorbid PTSD and depression, compared to experiencing either disorder alone. For example, the comorbidity of these two psychological conditions is associated with more severe symptoms than either disorder alone, as well as lower global functioning (Gros, Price, Magruder & Frueh, 2012; Shalev et al., 1998). In particular, in comparison to PTSD, comorbid PTSD and depression has been shown to be related to elevated dysphoria and re-experiencing, higher levels of negative affect, lower levels of positive affect, and more functional impairment in domains such as work and family (Post, Zoellner, Youngstorm, & Feeny, 2011). Co-occurrence of PTSD and depression has also been shown to be associated with more functional impairment such as impaired health care utilization, higher severity of psychiatric medical illness, and lower quality of life than when PTSD or depression occur in isolation (Campbell et al., 2007). Individuals with PTSD and depression experience more cognitive impairment than those with PTSD alone, such as deficits in verbal memory, divided attention, and working memory (Nijdam, Gersons, & Olff, 2013).

Comorbidity is also associated with worse therapy outcomes than either disorder alone, and it has been shown that comorbidity compromises treatment response of these disorders (Gros et al., 2012). For example, this comorbidity has been shown to negatively impact compliance with therapy homework and participation in prolonged exposure exercises (Scott & Stradling, 1997). Because of the high rate of comorbid depression in

PTSD and the deleterious effects of this comorbidity, the current study aims to increase our understanding regarding comorbid depression within the context of PTSD.

It should be acknowledged that there are multiple methods of approaching understanding comorbid psychopathology. One such method is explaining how two disorders arise through a common factor (Najavits, 2009). The common factor, or factors, can be stood as dimensions that etiologically underlie the disorders. Examining potential underlying dimensional constructs these two disorders may share is an important yet understudied area of research. As such, this project will focus on investigating these psychological dimensions of PTSD and depression with the goal of better understanding this complex comorbid relationship.

This paper will first acknowledge some of the differences between these disorders, as well as an overview of different explanations of comorbidity. The rationale for studying underlying dimensions will be discussed. Then, although not an exhaustive list, variables that may potentially serve as underlying dimensions will be outlined. Methodology regarding analyzing these relationships as well as potential limitations will be discussed.

Distinctions Between PTSD and Depression

A diagnosis of PTSD requires a traumatic experience to occur, specifically a trauma that involves exposure to actual or threatened death, serious injury, or sexual violence (i.e. a Criterion A trauma). Although depression is often linked with stressful life events, it does not necessitate a trauma. Gros, Price, Magruder and Frueh (2012) note that in a study of over 1000 veterans, the only symptom that differentiated participants with PTSD from those with MDD was the presence of a Criterion A trauma. In this study,

all individuals were given the PTSD Checklist (PCL), and afterwards, veterans were assessed for traumatic history using the Trauma Assessment for Adults Questionnaire (TAA). For those who experienced a Criterion A traumatic event, the Clinician Administered PTSD Scale (CAPS) was given. Individuals with MDD reported similar symptom severity on the PCL compared to those with diagnosed PTSD, but this group could not be diagnosed with PTSD due to their referent trauma not meeting the characteristics described in the DSM. Of note, the authors did not specify what their criteria was for a Criterion A trauma, and it is unclear what referent stressful life event the non-Criterion A group would have been reflecting on while filling out the PCL. Nonetheless, trauma exposure may be an important distinction between PTSD and MDD.

Other distinctions can be surmised from the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 2013). For example, fear is more obviously salient in PTSD, as well as symptoms brought about by fear associations, such as exaggerated startle and hypervigilance. PTSD also more frequently involves symptoms of dissociation. Recurrent thoughts about death are more common in depression and match with depressive symptomology, though thoughts about death are certainly not uncommon in PTSD, particularly if the Criterion A trauma posed a significant risk of death. Weight and appetite change are more common in depression. These distinctions imply that although there are many commonalities between PTSD and depression, they are unique disorders in several ways. This uniqueness suggests that there are underlying dimensions of each disorder that are not shared by both.

As further evidence for distinctness, a study using latent profile analysis investigated a large trauma-exposed sample from an epidemiological dataset (Cao et al.,

2015). The authors showed that a larger portion of people were classified as having predominantly PTSD or predominantly depression compared to the portion of people that were classified in the comorbid group. The authors concluded that PTSD and depression are independent sequelae from trauma rather than a manifestation of a single psychopathology.

There are several potential biological distinctions between PTSD and depression. For example, Savic, Knezevic, Damjanovic, Spiric, and Matic (2012) discuss the dexamethasone suppression test (DST), which is used to test the self-regulation of Hypothalamic-Pituitary-Adrenal (HPA) axis via glucocorticoid receptors. They explain that during this test, individuals with PTSD typically hyper-suppress cortisol, while those with depression typically hypo-suppress cortisol. However, the authors also note that suppression on the DST may be more influenced by traumatic and stressful life event exposure, as opposed to the specific psychopathologies. Other research related to stress reactions show an exaggerated startle response for individuals with PTSD (Griffin, 2008). In contrast, individuals with an anxiety disorder and comorbid MDD, compared to an anxiety disorder alone, show a reduced startle (Yancey, Vaidyanathan, & Patrick, 2015. Additionally, individuals with comorbid PTSD and MDD, compared to MDD alone, show a greater startle response (Jovanovic et al., 2010). In sum, PTSD and disorders that share a similar fear response show an increased startle reaction and stress response, consistent with the hypervigilance present in PTSD, while MDD facilitates a blunted startle response.

Additionally, Brain-Derived Neurotrophic Factor has been found to be consistently elevated in individuals with PTSD (Hauck et al., 2010), but reduced in

individuals with depression (Levinson, 2006), though the nature of BDNF shows more mixed evidence in depression. There are also many biological similarities between individuals with PTSD and depression, which will be discussed throughout this paper.

Notably, much of the difference between PTSD and depression lies in their respective diagnostic criteria. However, the DSM consists of arguably untested and prescriptive criteria in many cases for disorders. Despite this issue, its criteria have driven the empirical literature, rather than using scientific evidence to support underlying mechanisms or dimensions.

Potential Reasons for PTSD-Depression Comorbidity

Najavits et al. (2009) outlines causal explanations of comorbidity for any combination of psychological disorders, and the authors' argument can be applied to the comorbidity of PTSD and depression. The first explanation is that Disorder X causes Disorder Y, and the second explanation is that Disorder Y causes Disorder X. Thus, the comorbidity between PTSD and depression can first be analyzed in whether or not each disorder causally affects the other. In one longitudinal study, major depression at baseline was a strong predictor of subsequent PTSD years later (Breslau & Schultz, 2013). However, there is evidence that a history of major depression increases the risk for exposure to traumatic events (Breslau, 2009), so it may not be that depression in and of itself causes PTSD. Additionally, the reverse has been shown; there has been found to be an increased risk for major depression in trauma-exposed persons with PTSD, but not for trauma-exposed persons without PTSD. Horesh et al. (2017) discusses models of either PTSD or depression as antecedents for the other disorder, and concluded through his literature search and longitudinal study that the relationship between PTSD and MDD is

bidirectional, and that neither disorder is secondary to the other. The authors also suggest that “core features” may influence this comorbidity.

If there is a relatively equal amount of evidence that PTSD causes depression and that depression causes PTSD, it is plausible that neither fully causes each other. The studies that find these disorders as risk factors for one another may be unintentionally excluding another variable or other variables that are the true cause of both. Najavits et al. (2009) continues that each disorder may impact the course of the other, even if not caused by it. There is some evidence for this explanation, as will be discussed in other sections of the paper.

The next causal explanation put forth by Najavits et al. (2009) is that each disorder arises independently, without any relation between them. A comorbidity rate of up to 85% between PTSD and depression makes this explanation unlikely. Additionally, given the enormous biological, psychological, and social risk factors that these disorders share, it is arguably implausible that they would be truly independent in their origin.

An additional explanation that has been put forth in the literature for the high comorbidity between PTSD and depression is the occurrence of symptom overlap. PTSD and depression do share several symptom commonalities, including persistent negative beliefs, persistent negative emotional state, anhedonia, detachment from others, diminished interest in activities, irritability, difficulty concentrating, and sleep disturbance. This overlap could indicate that comorbidity is a superficial artifact of an inefficient diagnostic classification system, rather than a causal phenomenon. However, in a study of 766 trauma-exposed individuals from the National Comorbidity Survey, Elhai et al. (2011) found that removing overlapping symptoms did not alter comorbidity,

which in this study was defined as the statistical unidimensionality of PTSD and depression. Therefore, the comorbidity must be due to other mechanisms.

Finally, Najavits (2009) states that both Disorder X and Disorder Y are caused by some other factor. This argument will be the focus of study in this paper, and the "other factor" proposed is a series of factors: underlying dimensions. Hierarchical models of psychopathology propose that there is a latent organization of higher order factors or dimensions that underlie psychological disorders, and are linked to etiological factors (Luyten, Vliegen, Boudewijn, Houdenhove, & Blatt, 2008). This model assumes that many disorders share etiological factors, a concept in developmental psychopathology known as multifinality. It is possible that there are underlying characteristics that contribute to the development of both PTSD and depression, as opposed to traits that are simply shared by the disorders or a consequence of the disorders. These commonalities would then be causally responsible for the manifestations of PTSD and depression.

Support for Underlying Dimensions

There are multiple reasons to believe PTSD and depression share underlying mechanisms. Firstly, the comorbidity rate is likely too high to be a coincidence, but not high enough to assume that one disorder in and of itself causes the other. This hypothesis can be investigated further by examining the odds of developing one of these disorders if one has the other diagnosis. For example, Pre-trauma depression has been found to be significantly correlated with PTSD symptoms at 1-month and 3-month time points following an earthquake ($r = .41$) (Kuijer, Marshall, & Bishop, 2014). In another study of 1,281 college students, pre-trauma distress measured by the Depression Anxiety Stress Scale was significantly related to PTSD two months after the initial assessment ($r = .46$)

(Frazier et al., 2011). A history of MDD was found to lead to a threefold increase in risk of PTSD (Breslau & Schultz, 2013). As further evidence, the DSM lists prior major depressive disorder as a risk factor for PTSD, and lists MDD as a common comorbidity in the PTSD section. However, the DSM does not list prior PTSD as a risk factor for MDD and it does not list PTSD as a common comorbidity in the major depressive disorder section. These studies, as well as the DSM structure, may imply that MDD as etiologically involved in PTSD is more likely than the reverse. However, although the correlation coefficients are significant regarding the relationship between earlier depression and later PTSD, the strength and causality of this relationship is still under debate.

Similarly, it would be important to examine the odds of developing depression if one already has PTSD. Although there is less research in this direction, PTSD has been shown to partially mediate the relationship between trauma exposure and MDD (Subica, Claypoole, & Wiley, 2012). There has also been found to be a significant risk for MDD in individuals with PTSD, but not in trauma-exposed individuals without PTSD (Breslau, Davis, Peterson, & Schultz, 2000). In a study of adolescents, PTSD preceded or emerged at the same time as depression in 70% of cases, although this group had a sample size of 10 individuals (Giaconia et al., 1995).

Another potential reason to believe that PTSD and depression share dimensions comes from the treatment literature. Psychological treatment of PTSD typically leads to improvement in depressive symptoms (Brunello et al., 2001). There is also an overlap in medications that are effective in reducing PTSD and depression symptoms (Brunello et al., 2001). It should be noted that analyzing shared treatment effectiveness and

concluding proof of a shared underlying mechanism might be considered by some researchers and clinicians to be backwards reasoning. It is therefore important to alternatively consider comorbidity from an etiological perspective.

Outside of common psychological etiology, one could argue that an underlying dimension of PTSD and depression is trauma exposure. Trauma exposure is a necessary precursor to PTSD, and stressful life events (which by definition includes trauma exposure) are thought to be precipitants of MDD (American Psychiatric Association, 2013). However, not all people who experience a traumatic event develop PTSD or depression. Thus, there may be characteristics of trauma exposure that increases an individual's risk for the development of PTSD and/or major depression. One such characteristic is whether a trauma was perpetrated through interpersonal means (e.g. physical or sexual abuse or assault), as opposed to non-interpersonal traumas (e.g. natural disaster, car accident, severe illness). Previous research has illustrated that interpersonal trauma is associated with higher rates of psychopathology and distress, including more severe PTSD symptoms. Thus, the current study aims to establish interpersonal trauma as more predictive of psychopathology than non-interpersonal trauma. Further, if underlying dimensions are to be understood as precursors to PTSD and depression, then by extension, interpersonal trauma may be more predictive of those dimensions. Therefore, the study also aims to establish the relationship between interpersonal trauma and the proposed underlying dimensions.

Advantages To Studying Psychopathology Through Underlying Dimensions

There is still much to be learned about the etiology of these disorders. Additionally, individuals are more likely to have both PTSD and depression than PTSD

alone (Gros, Price, Magruder, & Frueh, 2012). Therefore, understanding how these disorders appear in combination may be more practical than either in isolation. The field seems to be moving towards more transdiagnostic and translational research that supports this aim. For example, the Research Domain Criteria (RDoC), put forth by the National Institute of Mental Health, are designed to integrate multiple levels of information to better understand basic dimensions of human functioning (Cuthbert & Insel, 2013). The RDoC is a new research classification system that synthesizes underlying dimensions of neurobiology, psychological experiences, as well as observable behavior. One purpose of this system is to pose an alternative understanding and labeling of disorders from the current classification systems, the Diagnostic and Statistical Manual of Mental Disorders (DSM) and the International Classification of Diseases (ICD). The RDoC may result in more targeted, effective treatment in line with precision medicine. Researchers and clinicians can tailor treatment to the individual based on maladaptive expressions of underlying dimensions rather than targeting broad disorders that may present very differently from person to person.

The RDoC outlines major domains that include underlying dimensions, and they can be used as a guide for navigating this research. The first domain is negative valence systems. These systems are primarily responsible for responses to aversive situations or context, such as fear, anxiety, and loss. The RDoC also has a domain covering positive valence systems. These systems are primarily responsible for responses to positive motivational situations or contexts, such as reward seeking, consummatory behavior, and reward and habit learning. There is also the domain of cognitive systems, which includes dimensions such as attention and memory. Another domain, systems for social processes,

includes dimensions such as attachment and response to faces. Finally, the domain of arousal/regulatory systems includes dimensions such as circadian rhythms and physiological arousal.

One current issue is that the RDoC is a new system of studying psychopathology. There is limited information available on potential underlying mechanisms to study, and these variables are not often operationally defined very clearly. However, given the accumulating evidence of transdiagnostic mechanisms, this new system is worth devoting research efforts to.

Treatment Implications

In general, studying etiology can lead to a better understanding and treatment of disorders. There is a potential for more effective and efficient therapies if we identify and treat underlying contributors to psychopathology rather than the symptom manifestations. This identification would be particularly advantageous if it allows us to treat two related disorders simultaneously, rather than executing diagnostic-specific treatments for one disorder, then the other.

The Unified Protocol for the Transdiagnostic Treatment of Emotional Disorders approaches this goal, as it is designed to treats both PTSD and depression, as well as anxiety disorders. The Unified Protocol targets a “general neurotic syndrome” (Farchione et al., 2012). The intervention also targets four core modules: increasing emotional awareness, facilitating flexibility in appraisals, identifying and preventing behavioral and emotional avoidance, and situational and interoceptive exposure to emotional cues (Ellard, Fairholme, Boisseau, Farchione, & Barlow, 2010). These four modules may illustrate underlying mechanisms of pathology that leads to PTSD and depression. They

also map on to some of the subsequently discussed underlying dimensions, and some dimensions have specifically been discussed in their relationship to the Unified Protocol (e.g. neuroticism and experiential avoidance, which can be considered similar to behavioral inhibition) (Gallagher, Thompson-Hollands, Bourgeois, & Bentley, 2015). Initial outcome data (N = 18) illustrates effectiveness for unified protocol for a variety of psychopathologies (Ellard, Fairholme, Boisseau, Farchione, & Barlow, 2010). Additionally, use of the protocol results in a high response rate for comorbid diagnoses. However, in this study sample, there was only one individual with a principal diagnosis of PTSD and two with MDD. Therefore, results have potentially limited generalizability for other individuals with these disorders. Still, the results are promising, and the protocol is theoretically sound, indicating that a treatment aimed at underlying dimensions and not discrete psychological disorders is a viable option.

In addition to the Unified Protocol, other interventions have been recently developed with goals to treat PTSD or traumatized populations with comorbid pathology. For example, Transdiagnostic Behavior Therapy has been successfully implemented for veterans with affective disorders (Gros, Szafranski, & Shead, 2016). “Vets Prevail”, an online intervention using general CBT principles, was shown to be effective in treating PTSD and depression (Hofboll, Blais, Stevens, Walt, & Gengler, 2016). In particular, treatments targeting PTSD and comorbid substance abuse are prevalent, such as Concurrent Treatment of PTSD and Substance Use Disorders Using Prolonged Exposure (COPE) and Seeking Safety (Westphal, Aldao, & Jackson, 2017). Importantly, these treatments modulate mechanisms that may underlie PTSD and depression, such as emotion dysregulation. Although comorbid depression was not always the target in these

interventions, they illustrate that transdiagnostic treatments are increasingly viable intervention options. Researchers have also proposed using RDoC mechanisms to track changes in the treatment of trauma reactions, PTSD and comorbid pathology (Stover & Keeshin, 2016; Zambrano-Vazquez et al., 2017).

Proposed Underlying Dimensions of PTSD and Depression

The negative valence systems can include dimensions such as negative effect, rumination, behavioral inhibition, emotion dysregulation, and neuroticism, and will be the primary focus of the underlying dimensions subsequently discussed. The positive valence systems can include dimensions such as positive affect. Cognitive systems will not be discussed in depth in this study, although it should be noted that rumination and neuroticism are highly related to cognitive systems, and this system may be a potential future direction for understanding this comorbidity. Systems for social processes also will not be discussed in depth in this paper, as there is currently limited research on how this domain relates to the PTSD-depression comorbidity. Arousal and regulatory systems will also not be discussed in depth in this project, aside from the previously mentioned differences in startle response.

The following underlying dimensions are not an exhaustive list of the potential contributors to comorbid PTSD and depression. Rather, they are the dimensions with a currently robust literature that fit well into the RDoC framework.

Heightened Negative Affect

Relationship with PTSD and depression. Negative affect is the predisposition to experience negative emotionality and to have a negative view of self (Watson & Clark, 1984). Individuals with PTSD or depression have higher negative affect (Bradley et al.,

2011; Kraal, Waldron-Perrine, Pangilinan, & Bieliauskas, 2015; Zoellner, Pruitt, Farach, & Jun, 2014). Further, individuals with PTSD have negative affective instability, meaning higher than average fluctuations in mood (Kashdan, Uswatte, Steger. & Julian 2006). A study of a general trauma exposed population using statistical hierarchical modeling found that the direct effects of negative affect on PTSD were similar in size, with the authors concluding that trait negative affect is a shared vulnerability that links these disorders (Post, Feeny, Zoellner, & Connell, 2015). Individuals with PTSD or depression also have lower self-esteem, which can be seen as a manifestation or consequence of negative affect (Kashdan, Uswatte, Steger. & Julian 2006).

It should be noted that there are difficulties inherent in conceptualizing negative affect as an underlying dimension of PTSD or depression, given that both disorders mention types of negative affect in their diagnostic criteria. Thus, it is intuitive that, statistically, measures of negative affect will often relate to measures of symptomology, given that they overlap. Still, it is important to discuss negative affect as a dimension of PTSD and depression, because if the comorbidity of these conditions or the conditions themselves can be heavily explained by negative affect, that has implications for treatment and diagnostic classification.

Neurological background. Neurological evidence can strengthen the argument behind underlying dimensions of comorbid PTSD and depression. There is increased activation in neural structures implicated in negative affect in both PTSD and depression (Ellard, Fairholme, Boisseau, Farchione, & Barlow, 2010). Overlapping neurological abnormality in PTSD and depression may contribute to propensity towards negative affect. For example, hyperexcitability of limbic structures, coupled with disrupted or

limited inhibitory control by cortical structures, distinguish individuals with anxiety and mood disorders from healthy controls, and may be one possible explanation for the increased intensity and frequency of negative emotional experience among individuals with these disorders (Wilamowska et al., 2010). Additionally, a hyperactive amygdala has been found in individuals with depression (Buchheim et al., 2012) and PTSD (Etkin & Wager, 2007; Zoellner, Pruitt, Farach, & Jun, 2014). The amygdala is implicated in emotional learning, fear, anxiety. There has been found to be hyperactivity in the insula for individuals with PTSD, as well as a hyperactive connection between the insula and limbic structures in those with depression (Avery et al., 2014). The insula is linked with emotional processing, and is clearly relevant for negative affect, although the insula could also logically be placed with other dimensions previously discussed in the literature, such as distress, dysphoria, or reward responses.

Although biological factors are not the primary scope of this study, there are other important biological variables to consider in addition to larger neurological anatomy, such as genetics, as the RDoC encourages researching dimensions on multiple levels of analysis. Biological links between these psychological variables and PTSD or Depression support the notion that they are underlying dimensions. There are genetic influences on negative affect, supporting its conceptualization as an underlying dimension. For example, negative affect has been shown to be heritable in a study of twins and multigenerational families (Baker, Cesa, Gatz, & Mellins, 1992). Further, in another twin study, Wichers et al. (2007) argue that the genetic risk of depression may be explained in part by negative affect. Although this study does not address PTSD, the authors discuss negative affect as an endophenotype for psychopathology, which offers support as an

underlying dimension, as well as the relationship between negative affect and stressors, which could relate to trauma and PTSD.

Cultural influences. There is little research regarding the relationship between culture and underlying dimensions. Thus, it is important to analyze any cultural variations of these psychological dimensions, which may have implications for how we understand these constructs. For example, Iranians have been shown to score higher on measures of negative affect than Americans (Joshanloo & Bakhshi, 2015). This study, conducted with national health data from over 2,000 Americans and 2,000 Iranians, concludes with a discussion of cultural variables that may have led to these results, such as a collectivistic and less wealthy overall society in Iran. The author's discussion points to the importance of analyzing larger societal variables in addition to biological variables to have a more comprehensive understanding of underlying dimensions.

Rumination

Relationship with PTSD and depression. Rumination is defined as the tendency to have persistent, uncontrollable, and intrusive thoughts (Roley et al., 2015). Repetitive and anticipatory rumination have been shown to moderate the relationship between PTSD and MDD symptoms (Roley et al., 2015). Trauma-related rumination has been shown to mediate the effects of depression on trauma intrusions. (Kubota, Nixon, & Chen, 2015). Compared to non-traumatized depressed patients, individuals with depression who have experienced trauma and individuals with PTSD ruminate more (Birrer & Michael, 2011).

Rumination is also a maladaptive form of cognitive coping (Roley et al., 2015). This coping style may manifest in these disorders in different but related ways. Individuals with PTSD ruminate on the causes and consequences of PTSD as an

avoidance strategy in order to resist processing the traumatic memory itself (Roley et al., 2015), whereas individuals with depression may ruminate on what they have done wrong or actions they haven't taken, leading to the excessive guilt that is commonly present in the disorder, but also in attempts to potentially avoid future distress.

Neurological background. Neurological abnormalities in PTSD and depression may contribute to ruminative tendencies. For example, there is hyperactivation of the medial prefrontal cortex in PTSD (Fonzo et al., 2010) and depression (Buchheim et al., 2012). This area of the brain is implicated in complex cognitive functioning. This hyperactivity, combined with dysregulation of the limbic structures, results in a "worry circuit" that drives rumination and the accompanying negative emotionality.

There is some mixed evidence regarding exactly how rumination is neurologically implicated in PTSD versus depression. For example, concerning individuals with depression, there is heightened connectivity within a cluster of regions known as the default mode network, which is thought to be responsible for the self-referentially, internally generated thoughts that are characteristic of rumination (Green & Ostrander, 2009). However, there has been shown to be decreased default mode connectivity in PTSD (Koch et al., 2016). Although these neurological findings seem contrary to one another, there is evidence that rumination is heightened in both disorders and that brain regions associated with this cognitive style are disrupted. The exact mechanisms by which rumination arises for each of these disorders or potential differences in the cognitive phenomenology may be further elucidated with future research.

A twin study estimated a 21% heritability of brooding and a 27% heritability of reflection, components of the Ruminative Response Scale (Moore et al., 2013). The

heritable influences on brooding were also shown to account for the majority of the relationship between brooding and depression. A similar relationship between rumination and depression that was largely accounted for by heritability has been shown in two other twin studies (Chen & Li, 2013; Johnson, Whisman, Corley, Hewitt, & Friedman, 2014).

Cultural influences. Freightman (2009) conducted a study in a small sample of individuals exposed to Hurricane Katrina prior to receiving treatment for their symptoms of traumatic stress or depression. Rumination was shown to be higher in Caucasian Americans following a trauma, compared to African Americans. The authors cite literature regarding high levels of resilience in the African-American community, and conclude statistically and theoretically that the lower rumination in African Americans enables them to be resilient to depression, which can perhaps extend to other psychopathology, such as PTSD.

Emotion Dysregulation

Relationship with PTSD and depression. Emotion dysregulation can be defined as a deficit in the ability to regulate intense and shifting emotional states (Bradley et al., 2011). In a study of 530 individuals recruited from the waiting rooms of medical centers, emotion dysregulation was shown to predict PTSD and depression by accounting for a large portion of variance of the symptoms of those disorders (Bradley et al., 2011). Moreover, the effects of emotion dysregulation were significant above the effects of childhood trauma exposure and negative affect. This finding illustrates that not only does negative emotionality contribute to this comorbidity, but also that the ability to manage and modulate that negative emotion is crucial. Additionally, this study provides further evidence that while traumatic stress is a significant vulnerability for PTSD and

depression, these life experiences may not be as impactful without a deficit in the underlying psychological abilities to adequately respond to that stress and manage the negative emotion that results from it. Particularly given the re-experiencing components of PTSD that lead an individual back to the emotional states present at the time of their trauma exposure, individuals with PTSD are unable to adapt to the consistent demand to regulate that emotional state.

This transdiagnostic variable complements previously discussed underlying dimensions. Whether negative emotionality is best described as negative affect, distress, or dysphoria, an individual who can more adaptively modulate those emotional states may not develop the dysfunction that defines psychological disorders. Other studies have similarly found that emotion dysregulation uniquely predicts PTSD and depression severity (Fairholme et al., 2013). Additionally, perceived emotional control, which can be considered to be a perception of emotion regulation, has been shown to moderate the relationship between neuroticism and generalized anxiety disorder (Bourgeois & Brown, 2015). While this study does not look at PTSD or depression, this model can be applied to the current discussion, with the idea that some underlying dimensions of PTSD and depression may influence the expression of other underlying dimensions, creating complexity in the understanding of psychopathology.

Although emotion dysregulation typically refers to overactivity of emotional experience and response, particularly within clinical settings, the opposite phenomenon can certainly be considered dysregulation as well. Numbness, or the relative absence of emotional response, is a common feature of both PTSD and depression (Gros, Price, Magruder, & Frueh, 2012). This numbness may be an underlying dimension of or

contributor to other symptoms of both pathologies. For example, numbness may relate to detachment and dissociation in PTSD and anhedonic tendencies in depression.

Neurological background. Overlapping neurological abnormality in PTSD and depression may contribute to difficulties in controlling emotional activation and expression. For example, there is hypoactivation of the dorsolateral prefrontal cortex in PTSD (Zoellner, Pruitt, Farach, & Jun, 2014), hyperactivation of the medial prefrontal cortex in PTSD (Fonzo et al., 2010; Hopper, Fewen, van der Kolk, & Lanius, 2007), and hyperactivation of medial prefrontal cortex in depression (Buchheim et al., 2012). As previously mentioned, the prefrontal cortex is implicated in complex cognitive functioning. Dysregulation in this region, whether it be hypo or hyperactivation, interferes with an individual's ability to cognitively regulate their emotional experience.

Genetic variations in the HPA axis have been shown to predict reduced connectivity between the amygdala and the frontal gyri, caudate, and parahippocampal gyrus (Pagliaccio et al., 2015). This illustrates a disruption between the limbic system of the brain, responsible for emotion, and cortical structures responsible for cognitive regulation. This disruption was shown to predict later difficulties in emotion regulation skills. A twin study has also shown a relationship between genetic factors and emotion regulation (Wang & Saudino, 2013).

Cultural influences. Cultures may also differ in typically practiced strategies of emotion regulation, making it difficult to compare the ways in which emotion regulation shapes psychopathology. For example, Kwon, Yoon, Joormann, and Kwon (2013) found that Korean college students are most likely to brood in attempts to regulate emotion, whereas Americans engage in more anger suppression, Further, reappraisal is highly

linked with depressive symptoms in Koreans whereas anger suppression relates most strongly with depressive symptoms in Americans. Tsai and Lau (2013) found that self-enhancement as an emotion regulation strategy may buffer against distress for European Americans, but emotion suppression may be more adaptive for Asian Americans. These two studies point to the complexities regarding the intersections between underlying dimensions, cultural context, and psychopathology. Complicating matters further, there is evidence to suggest that cultural variations in emotion regulation may be attributed to personality traits, including neuroticism (Matsumoto, 2016).

Neuroticism

Relationship with PTSD and depression. Neuroticism is thought to be a stable personality trait reflecting vulnerability to negative emotional experiences, particularly worry (Breslau & Schultz, 2013; James, Kampen, Miller, & Engdahl 2013). A great deal of research has linked neuroticism to various independent psychopathologies. In latent factor modeling, neuroticism has been linked with depression, as well as several anxiety disorders that share traits with PTSD, including generalized anxiety disorder, obsessive compulsive disorder, and social anxiety disorder (Rosellini & Brown, 2011). Neuroticism is a risk factor for depression, and accounts for a substantial portion of the heritability of depression, as stated in the DSM-5 (American Psychiatric Association, 2013).

There is also evidence that neuroticism links PTSD and depression. A study of a general trauma exposed population using statistical hierarchical modeling found that the direct effects of neuroticism on PTSD and MDD were similar in size (Post, Feeny, Zoellner, & Connell, 2015). The authors concluded that trait neuroticism is a shared vulnerability that links these disorders.

There are several studies that have examined neuroticism as an underlying dimension of PTSD and depression, and importantly, many of these studies are longitudinal, which is a rare strength for research in this area (James, Kampen, Miller, & Engdahl 2013; Parslow, Form, & Christensen, 2005; Spinhoven, Pennix, van Hemert, & Rooij, 2014). For example, in a study of 271 veterans from Operation Enduring Freedom/ Operation Iraqi Freedom (OEF/OIF), pre-deployment neuroticism predicted later depression and PTSD (James, Kampen, Miller, & Engdahl, 2013). Higher pre-trauma levels of neuroticism predicted subsequent development of PTSD at six months, 12 months, and 24 months post-deployment. By examining multiple time points, this study both provides the longitudinal evidence supporting neuroticism as a causal contribution to psychopathology, and also illustrates the consistency over time that this dimension has an effect by measuring multiple time-points. Neuroticism was also shown to be a more significant and consistent predictor of PTSD and depression than several other variables, such as stressful pre-deployment life experiences, perceived threat from those experiences, and social support.

Other studies have shown a similar result, with neuroticism predicting comorbidity between PTSD and other disorders, mostly depression, four years after baseline assessment in a community sample of over 2400 individuals (Spinhoven, Pennix, van Hemert, Rooij, & Elzinga, 2014). In a study of over 1500 college students, pre-trauma neuroticism predicted PTSD severity two months later (Frazier et al., 2011). Similarly, pre-earthquake neuroticism predicted PTSD up to three years later in a sample of 307 citizens (Kuijer, Marshall, & Bishop, 2014).

A study of over 2000 adults in a community based survey found that pre-trauma neuroticism was predictive of PTSD over eight years later (Parslow, Form, & Christensen, 2005). In a similar longitudinal study of over 1000 civilians, baseline neuroticism predicted PTSD ten years later for those who experienced a trauma in between the two time points (Breslau & Schultz, 2013). This relationship was partly influenced by the presence of pre-existing depression; for individuals who had major depression at baseline, neuroticism did not increase the risk of later PTSD. However, the risk for PTSD associated with neuroticism was increased for those without baseline depression. The authors explain this finding by explaining that there was a ceiling effect of neuroticism scores in those with depression. The accumulating evidence of the longitudinal effects of neuroticism and depression makes a compelling case for it as a causal underlying dimension. However, it will be necessary for future research to isolate depression from neuroticism to investigate the strength of each variable in terms of its contribution to later psychopathology.

There is research linking neuroticism with other underlying dimensions of PTSD and depression, in addition to relationships with both disorders. In a study combining several datasets with mixed veteran and civilian samples, neuroticism was shown to have a significantly stronger correlation with dysphoria than with three other PTSD symptom scales, and is highly correlated with depression (Watson, Gamez, & Sims, 2005). The investigators also found that neuroticism was most strongly related to disorders characterized by pervasive distress, compared to specific phobias, panic, and social anxiety, and both PTSD and depression can be considered to be distress disorders. Similarly, neuroticism has been found to be generally significantly correlated with

symptoms of posttraumatic stress disorder, depression, and general distress in a sample of 86 individuals who had experienced either a natural disaster or sexual assault (Borja, Callahan, & Rambo, 2009). Interestingly, neuroticism in this study not only had an independent effect on psychopathology, but also influenced the degree to which individuals' symptoms were affected by social support. This finding is a reminder that underlying dimensions do not act in isolation, but have an impact on other psychological and environmental variables that in turn impact psychopathology. It is important for future research to examine multiple variables in combination to understand this larger context of how underlying dimensions interact. Neuroticism and behavioral inhibition have been shown to predict depression over time: specifically, that higher levels of these traits predict less symptom reduction over a one year period (Naragon-Gainey, Gallagher, & Brown, 2013).

Neurological background. Neurological abnormalities linked to neuroticism include atypical functioning and structure in the hippocampus, parahippocampus, prefrontal cortex, anterior cingulate cortex, temporal gyrus, insula, and amygdala (Canli et al., 2001; Chan, Norbury, Goodwin, & Harmer, 2008; Coen et al., 2011; Haas, Constable, & Canli, 2008; Haas, Omura, Constable, & Canli, 2007; Hooker, Verosky, Miyakawa, Knight, & D'Esposito, 2008). The regions pertinent to neuroticism have been also implicated in both depression and PTSD (Green and Ostrander, 2009; Haas, Constable, & Canli, 2008; Haas, Omura, Constable, & Canli, 2007). These regions are responsible for much of the phenomenology present in these disorders, such as the modification of emotional memories, perception of danger, negative emotionality,

suppression of the fear response or other negative emotionality, and executive functioning.

Neuroticism has been shown to have a strong genetic component in a study of nearly 15,000 Finnish twins (Viken, Rose, Kaprio, & Koskenvuo, 1994). The researchers also found genetic support for neuroticism across ages and age cohorts, though environmental factors become more influential as individuals get older. This study shows that the biological backing of underlying dimensions is robust over time.

Cultural influences. Levels of neuroticism have been shown to be higher in Egyptians than Americans and British participants (Ibrahim, 1979). The authors hypothesize that social constraints imposed by Egyptian society may be responsible for neuroticism, and related constructs of anxiety and emotional lability. Although this study is older, and interpretations of Egyptian culture may not still be accurate, the implications of societal pressures on negative emotionality is still relevant. Neuroticism also influences the expression of emotion regulation (Matsumoto, 2016), illustrating that the underlying dimensions discussed in this paper interact.

Behavioral Inhibition

Relationship with PTSD and depression. Behavioral inhibition can be considered as an attempt to resolve conflict between approaching reward and avoiding punishment or threat (Contractor, Elhai, Ractliffe, & Forbes, 2013). It is intuitive that behavioral inhibition would relate to PTSD; PTSD is heavily characterized by avoidance of perceived threats in terms of trauma reminders, which unfortunately, maintains the disorder. As evidence, PTSD related more to behavioral inhibition than behavioral activation (Contractor, Elhai, Ractliffe, & Forbes, 2013). Childhood behavioral inhibition

has been shown to be a risk factor for adult depression (Gladstone & Parker, 2006). Behavioral avoidance develops as an attempt to manage negative affect in both PTSD and depression, as well as other mood and anxiety disorders (Ellard, Fairholme, Boisseau, Farchione, & Barlow, 2010). In a study consisting of hundreds of people with either depression or PTSD, behavioral inhibition was linked with neuroticism (Brown, 2007; Campbell-Sills, Liverant, & Brown, 2004). As previously discussed, there is strong evidence to propose neuroticism as an underlying dimension of these disorders. Therefore, the strong relationship between neuroticism and behavioral inhibition increases the probability that behavioral inhibition is also a viable underlying dimension.

Behavioral Activation and Therapeutic Exposure (BA-TE) is a new treatment counteracting behavioral inhibition. In BA-TE, individuals first engage in behavioral activation, which reduces situational avoidance and increases the likelihood of environmental reinforcement by scheduling value- and positivity-based activities. Behavioral Activation has long been used for depressive symptoms. Then, individuals engage in imaginal exposures that target more PTSD specific symptoms, particularly re-experiencing. BA-TE has been shown to be effective in improving PTSD, overlapping symptoms of PTSD and depression, but not non-overlapping symptoms of depression (Gros et al., 2012). This treatment finding illustrates that behavioral inhibition may underlie the overlap between PTSD and depression, particularly compared to depression that is not related to PTSD.

Neurological background. The behavioral inhibition and behavioral activation systems discussed by Contractor, Elhai, Ractliffe, and Forbes (2013) are theorized to be linked to neurological systems surrounding the fear and distress experience. Thus, the

previously discussed neurological components linked with the stress response, reward response, and emotional processing are also implicated in behavioral inhibition. For example, the right-frontal areas of the cortex, previously reviewed as important for modulating emotion, have been associated with behavioral inhibition (Campbell-Sills, Liverant, & Brown, 2004). Behavioral inhibition has also been linked with the anterior cingulate cortex (Migliorini et al., 2015), which, as previously discussed, is a brain region linked with neuroticism and reward response as well. However, there is a gap in the literature specifically examining the relationship between the neurological behavioral inhibition system and comorbid PTSD and depression.

Behavioral inhibition has also been shown to have genetic influences. Specifically, behavioral inhibition is related to an allele of the corticotropin releasing hormone locus, which mediates the stress response (Smoller et al., 2003). Additionally, genetic variability in the dopamine receptor D2 has been linked with behavioral inhibition (Hamidovic, Dlugos, Skol, Palmer, & de Wit, 2009). The met allele of Brain Derived-Neurotrophic Factor (BDNF) also has been shown to have a relationship with higher scores on a scale of behavioral inhibition (Johnson, Carver, Joormann, & Cuccaro, 2016).

Cultural influences. Eastern individuals have been shown to have more behavioral inhibition in general than Western individuals, starting from a very young age (Rubin et al., 2006). The authors of this study, conducted with toddlers from China, South Korea, Italy, Australia, and Canada, conclude that although behavioral inhibition is a universal phenomenon, culture imparts meaning on this behavior, and thus defines the adaptiveness of this dimension.

Limitations of the Current Literature

Overlap in Dimensions and Symptomotology

One purpose of studying underlying dimensions of comorbidity is to solve the issue of the amount of overlapping symptomology. However, underlying dimensions that have been discussed in the literature themselves seem to overlap. For example, anhedonia appears to be a behavioral component of restricted positive affect. Negative affect and positive affect are distinct spectrums, but clearly related. Neuroticism and negative affect are occasionally used interchangeably in the literature. Researchers are labeling these experiences as underlying dimensions, but if there is considerable overlap, it is possible that these constructs can be reduced even further into their own underlying dimensions. Additionally, dimensions overlap with the symptom criteria for PTSD and Depression. Most clearly, negative affect is partially subsumed in the requirements for a diagnosis of depression. While the overlap can be problematic in terms of conceptualizing the relationship between dimensions and diagnoses, significant overlap may also be an indication that underlying dimensions can be substituted for diagnostic categories in terms of explaining and defining clinical pathology.

Lack of Operationalization

Researchers currently use varying terms for constructs which seem to be interchangeable, or at least highly similar. For example, it is not immediately apparent how and to what extent negative affect differs from general distress, and neuroticism. This is additively problematic when attempting to understand neurological underpinnings of these dimensions, as it is unclear if neurological evidence supporting one relationship

will apply to the other linked constructs. It is possible that researchers are using different words for what may be the same experience, psychologically and/or neurologically.

Minimal Theoretical Backing

There are theoretical complications in studying underlying dimensions. Currently, there does not appear to be any particular theory unifying these variables or illustrating how they tie together to form a cohesive story of how the variables contribute to PTSD and depression. However, there are theories that may help explain part of the interactions of underlying dimensions. For example, the tripartite model of depression and anxiety groups symptoms of depression and anxiety into three categories: nonspecific distress, somatic tension and arousal, and anhedonia/low positive affect (Clark & Watson, 1991; Watson et al., 1995). Though PTSD is no longer an anxiety disorder, much of its symptomology overlaps with the anxious symptomology Clark & Watson (1991) referred to. Additionally the tripartite model identifies some of the underlying dimensions previously discussed.

Related, the triple vulnerability model identifies dimensions of emotional disorders under three categories of vulnerability: a general biological vulnerability, a general psychological vulnerability, and a more disorder-specific psychological vulnerability (Barlow, 2000). These vulnerability factors mirror the neurological abnormalities and common psychological dimensions discussed in this paper. Additionally, it acknowledges that there are elements unique to each psychopathology, helping to explain the distinctions between PTSD and depression.

Although there may be advantages to a theory that unified these dimensions, there is an alternative perspective that following a theory can potentially be limiting. The

RDoC is a new system of research, and attempting to apply a unifying theory may narrow the scope of analysis too soon. Additionally, The RDoC is intentionally atheoretical so that there may be a focus on mechanisms. It may prove more useful to explore proposed dimensions first, and develop theory later.

Exclusion of Cultural Frameworks

Research regarding underlying dimensions has historically frequently neglected to consider cultural variables. Underlying dimensions are assumed to be universal, and the impact of culture is either minimized or completely ignored (Kirmayer & Crafa, 2014). These authors also state, "It is possible that people vary individually and culturally in ways that not only change some parameters within a given [neural] circuit but that actually alter the functions of that circuit in relation to the larger organization of behavior." Thus, all of the psychological dimensions that have been discussed and their neural underpinnings have the potential to vary as a function of culture. Future research can include cultural variables to verify which of these dimensions are indeed universal; for example, culture has not been examined in relation to dysphoria or distress. For the variables that are not universal, understanding the interactions between culture, biology, and these psychological dimensions can result in an even stronger framework and aligns with the personalized medicine approach.

Research that uncovers cultural variations in underlying dimensions generates questions regarding how universal these psychological variables are in terms of the ways in which they underlie psychopathology and in the ways in which they impact functioning. For dimensions that may differ between cultures, it is unknown whether those differences put individuals from a culture at more or less risk for psychopathology.

The differences in underlying dimensions across cultures do not necessarily suggest that these variables do not relate to PTSD and depression in a culturally generalizable manner. Rather, they point to the importance of including culture as a variable in research to obtain a more nuanced understanding of underlying dimensions amplify the robustness of transdiagnostic variables. Thus, the current study will collect demographic variables related to culture in order to explore the possible impact of culture on these relationships.

Cross Sectional Studies

Researchers who have previously labeled variables as underlying dimensions have rarely studied these traits longitudinally to know if they are casually connected. Related, researchers often label shared variables as underlying dimensions without providing a clear pathway in terms of etiology. In other words, it is not apparent how any of these characteristics in isolation lead to the complex phenomenologies that are PTSD and depression. It is however, plausible that these dimensions in combination are causally connected to later psychopathology, and some variables do demonstrate this in longitudinal studies (e.g. emotion dysregulation, neuroticism, behavioral inhibition). As the current study is also cross sectional, it will be important to note the limitations in inferring causal relationships from the results.

Lack of Comprehensive and Comparative Research

There are several proposed underlying dimensions, and there has not been investigation regarding which one(s) may be more impactful in the development of psychopathology. Further, it is unlikely that a single underlying dimension is enough to explain an entire disorder. There are probably multiple underlying dimensions for each disorder. There may be some that are unique to PTSD or to MDD, some that may be

shared by PTSD and MDD but not other disorders, or some that may overlap with other disorders. These concepts are illustrated by developmental psychopathology principles of equifinality (i.e., there are many possible pathways toward a disorder) and multifinality (i.e., the same etiological factors result in a variety of disorders) (Cicchetti & Rogosch, 1996; Luyten, Vliegen, Boudewijn, Houdenhove, & Blatt, 2008). It would be beneficial for future studies to examine these variables in combination and through comparative methods. Studying multifinality through underlying dimensions can elucidate risk factors for PTSD and depression, as well as other psychological disorders. Improved understanding of these risk factors can inform treatment or potentially prevent the development of psychopathology for individuals who exhibit those vulnerability factors.

As discussed, a number of underlying dimensions have been hypothesized to overlap with PTSD and major depression. Specifically, the following psychological variables will be examined as they relate to PTSD and major depression: negative affect, rumination, behavioral inhibition, neuroticism, and emotion dysregulation. Although previous studies have demonstrated a relationship between these dimensions and PTSD and depression, there has not been investigation regarding which dimensions(s) may be more salient in the development of psychopathology. It is unlikely that a single underlying dimension explains the development of either of these disorders entirely. However, examining these variables in combination through comparative methods may elucidate which dimension serves as the greatest risk factor for PTSD and comorbid depression. Understanding the comparative risk of these psychological variables can inform the focus of transdiagnostic interventions. Thus, the primary aim of the current

study is to investigate which underlying dimension emerges as a more significant predictor of PTSD and comorbid depressive symptoms.

Despite the complications with studying underlying dimensions, there is substantial evidence of their existence, both through their continued emergence in the literature, their established relationships with PTSD and depression as well as other disorders, and biological underpinnings. The aforementioned studies have been conducted with a variety of populations: men and women, citizens and military personnel, in the United States and internationally, at sites ranging from schools to community surveys to health clinics, with a number of trauma types. Thus, the combination of these studies is quite generalizable and represents a large portion of the individuals with PTSD and depression. The previously referenced underlying dimensions are likely contributors to comorbid PTSD and depression. Thus, investigating these variables further, more comprehensively, and with more clarity could be extremely useful in enhancing our understanding of these psychopathologies.

Psychological disorders are complex in and of themselves, and understanding two comorbid disorders and the relationship between them is even more complicated. Given the high comorbidity of PTSD and depression, and the deleterious effects of that comorbidity, it is important to attempt to elucidate this complexity. As stated previously, comorbid depression in the context of PTSD is associated with more severe symptomatology, impaired functioning in a variety of domains, difficulties in health care utilization, and worse outcomes in therapy. The proposed underlying dimensions may help elucidate the psychological and biological underpinnings of these consequences.

Identifying those underpinnings can shape a new way of conceptualizing these disorders beyond their symptom classification.

Current Study Aims

Because of the high rate of comorbid depression in PTSD and the deleterious effects of these two co-occuring conditions, the current study aims to increase our understanding regarding comorbid depression within the context of PTSD**.** In particular, examining potential underlying dimensional constructs these two disorders may share is an important and newly studied area of research. As such, this project focuses on investigating these psychological dimensions of PTSD and depression with the goal of better understanding this complex comorbid relationship.

A number of underlying dimensions have been hypothesized to overlap with PTSD and major depression. Specifically, the following psychological variables will be examined as they relate to PTSD and major depression: negative affect, rumination, behavioral inhibition, neuroticism, and emotion dysregulation. Although previous studies have demonstrated a relationship between these dimensions and PTSD and depression, there has been a paucity of research regarding which dimensions(s) may be more salient in the development of psychopathology. These underlying dimensions have previously been studied in isolation. Examining these variables in combination through comparative methods may elucidate which dimension serves as the greatest risk factor for PTSD and comorbid depression. Understanding the comparative risk of these psychological variables can inform the focus of clinical interventions. Thus, the primary aim of the current study is to investigate which underlying dimension emerges as a more significant predictor of PTSD and comorbid depressive symptoms.

Previous research has illustrated that interpersonal trauma is associated with higher rates of psychopathology and distress, including more severe PTSD symptoms. Thus, the current study aims to establish interpersonal trauma as more predictive of psychopathology than non-interpersonal trauma. Further, if underlying dimensions are to be understood as precursors to PTSD and depression, then by extension, interpersonal trauma may be more predictive of those dimensions. Therefore, the study also aims to establish the relationship between interpersonal trauma and negative affect, rumination, behavioral inhibition, neuroticism, and emotion dysregulation.

Hypotheses:

Hypothesis 1: PTSD and depression scores will be significantly and positive correlated with one another, such that higher PTSD scores are related to higher depression scores.

Hypothesis 2: PTSD and depression severity will be predicted by negative affect, rumination, behavioral inhibition, neuroticism, and emotion dysregulation. These relationships will also be positively linked; for example, individuals with higher rumination scores will have higher PTSD and depression scores.

Hypothesis 3: Neuroticism and emotion dysregulation will have the strongest relationships with PTSD and depression. In other words, the coefficient representing the association between Neuroticism and PTSD will be significantly higher than the coefficient representing the association between behavioral inhibition and PTSD, for example.

Hypothesis 4: The underlying dimensions will have stronger relationships with comorbid PTSD and depression than with either disorder alone, as well as compared to individuals with no clinically significant psychopathology.

Hypothesis 5: Individuals who identify an interpersonal trauma as their index trauma will have higher PTSD and depression scores, compared to those who identify a non-interpersonal trauma as their index event.

Hypothesis 6: The relationship between the underlying dimensions and PTSD and depression will be more pronounced in individuals who have experienced interpersonal trauma, compared to those who identify a non-interpersonal trauma as their index event.

Exploratory analyses will examine the relationships between gender, ethnicity, and age of traumatic exposure and PTSD and depression. Given the minimal literature examining these variables as they relate to underlying dimensions and PTSD or depression, there are no specific hypotheses regarding these variables.

Method

Participants

Participants included adults, and there were no exclusion criteria. In order to maximize the number of participants in each diagnostic category, 942 adult participants were recruited. The questionnaire battery was expected to take 30 minutes to complete; participants who completed the survey in under 10 minutes (N = 206) were deemed to be not adequately attending to survey content, and were thus excluded. Of this total sample, 19 were excluded due to a significant portion of missing data (i.e. answered less than 80% of items). Therefore, the final sample for analysis consisted of 717 individuals, ranging in ages from 18-72 (M = 30, SD = 12). 32% were male, 67% were female, and

1% identified otherwise (e.g., Transgender Male, Transgender Female, or Genderqueer/ Nonbinary). The sample was ethnically diverse, with 4% Asian, 4% Hispanic, 4% Multiracial, 19% African- American, and 66% Caucasian individuals. A small percentage of individuals identified as American Indian and Middle Eastern. The majority of individuals (55%) completed some college; 10% had a high school education, 23% completed college, 5% had some post-undergraduate education, and 7% completed graduate school. Table 1 in the appendix illustrates characteristics of these participants.

Participants were asked to identify their index trauma, defined subjectively as the trauma that impacted them the most. Based on the index trauma they endorsed, participants were organized into interpersonal index trauma or non-interpersonal index trauma groups. Interpersonal traumas included physical assault, assault with a weapon, sexual assault, and unwanted or uncomfortable sexual experience. Non-interpersonal traumas included natural disaster, fire or explosion, transportation accident, serious accident at work or home, exposure to toxic substances, and life threatening illness or injury.

Participants were recruited from the University of Missouri-St. Louis subject pool using the SONA system, Amazon's Mechanical Turk database, as well as from the community through internet advertisements. As an incentive for participation, the UMSL students received research credit, Mechanical Turk participants were compensated for completing the survey, and community participants entered their contact information into a raffle to win an Amazon gift card. Participants' contact information were collected separately from survey responses to ensure confidentiality.

Measures

Demographics. Participants completed a questionnaire requesting information about their sex, age, ethnicity, and years of education.

Life Events Checklist. This questionnaire consists of 17 items that assesses the types of traumatic events an individual has experienced. Items include events such as: "natural disaster", "transportation accident", "physical assault", and "sexual assault." Participants checked a box if a particular trauma happened to them, and they checked a separate box if they witnessed the trauma. Afterwards, participants who endorsed multiple traumas were asked to mark which event was most impactful for them.

Posttraumatic Stress Disorder Checklist (PCL). The PCL consists of 17 items that assess the PTSD symptoms an individual has experienced in the past month. Participants were reminded to consider these symptoms in the context of the trauma they marked as most impactful. Participants rated how much they have been bothered by particular symptoms on a Likert scale from 1 (not at all) to 5 (extremely). Items include symptoms such as "repeated, disturbing memories, thoughts, or images of a stressful experience in the past", "avoiding thinking about or talking about a stressful experience from the past or avoiding having feelings related to it", "feeling distant or cut off from other people", and "feeling jumpy or easily startled." Cronbach's alpha in this sample was 0.95. Participants were coded diagnostically as having PTSD if their score was equal to or greater than 33, which is the standard clinical cutoff for this measure.

Beck Depression Inventory (BDI-II). The BDI-II consists of 21 items that assess the depressive symptoms an individual has experienced in the past two weeks. Participants rated how much they have been bothered by particular symptoms on a Likert scale from 0-3, with higher ratings indicating more severe symptoms. Items include

symptoms such as sadness, guilt, anhedonia, fatigue, and sleeping difficulties. Cronbach's alpha in this sample was 0.94. Participants were coded diagnostically as having depression if their score was equal to or greater than 20 (indicative of moderate depressive levels).

Big Five Inventory. The Big Five Inventory consists of 44 items that assess the main personality traits: conscientiousness, agreeableness, extraversion, openness, and neuroticism. The measure begins with a prompt, "I see myself as someone who…" Participants rated how much particular tendencies and behaviors apply to them on a Likert scale from 1 (disagree strongly) to 5 (agree strongly). Sample items related to neuroticism include: (I see myself as someone who) "can be tense" and "worries a lot." Cronbach's alpha in this sample was 0.79.

Difficulties in Emotion Regulation Scale (DERS). The DERS consists of 36 items that assess multiple facets of emotion regulation, including emotional awareness, impulsivity, acceptance of emotions, and strategies for coping with negative emotions. Participants rated how often particular emotional experiences apply to them on a Likert scale from 1 (almost never) to 5 (almost always). Sample items include "I have difficulties making sense out of my feelings", "When I'm upset, I start to feel very bad about myself" and "When I'm upset, I become out of control." Cronbach's alpha in this sample was 0.94.

Positive And Negative Affect Schedule (PANAS). The PANAS consists of 60 items that assess positive and negative emotional experiences. Participants rated the extent to which they have felt those emotions over the past several weeks on a Likert

scale ranging from 1 (not at all/very slightly) to 5 (extremely). Cronbach's alpha in this study was 0.96.

Ruminative Thoughts Style Questionnaire (RTSQ). The RTSQ consists of 20 items that examine ruminative tendencies. Participants rated how well statements regarding rumination describe them on a Likert scale ranging from 1 (not at all) to 7 (very well). Sample items include "I find that my mind often goes over things again and again", "When I am anticipating an interaction, I will imagine every possible scenario and conversation", and "When I am worrying about something, thoughts of it interfere with what I am working on." Cronbach's alpha in this sample was 0.96.

Behavioral Inhibition System/ Behavioral Activation System Scale (BIS/BAS). The BIS/BAS scale consists of 24 items that examine tendencies toward behavioral inhibition and behavioral activation. Participants rated how true an item is for them on a Likert scale ranging from 1 (very true for me) to 4 (very false for me). Sample items include "I'm always willing to try something new if I think it will be fun" and "I worry about making mistakes." Cronbach's alpha in this sample was 0.79.

Data analytic plan

Regarding Hypothesis 1, a correlation test was used to assess the relationship between PTSD and depression scores. Regarding Hypothesis 2, a regression analysis was conducted to establish the relationships between negative affect, rumination, neuroticism, emotion dysregulation, PTSD, and depression. Previous studies have used single linear regression analyses to illustrate the relationships between psychological constructs and pathology; however, these studies have typically lacked more sophisticated statistical models of investigating multiple dimensions in combination. Therefore, regarding

Hypothesis 3, a multiple regression analysis was conducted to establish neuroticism and emotion dysregulation as predicting more of the variance in PTSD and depression scores, compared to the other underlying dimensions. In this multiple regression, all underlying dimensions were entered into the model as potential predictor variables.

With similar aims of modeling the complexity of underlying dimensions in their relationship to pathology, Regarding Hypothesis 4, a multinomial logistic regression was conducted to examine the relationships between underlying dimensions and comorbid PTSD and depression. To conduct this analysis, participants were divided into diagnostic groups based on recommended clinical cutoff scores on the depression and PTSD measures; the groups consisted of individuals with neither PTSD nor depression, significant PTSD symptoms but not depression symptoms, significant depression but not PTSD, and both PTSD and depression symptoms that excess the designated cutoff scores. Previous studies have used logistic regression to analyze predictors of PTSD and depression comorbidity, though not with the currently discussed underlying dimensions (Farhood, Fares, Sabbagh, and Hamady, 2016; Horesh et al., 2017). Doing both of these types of regression analyses (multiple regression and logistic regression) illustrated how these underlying dimensions relate to PTSD and depression as continuous constructs and categorical constructs in their respective diagnostic labels.

Regarding Hypothesis 5, first the sample was narrowed to individuals who endorse an exposure to trauma. Then, a T-test was used to compare the severity psychopathology between the two groups (interpersonal index trauma and non-interpersonal index trauma). Regarding Hypothesis 6, after the sample is divided into the two groups (interpersonal index trauma and non-interpersonal index trauma), regression

analyses were conducted between each underlying dimension and PTSD and depression scores. Regression coefficients were standardized and transformed into a Z-score: these z-scores were then compared to see relative predictability of underlying dimensions in regards to symptom severity, as it relates to trauma type. The underlying dimensions used in this analysis were based on the results of Hypothesis 3; in other words, whichever underlying dimensions emerge as significant predictors of PTSD and depression were then analyzed in the interpersonal and non-interpersonal groups.

As previously stated, some underlying dimensions are embedded into aspects of diagnostic criteria, and the dimensions themselves may overlap in their definitions. This occurrence raises potential issues of multicollinearity; if the measurement of PTSD and depression overlaps with the measurement of underlying dimensions, or if the measurement of one underlying dimension overlaps with another, it is possible that these constructs will be very highly related. These relationships are problematic, as it becomes difficult to interpret the unique contribution of each dimension to PTSD and depression. In preparation for this study; a brief literature search was conducted searching the terms "multicollinearity" and "underlying dimension" as well as "multicollinearity" and "research domain criteria"; no articles emerged that have addressed this issue conceptually or methodologically. On the contrary, several of previously discussed studies have used symptom criteria in their construction of underlying dimensions, such as in the case of latent profile analysis. It should be noted that multicollinearity is typically considered an issue when two variables are correlated at 0.75 or higher (Meyers, Gamst, & Guarino, 2013); in the previously cited studies that reported correlation or

regression coefficients, no underlying dimension was related to measures of depression or PTSD at that high of a degree (correlations ranged from approximately 0.40 to 0.60).

Multicollinearity was tested by investigating correlation coefficients between all variables. No variables were correlated at 0.75 or higher; thus, it can be assumed that predictor variables in the study do not overlap in their constructs to a problematic level, and regression analyses can proceed. Multicollinearity was also tested within the multiple regression analysis. Variance inflation factors ranged from 1.70-2.36 for the underlying dimensions; these values are within an acceptable range, according to Meyers, Gamst, & Guarino (2013).

Results

Participants had an average PCL score of 37.87 (SD = 16.5), and an average BDI score of 16.41 (SD = 12.34). This indicates that, per self-report measures, the overall sample had clinically significant PTSD and mild depression symptoms. The elevated PTSD scores may be due to a self-selecting participant effect; because the study was advertised with the title “Stress and Depression,” it is possible that some participants were interested in taking this study due to their relationship with the expected content.

Additionally, there was a relationship between age and PTSD and depression scores. Age exhibited a small, but significant inverse correlation with the PCL ($r = -.078$, $p < .05$), and the BDI ($r = -.082$, $p < .05$). Thus, as age increased, scores reflecting psychopathology decreased.

Participants had an average PANAS negative affect subscale score of 21.86 (SD = 9.36), an average RTSQ score of 25.01 (SD = 7.24), an average BFI neuroticism subscale score of 25.02 (SD = 7.24), an average DERS score of 89.48 (SD = 26.09), and an

average BIS score of 20.87 (SD = 4.48). Normative PANAS negative affect subscale scores range between 14.2 and 19.5; thus, this study has slightly higher negative affect than a normative sample. Normative BFI neuroticism scores are 25.36 for individuals age 30 (the average age of this sample), and are thus comparable to this study. The sample the DERS was normed on had an average score of 78 for women and 80.66 for men (Gratz & Roemer, 2004). Thus, this sample had a slightly higher average score. See Table 1 for an overview of participant clinical characteristics and demographic information.

The first hypothesis, that PTSD and depression scores will be significantly and positively correlated with one another, was supported ($R = .662$). Consistent with previous literature, this correlation test confirms that higher PTSD scores are related to higher depression scores.

The second hypothesis was also supported; PTSD and depression severity were predicted by the various underlying dimensions in a linear regression analysis. Depression was predicted by negative affect ($R = .672$, $P < .001$), rumination ($R = .543$, $p < .001$), behavioral inhibition ($R = .296$, $p < .001$), neuroticism ($R = .619$, $P < .001$), and emotion dysregulation ($R = .664$, $P < .001$). PTSD was also predicted by negative affect ($R = .653$, $p < .001$), rumination ($R = .437$, $p < .001$), behavioral inhibition ($R = .156$, $p < .001$), neuroticism ($R = .425$, $p < .001$), and emotion dysregulation ($R = .549$, $p < .001$). Thus, individuals with higher PTSD and depression scores also have higher scores on all underlying dimensions. See Tables 2 and 3 for summaries.

Hypothesis 3 was mostly unsupported. A multiple regression was conducted to examine the relative power of these underlying dimensions in predicting PTSD and depression. The depression model, with all predicting variables combined, was

significant (R^2 = .592, p <.001). Within this model, negative affect was the most predictive of depression (Standardized B = .332, p <.001), followed by neuroticism (B = .274, p < .001), emotion dysregulation (B = .240, p <.001), and finally rumination (B = .074, p = .034). Behavioral inhibition, while predictive within a linear regression, no longer predicted depression in this full model. The PTSD model, with all predicting variables combined, was significant (R^2 = .686, p <.001). Within this model, negative affect was again the most predictive of PTSD (B = .486, p <.001), followed by emotion dysregulation (B = .154, p = .001), rumination (B = .132, p = .002), and behavioral inhibition (B = -.124, p = .002). Neuroticism, while predictive within a linear regression, no longer predicted PTSD in this full model. See Tables 4 and 5 for a summary.

It was anticipated that within a multiple regression, neuroticism and emotion dysregulation would have the strongest relationships with PTSD and depression. However, emotion dysregulation was still a significant predictor of PTSD and depression, but not quite to the degree of negative affect, and neuroticism did not predict PTSD with all other dimensions accounted for in the model. Notably, each model had numerous significant predictors, indicating that these underlying dimensions each contribute a unique variance to PTSD and/or depression.

To analyze Hypothesis 4, participants were separated into diagnostic categories based on their PCL and BDI score. There were 307 participants with no diagnostic assignment (e.g. minimal to no symptoms of PTSD or depression), 80 participants with significant depression only (BDI equal to or greater than 20; Beck, 1996), 150 participants with significant PTSD only (PCL equal to or greater than 33; Weathers et al., 2013), and 177 participants with comorbid PTSD and depression. A multinomial logistic regression was

then conducted, dummy coding for diagnostic categories, with the comorbid category as a reference group. The multinomial logistic regression model fit indices show a -2 log likelihood value of 1330.97, which is statistically significant ($p < .001$). Therefore, overall, diagnostic category can be predicted at a better than chance level using the underlying dimensions as predictors. Nagelkerke's Pseudo R^2 is .504, indicating that the underlying dimensions in the model account for 50% of the variance associated with diagnostic category. In the overall model, several dimensions emerged as significant predictors, including negative affect ($p < .001$), neuroticism ($p < .001$), emotion dysregulation ($p < .001$), and rumination ($p = .01$).

Comorbid vs. PTSD only: Participants with PTSD only differed significantly from those with PTSD and Depression comorbidity with regard to neuroticism (Odds Ratio = .88, 95% Confidence Interval: .83-.94) and emotion dysregulation (OR = .96, 95% CI: .95-.98). This indicates that an increase of 1 on the neuroticism subscale of the BFI increases the odds of having comorbidity, as opposed to just PTSD, by 1.14 times (1/.88). Similarly, an increase of 1 on the DERS increases the odds of having comorbidity as opposed to only PTSD by 1.04 times (1/.96).

Comorbid vs. Depression only: Participants with Depression only differed significantly from those with PTSD and Depression comorbidity with regard to rumination (OR = 1.023, 95% CI: 1.006-1.040). Therefore, an increase of 1 on the RTSQ increases the odds of having depression only as opposed to comorbidity by 1.02 times.

Comorbid vs. No diagnosis: Participants with no diagnosis differed significantly from those with PTSD and Depression comorbidity with regard to negative affect (OR = .884, 95% CI: .849-.919), neuroticism (OR = .825, 95% CI: .776-.877), and emotion

dysregulation (OR = .977, 95% CI: .963-.991). Therefore, an increase of 1 on subscales representing negative affect, neuroticism, and emotion dysregulation increases the odds of having comorbidity, as opposed to no diagnosis, by 1.14, 1.20, and 1.02 times, respectively.

Similar to Hypothesis 3, there is mixed support for Hypothesis 4. Neuroticism and emotion dysregulation illustrated a stronger relationship with comorbidity than with PTSD only. Additionally, neuroticism, emotion dysregulation, and negative affect illustrated a stronger relationship with comorbidity than with no clinically significant psychopathology. However, no underlying dimension in this analysis showed a stronger relationship with comorbidity than with depression; to the contrary, rumination showed a stronger relationship with depression, compared to comorbidity. See Table 6 for a summary of these following results.

Regarding Hypothesis 5, PCL and BDI scores were compared between individuals who identified an interpersonal index trauma (N = 191) versus noninterpersonal (N = 209). Other individuals in the full sample either did not endorse a trauma or endorsed a trauma that did not clearly fall under one of these two categories, such as combat trauma or severe human suffering. Hypothesis 5 was supported; individuals with interpersonal trauma had higher PTSD scores ($T = 4.66$, $p < .001$; interpersonal M = 42.31, non-interpersonal M = 33.28) and depression scores ($T = 5.60$, $p < .001$; interpersonal M = 18.77, non-interpersonal M = 13.33), compared to those with non-interpersonal trauma.

Hypothesis 6 was aimed at further investigating the differences between interpersonal and non-interpersonal trauma. Because there was some variability in the results of Hypothesis 3, regarding the relationships between each underlying dimension and PTSD

and depression, all underlying dimensions were again investigated in regards to trauma type. For those with non-interpersonal trauma, depression was significantly predicted by negative affect ($R = .65$, $p < .001$), rumination ($R = .54$, $p < .001$), neuroticism ($R = .62$, $p < .001$), emotion dysregulation ($R = .61$, $p < .001$), and behavioral inhibition ($R = .42$, $p < .001$). PTSD was predicted by negative affect ($R = .64$, $p < .001$), rumination ($R = .33$, $p < .001$), neuroticism ($R = .30$, $p < .001$), and emotion dysregulation ($R = .52$, $p < .001$). Behavioral inhibition was not a significant predictor for this group. See Tables 7 and 8 for summaries of these regressions.

For those with interpersonal trauma, depression was significantly predicted by negative affect ($R = .70$, $p < .001$), rumination ($R = .52$, $p < .001$), neuroticism ($R = .66$, $p < .001$), emotion dysregulation ($R = .67$, $p < .001$), and behavioral inhibition ($R = .39$, $p < .001$). PTSD was predicted by negative affect ($R = .64$, $p < .001$), rumination ($R = .44$, $p < .001$), neuroticism ($R = .43$, $p < .001$), emotion dysregulation ($R = .55$, $p < .001$), and behavioral inhibition ($R = .20$, $p < .01$).

Fisher's r-to-z transformation was then used to compare regression coefficients between trauma types. Hypothesis 6 was then shown to be unsupported; the relationship between the underlying dimensions and PTSD and depression was not more pronounced in individuals who had experienced interpersonal trauma.

Secondary Analyses

Gender was investigated as a variable that could potentially differ in terms of psychopathology and underlying dimensions. There was a significant effect of gender on PTSD scores, in that females had significant higher PTSD scores than males ($T = 2.61$, $p < .01$; female $M = 39$ (16.3), male $M = 35.05$(16.58)). Females also had significantly

higher neuroticism scores ($T = 6.01$, $p < .001$; female $M = 26.12$ (7.05), male $M = 22.68$ (7.01)) and behavioral inhibition scores ($T = 7.20$, $p < .001$; female $M = 21.71$ (4.21), male $M = 19.19$ (4.56)).

Because only one ethnic minority was significantly represented in this sample, African Americans were compared to Caucasians regarding these variables, as well. African Americans had significantly higher negative affect ($T = 2.30$, $p < .05$), and Caucasians had significantly higher neuroticism ($T = 3.66$, $p < .001$) and behavioral inhibition ($T = 4.61$, $p < .001$).

Age of index trauma exposure was also examined as a potential predictor; however, there were no significant relationships between age of trauma and levels of psychopathology or any of the underlying dimensions.

Discussion

The results indicate several relationships among PTSD, depression, and various underlying dimensions. Consistent with previous literature, there was a strong correlation between PTSD and depression scores, highlighting the comorbidity of these disorders and illustrating the importance of understanding underlying dimensions. Higher scores on the underlying dimensions were each related to higher scores on both measures of psychopathology. These relationships also confirm previous literature suggesting that the psychological constructs studied (negative affect, neuroticism, emotion dysregulation, rumination, and behavioral inhibition) are all potential dimensions of comorbid PTSD and depression (Bradley et al., 2011; Contractor, Elhai, Ractliffe, & Forbes, 2013; Kubota, Nixon, & Chen, 2015; Post, Feeny, Zoellner, & Connell, 2015).

However, when all underlying dimensions were combined into one multiple regression model, negative affect, emotion dysregulation, and rumination were unique predictors of both PTSD and depression. Neuroticism was predictive of depression, but not PTSD when all other variables were taken into account. Behavioral inhibition was predictive of PTSD, but not depression when all other variables were taken into account. Therefore, while all the aforementioned dimensions may underlie these disorders, negative affect, emotion dysregulation, and rumination are most strongly associated with the specific comorbidity. In particular, negative affect showed the strongest relationship. Thus, PTSD and depression comorbidity may be best explained and targeted by shared general negative emotionality.

Similarly, in the multinomial logistic regression, not all dimensions were equally powerful in their relationship to comorbidity. Neuroticism and emotion dysregulation separated those with comorbidity from those with just PTSD, in that individuals with comorbidity were higher on these constructs (more neurotic, more emotionally dysregulated). Rumination separated those with comorbidity from those with just depression, in that individuals with depression were higher on this construct; this is a contrast from previous literature illustrating that comorbidity is generally associated with more pathology in their psychological processes. Rumination may therefore be more of a depressive process. As further support, Olatunji, Naragon-Gainey, and Wolitzky-Taylor (2013) found rumination to be linked more with depressive disorders than with anxiety disorders. Negative affect, neuroticism, and emotion dysregulation separated those with comorbidity from those with no significant psychopathology.

The multinomial logistic regression results also provide an important contrast to the multiple regression results. Although these statistical tests have similarities, dividing participants into categorical constructs significantly changed how the underlying dimensions related to psychopathology. This occurrence brings up important methodological questions for research in this area. If answering the same research question within the same sample in two different analyses results in different outcomes, interpretations of those results may need to be scrutinized more thoroughly. This illustrates the importance of replication in psychological research, to be more confident which dimensions are truly and highly related to comorbidity.

Again similar to previous research (e.g. Forbes et al., 2012), interpersonal trauma was associated with a higher degree of psychopathology. For individuals with interpersonal trauma, all underlying dimensions predicted both PTSD and depression scores. For individuals with non-interpersonal trauma, all underlying dimensions predicted depression and PTSD scores, with the exception that behavioral inhibition was not related to PTSD. This suggests that the relationship between behavioral inhibition and PTSD may be unique to those with interpersonal trauma. Perhaps these individuals, betrayed by others through abuse or assault, have a particular wariness of behavioral engagement, which often involved approaching others. This trauma type variation may be the reason why, compared to other dimensions, behavioral inhibition explained less variance in PTSD scores within the multiple regression model, as well as the reason why behavioral inhibition did not emerge as a significant predictor in multinomial logistic regression results. Finally on the point of trauma type, the relationship between the underlying dimensions and comorbidity was not more pronounced for those with

interpersonal trauma. This suggests that regardless of trauma type, underlying dimensions of PTSD and depression remain relatively constant. To this author's knowledge, no studies to date have examimed how trauma type may impact the relationship between underlying dimensions and psychopathology.

Similar to previous research (e.g. Resick, 2001), females had more pronounced PTSD scores than males in this study, potentially driven by increased neuroticism and behavioral inhibition. As just discussed, behavioral inhibition was linked with PTSD only in interpersonal trauma, and several types of interpersonal trauma, namely sexual abuse and assault, and more prevalent in females.

Because the field of underlying dimensions research is relatively new, there is a dearth of research regarding how constructs relate to different demographic variables, such as gender, and particularly when it comes to ethnicity in race. In this study, there were a few differences in the levels of negative affect, neuroticism, and behavioral inhibition endorsed by African-Americans versus Caucasians. These differences may or may not have consequences in terms of comorbidity. Importantly, these differences serve as a contrast to the atheortical position of the Research Domain Criteria; there would be no reason within this framework to assume that underlying dimensions could differ from person to person or culture to culture. Clearly, much is to be investigated in terms of the interaction between demographic, cultural, and psychological variables.

The results of this study have potential implications for clinical work for individuals who have experienced trauma and have PTSD, depression, or both. Although there was some analytic variability in how exactly these underlying dimensions relate to PTSD and depression, these constructs are each still promising avenues to understanding

that comorbidity. As such, transdiagnostic interventions such as the Unified Protocol for the Transdiagnostic Treatment of Emotional Disorders or Transdiagnostic Behavior Therapy may be of particular use to this population. Further, transdiagnostic treatments that target specific underlying dimensions, such as Emotion Regulation Therapy, may be of use. Moreover, new transdiagnostic interventions can be creased that incorporate more of these dimensions. Clinicians may also choose to add modules targeting a specific dimension within the evidence-based treatment they are using. Further research would be needed to determine how best to incorporate these modules into an evidence-based psychotherapy while still maintaining theoretical fidelity. For individuals not engage in an evidence-based treatment, clinicians may consider skills training aimed at reducing negative affect, neuroticism, and rumination, as well as increasing emotion regulation and behavioral activation.

Decisions on which underlying dimensions to focus on may be informed by psychopathology or comorbidity, gender of the patient, or trauma type. Additionally, while age of trauma was not a significant predictor in this study, earlier traumatic exposure has previously been linked with more severe PTSD and depression, and presumably would be related to these underlying dimensions. Thus, further research should continue to investigate this construct as it relates to underlying dimensions, which may inform treatment. In addition to incorporating dimensions into interventions, assessing a patient's level of neuroticism or emotion dysregulation may be an alternate way of tracking progress in therapy, in conjunction with typical PTSD and/or depression measures.

Finally, the results of this study and future underlying dimensions research may have eventual implications for our diagnostic system. If certain constructs are consistently found to underlie depression, PTSD, their comorbidity, and other psychological disorders, future classification systems may be comprised of how individuals fall on spectrums of psychological constructs, rather than discrete diagnostic categories.

Study Limitations

There were various limitations in this study. Firstly, while the initial sample was sizeable, a large fraction of data had to be excluded for various reasons. The final sample size was more than robust enough for the current analyses, and excluding participants was aimed at protecting the overall integrity of the data. Still, this may potentially speak to drawbacks of using data collected from an internet survey. As with all data collected online, there are certain potential risks regarding quality of data, as the experimenter cannot assess the participants firsthand. On the other hand, online participants may be more forthcoming with their symptoms and traumatic experience when they are not imagining an in-person experimenter analyzing them. The sample had some demographic variability, but was largely Caucasian and female. This may limit the generalizability of the results to a more diverse overall population. Additionally, the sample was non-clinical, and diagnoses were inferred based on self-reports. Therefore, the results may not be generalizable to individuals with diagnosable PTSD or depression. Additionally, this study focused on one particular comorbidity, and future research may benefit from examining PTSD and anxiety disorders or pathological eating, for example. Finally, theories of underlying dimensions reflect a causal, longitudinal relationship. This cannot

be achieved by the current study's cross-sectional methodology. Thus, similar to many previous studies on the topic, the results of this study have limitations in inferring causality.

Conclusion

Despite the limitations, there are various implications of these results. No known study to date has examined all of these underlying dimensions in combination, let alone attempting to examine the relative contribution of each dimension to comorbid PTSD and depression. Negative affect, neuroticism, emotion regulation, rumination, and behavioral inhibition do relate to PTSD and depression, and the results of this study provide some insight into the nature of that relationship. The relationship between dimensions and comorbidity appears to be more complex than is currently known, and different statistical methods may result in different outcomes. Still, when treating individuals for PTSD and depression, it may be beneficial to assess for how they relate to these underlying dimensions, as these constructs may be a target of treatment. Future research is needed to investigate the specific pathways in which underlying dimensions result in trauma-related psychopathology.

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Appendix

Table 1
Participant Characteristics (N = 717)

Age	M = 29.81 SD = 12.036 Range = 18-72

	Frequency	Percentage
Gender		
Male	229	31.9
Female	481	67.1
Other (genderqueer, transgender)	7	1
Ethnicity		
American Indian	6	0.8
Asian	29	4
Black/African American	133	18.5
Hispanic	31	4.3
Middle Eastern	12	1.7
Multiracial	25	3.5
Caucasian	474	66.1
Other	6	0.8
Level of Education		
Some high school	3	0.4
Completed high school	69	9.6
Some college	365	55.1
Completed college	168	23.4
Some graduate school	33	4.6
Completed graduate school	49	6.8
Yearly Household Income		
Below $15,000	116	16.2
15,000-24,999	104	14.5
25,000-39,999	115	16.0
40,000-54,999	102	14.2
55,000-69,999	95	13.2
70,000-84-999	60	8.4
85,000-99,999	38	5.3
100,000-149,999	56	7.8
$150,000+	29	4.0

Note. Missing data accounts for any percentages not summing to 100.

	M	SD	Range
PCL	37.87	16.50	17-85
BDI	16.41	12.34	0-60
PANAS_Negative Affect	21.86	9.36	10-50
RTSQ	90.78	27.5	20-140
BFI_Neuroticism	25.01	7.24	8-40
DERS	89.48	26.09	40-167
BIS	20.87	4.48	7-28

Table 2
Underlying dimensions as predictors of BDI scores: Linear regression

Dimension	R	R^2	F	p
PANAS-Negative Affect	.672	.451	583.212	.000
RTSQ	.542	.295	290.166	.000
BFI-Neuroticism	.619	.383	436.877	.000
DERS	.664	.441	549.424	.000
BIS	.296	.087	66.137	.000

Table 3
Underlying dimensions as predictors of PCL scores: Linear regression

Dimension	R	R^2	F	p
PANAS-Negative Affect	.653	.427	441.192	.000
RTSQ	.437	.191	135.846	.000
BFI-Neuroticism	.425	.181	129.470	.000
DERS	.549	.301	250.198	.000
BIS	.156	.024	14.351	.000

Table 4
Underlying dimensions as predictors of BDI scores: Multiple regression

Model Summary

	R	R^2	F	p
	.770	.592	196.867	.000

Dimension	Standardized Beta	t	p
PANAS-Negative Affect	.332	9.426	.000
RTSQ	.074	2.119	.034
BFI-Neuroticism	.274	7.282	.000
DERS	.240	6.355	.000
BIS	-.007	-0.226	.821

Table 5
Underlying dimensions as predictors of PCL scores: Multiple regression

Model Summary

	R	R^2	F	p
	.686	.470	99.968	.000

Dimension	Standardized Beta	t	p
PANAS-Negative Affect	.486	11.198	.000
RTSQ	.136	3.131	.002
BFI-Neuroticism	.057	1.172	.242
DERS	.154	3.359	.001
BIS	-.124	-3.113	.002

Table 6
Underlying dimensions as predictors of diagnosis: Multinomial logistic regression Comparisons to reference group (participants with comorbidity)

No Diagnosis

	B	SE	Wald	Exp(B)	95% C.I. for Exp(B) Lower	Upper
PANAS-Neg Affect	-.124	.020	37.637	.884***	.849	.919
RTSQ	-.003	.007	.204	.997	.983	1.011
BFI-Neuroticism	-.192	.031	38.696	.825***	.776	.877
DERS	-.024	.007	10.300	.977**	.963	.991
BIS	.034	.039	.786	1.035	.959	1.116

Depression only

	B	SE	Wald	Exp(B)	95% C.I. for Exp(B) Lower	Upper
PANAS-Neg Affect	.003	.019	.028	1.003	.967	1.041
RTSQ	.023	.009	6.853	1.023**	1.006	1.040
BFI-Neuroticism	-.055	.035	2.507	.947	.885	1.013
DERS	-.001	.008	.022	.999	.983	1.015
BIS	-.062	.040	2.372	.940	.869	1.017

PTSD only

	B	SE	Wald	Exp(B)	95% C.I. for Exp(B) Lower	Upper
PANAS-Neg Affect	-.028	.019	2.273	.972	.937	1.008
RTSQ	.007	.007	1.032	1.007	.993	1.021
BFI-Neuroticism	-.125	.031	16.461	.882***	.830	.937
DERS	-.037	.003	24.870	.963***	.949	.978
BIS	.006	.038	.029	1.007	.934	1.084

Note. *p <.05, **p <.01, ***p < .001

Table 7
Underlying dimensions as predictors of BDI scores: Linear regression

	Interpersonal Trauma			Non-Interpersonal Trauma		
Dimension	R	R^2	F	R	R^2	F
PANAS-Negative Affect	.698	.488	178.914***	.649	.421	150.784***
RTSQ	.518	.269	68.741***	.535	.286	79.846***
BFI-Neuroticism	.658	.432	144.031***	.619	.383	127.216***
DERS	.669	.447	152.806***	.612	.374	120.068***
BIS	.394	.155	34.120***	.417	.174	41.775***

Note: *p<.05, **p<.01, ***p<.001

Table 8
Underlying dimensions as predictors of PCL scores: Linear regression

	Interpersonal Trauma			Non-Interpersonal Trauma		
Dimension	R	R^2	F	R	R^2	F
PANAS-Negative Affect	.635	.403	127.054***	.642	.412	145.146****
RTSQ	.440	.193	44.781***	.332	.110	24.673***
BFI-Neuroticism	.425	.180	41.623***	.296	.088	19.661***
DERS	.549	.301	81.480***	.522	.273	75.470***
BIS	.196	.038	7.413***	.069	.005	.963

Note: *p<.05, **p<.01, ***p<.001

TEST BATTERY

Demographic Information

Please choose the best description from the options

Gender:
Male
Female

Age: _____

Ethnicity:
American Indian
Asian, Asian American, or Pacific Islander
Black, African American
Hispanic
Multiracial, Biracial
White, non-hispanic
Other: _____

Level of Education completed:
Some high school
Completed high school
Some college
Completed college
Some graduate or professional school
Completed graduate or professional school

LEC-5

Listed below are a number of difficult or stressful things that sometimes happen to people. For each event, check one or more of the boxes to the right to indicate that (a) it happened to you personally; (b) you witnessed it happen to someone else; (c) you learned about it happening to a close family member or close friend; (d) you were exposed to it as part of your job (for example; paramedic, police, military, or other first responder)

Be sure to consider your entire life (growing up as well as adulthood) as you go through the list of events.

Event	Happened to me	Witnessed it	Learned about it	Part of my job
Natural disaster				
Fire or explosion				
Transportation accident				
Serious accident at work, home, or during recreational activity				
Exposure to toxic substance (for example, dangerous chemicals, radiation)				
Physical Assault (for example, being attacked, hit, slapped, kicked, beaten up)				
Assault with a weapon (for example, being shot, stabbed, threatened with a knife, gun, bomb)				
Sexual Assault (rape, attempted rape, made to perform any sexual act through force or threat of harm				
Other unwanted or uncomfortable sexual experience				
Combat or exposure to a war-zone (in the military or as a civilian)				
Captivity (for example, being kidnapped, abducted, held hostage, prisoner of war)				
Life-threatening illness or injury				
Severe human suffering				
Sudden violent death (for example, homicide, suicide)				
Sudden accidental death				
Serious injury, harm, or death you caused to someone else				

Any other very stressful event or experience				

If you reported having experienced multiple events, please select the one that you believe has impacted you the most.

For the event you selected as your most impactful event, write the age at which you first experienced this event. If you cannot remember an exact age, please estimate to the best of your ability.

PCL- C

Below is a list of problems and complaints that people sometimes have in response to stressful life experiences. Please read each one carefully and select a response to indicate how much you have been bothered by that problem in the last month. While reading, please keep in mind the stressful life experience you previously marked as most impactful.

Response	Not at all (1)	A little bit (2)	Moderately (3)	Quite a bit (4)	Extremely (5)
Repeated, disturbing memories, thoughts, or images of a stressful experience from the past?					
Repeated, disturbing dreams of a stressful experience from the past?					
Suddenly acting or feeling as if a stressful experience were happening again (as if you were reliving it)?					
Feeling very upset when something reminded of you of a stressful experience from the past?					
Having physical reactions (e.g., heart pounding, trouble breathing, or sweating) when something reminded you of a stressful experience from the past?					
Avoid thinking about or talking about a stressful experience or avoid having feelings related to it?					
Avoid activities or situations because they remind you of a stressful experience from the past?					
Trouble remembering important parts of a stressful experience from the past?					
Loss of interest in things that you used to enjoy?					

Feeling distant or cut off from other people?					
Feeling emotionally numb or being unable to have loving feelings for those close to you?					
Feeling as if your future will somehow be cut short?					
Trouble falling or staying asleep?					
Feeling irritable or having angry outbursts?					
Having difficulty concentrating?					
Being “super alert” or watchful, on guard?					
Feeling jumpy or easily startled?					

BDI-II

1. Sadness
0 I do not feel sad.
1 I feel sad much of the time.
2 I am sad all the time.
3 I am so sad or unhappy that I can't stand it.

2. Pessimism
0 I am not discouraged about my future.
1 I feel more discouraged about my future than I used to be.
2 I do not expect things to work out for me.
3 I feel my future is hopeless and will only get worse.

3. Past Failure
0 I do not feel like a failure.
1 I have failed more than I should have.
2 As I look back, I see a lot of failures.
3 I feel I am a total failure as a person.

4. Loss of Pleasure
0 I get as much pleasure as I ever did from the things I enjoy.
1 I don't enjoy things as much as I used to.
2 I get very little pleasure from the things I used to enjoy.
3 I can't get any pleasure from the things I used to enjoy.

5. Guilty Feelings
0 I don't feel particularly guilty.
1 I feel guilty over many things I have done or should have done.
2 I feel quite guilty most of the time.
3 I feel guilty all of the time.

6. Punishment Feelings
0 I don't feel I am being punished.
1 I feel I may be punished.
2 I expect to be punished.
3 I feel I am being punished.

7. Self-Dislike
0 I feel the same about myself as ever.
1 I have lost confidence in myself.
2 I am disappointed in myself.
3 I dislike myself.

8. Self-Criticalness
0 I don't criticize or blame myself more than usual.

1 I am more critical of myself than I used to be.
2 I criticize myself for all of my faults.
3 I blame myself for everything bad that happens.

9. Suicidal Thoughts or Wishes
0 I don't have any thoughts of killing myself.
1 I have thoughts of killing myself, but I would not carry them out.
2 I would like to kill myself.
3 I would kill myself if I had the chance.

10. Crying
0 I don't cry anymore than I used to.
1 I cry more than I used to.
2 I cry over every little thing.
3 I feel like crying, but I can't.

11. Agitation
0 I am no more restless or wound up than usual.
1 I feel more restless or wound up than usual.
2 I am so restless or agitated that it's hard to stay still.
3 I am so restless or agitated that I have to keep moving or doing something.

12. Loss of Interest
0 I have not lost interest in other people or activities.
1 I am less interested in other people or things than before.
2 I have lost most of my interest in other people or things.
3 It's hard to get interested in anything.

13. Indecisiveness
0 I make decisions about as well as ever.
1 I find it more difficult to make decisions than usual.
2 I have much greater difficulty in making decisions than I used to.
3 I have trouble making any decisions.

14. Worthlessness
0 I do not feel I am worthless.
1 I don't consider myself as worthwhile and useful as I used to.
2 I feel more worthless as compared to other people.
3 I feel utterly worthless.

15. Loss of Energy
0 I have as much energy as ever.
1 I have less energy than I used to have.
2 I don't have enough energy to do very much.
3 I don't have enough energy to do anything.

16. Changes in Sleeping Pattern
0 I have not experienced any change in my sleeping pattern.
la I sleep somewhat more than usual.
lb I sleep somewhat less than usual.
2a I sleep a lot more than usual.
2b I sleep a Iot less than usual.
3a I sleep most of the day.
3b I wake up 1-2 hours early and can't get back to sleep.

17. Irritability
0 I am no more irritable than usual.
1 I am more irritable than usual.
2 I am much more irritable than usual.
3 I am irritable all the time.

18. Changes in Appetite
0 I have not experienced any change in my appetite.
la My appetite is somewhat less than usual.
lb My appetite is somewhat greater than usual.
2a My appetite is much less than before.
2b My appetite is much greater than usual.
3a I have no appetite at all.
3b I crave food all the time.

19. Concentration Difficulty
0 I can concentrate as well as ever.
1 I can't concentrate as well as usual.
2 It's hard to keep my mind on anything for very long.
3 I find I can't concentrate on anything.

20. Tiredness or Fatigue
0 I am no more tired or fatigued than usual.
1 I get more tired or fatigued more easily than usual.
2 I am too tired or fatigued to do a lot of the things I used to do.
3 I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex
0 I have not noticed any recent change in my interest in sex.
1 I am less interested in sex than I used to be.
2 I am much less interested in sex now.
3 I have lost interest in sex completely.

PANAS

This scale consists of a number of words and phrases that describe different feelings and emotions. Read each item and then mark the appropriate answer in the space next to that word. Indicate to what extent you have felt this way during the past few weeks.

Use the following scale to record your answers:

1 very slightly or not at all
2 a little
3 moderately
4 quite a bit
5 extremely

_____ cheerful _____ sad _____ active _____ angry at self

_____ disgusted _____ calm _____ guilty _____ enthusiastic

_____ attentive _____ afraid _____ joyful _____ downhearted

_____ bashful _____ tired _____ nervous _____ sheepish

_____ sluggish _____ amazed _____ lonely _____ distressed

_____ daring _____ shaky _____ sleepy _____ blameworthy

_____ surprised _____ happy _____ excited _____ determined

_____ strong _____ timid _____ hostile _____ frightened

_____ scornful _____ alone _____ proud _____ astonished

_____ relaxed _____ alert _____ jittery _____ interested

_____ irritable _____ upset _____ lively _____ loathing

_____ delighted _____ angry _____ ashamed _____ confident

_____ inspired _____ bold _____ at ease _____ energetic

_____ fearless _____ blue _____ scared _____ concentrating

_____ disgusted _____ shy _____ drowsy _____ dissatisfied with self

BFI

Here are a number of characteristics that may or may not apply to you. For example, do you agree that you are someone who likes to spend time with others? Please write a number next to each statement to indicate the extent to which you agree or disagree with that statement.

1 Disagree strongly
2 Disagree a little
3 Neither agree nor disagree
4 Agree a little
5 Agree Strongly

I see myself as someone who…

1. is talkative
2. tends to find fault with others
3. does a thorough job
4. is depressed, blue
5. is original, comes up with new ideas
6. is reserved
7. is helpful and unselfish with others
8. can be somewhat careless
9. is relaxed, handles stress well
10. is curious about many different things
11. is full of energy
12. starts quarrels with others
13. is a reliable worker
14. can be tense
15. is ingenious, a deep thinker
16. generates a lot of enthusiasm
17. has a forgiving nature
18. tends to be disorganized
19. worries a lot
20. has an active imagination
21. tends to be quiet
22. is generally trusting
23. tends to be lazy
24. is emotionally stable, not easily upset
25. is inventive
26. has an assertive personality
27. can be cold and aloof
28. perseveres until the task is finished
29. can be moody
30. values artistic, aesthetic experiences
31. is sometimes shy, inhibited

32. is considerate and kind to almost everyone
33. does things efficiently
34. remains calm in tense situations
35. prefers work that is routine
36. is outgoing, sociable
37. is sometimes rude to others
38. makes plans and follows through with them
39. gets nervous easily
40. likes to reflect, play with idea
41. has few artistic interests
42. likes to cooperate with others
43. is easily distracted
44. is sophisticated in art, music, or literature

DERS

Please indicate how often the following 36 statements apply to you by marking the appropriate number from the scale above (1 – 5) in the box alongside each item.
1 Almost never (0-10%)
2 Sometimes (11-35%)
3 About half the time (36-65%)
4 Most of the time (66-90%)
5 Almost always (91-100%)

1. I am clear about my feelings
2. I pay attention to how I feel
3. I experience my emotions as overwhelming and out of control
4. I have no idea how I am feeling
5. I have difficulty making sense out of my feelings
6. I am attentive to my feelings
7. I know exactly how I am feeling
8. I care about what I am feeling
9. I am confused about how I ffeel
10. When I'm upset, I acknowledge my emotions
11. When I'm upset, I become angry with myself for feeling that way
12. When I'm upset, I become embarrassed for feeling that way
13. When I'm upset, I have difficulty getting work done
14. When I'm upset, I become out of control
15. When I'm upset, I believe that I will remain that way for a long time
16. When I'm upset, I believe that I'll end up feeling very depressed
17. When I'm upset, I believe that my feelings are valid and important
18. When I'm upset, I have difficulty focusing on other things
19. When I'm upset, I feel out of control
20. When I'm upset, I can still get things done
21. When I'm upset, I feel ashamed with myself for feeing that way
22. When I'm upset, I know that I can find a way to eventually feel better
23. When I'm upset, I feel like I am weak
24. When I'm upset, I feel like I can remain in control of my behaviors
25. When I'm upset, I feel guilty for feeling that way
26. When I'm upset, I have difficulty concentrating
27. When I'm upset, I have difficulty controlling my behaviors
28. When I'm upset, I believe that there is nothing I can do to make myself feel better
29. When I'm upset, I become irritated with myself for feeling that way
30. When I'm upset, I start to feel very bad about myself
31. When I'm upset, I believe that wallowing in it is all I can do
32. When I'm upset, I lose control over my behaviors
33. When I'm upset, I have difficulty thinking about anything else
34. When I'm upset, I take time to figure out what I'm really feeling
35. When I'm upset, it takes me a long time to feel better
36. When I'm upset, my emotions feel overwhelming

RTSQ

For each of the items below, please rate how well the item describes you on a scale of 1 (not at all) to 7 (very well).

1. I find that my mind often goes over things again and again
2. When I have a problem, it will gnaw on my mind for a long time
3. I find that some thoughts come to mind over and over throughout the day
4. I can't stop thinking about some things
5. When I am anticipating an interaction, I will imagine every possible scenario and conversation
6. I tend to replay past events.
7. I find myself daydreaming about things I wish I had done differently
8. When I feel I have a bad interaction with someone, I tend to imagine various scenarios where I would have acted differently
9. When trying to solve a complicated problem, I find that I just keep coming back to the beginning without ever finding a solution.
10. If there is an important event coming up, I think about it so much that I work myself up
11. I have never been able to distract myself from unwanted thoughts
12. Even if I think about a problem for hours, I still have a hard time coming to a clear understanding
13. It is very difficult for me to come to a clear conclusion about some problems, no matter how much I think about it
14. Sometimes I realize I have been sitting and thinking about something for hours
15. When I am trying to work out a problem, it is like I have a long debate in my mind where I keep going over different points
16. I sit and reminisce about events from the past
17. When I am worrying about something, thoughts of it interfere with what I am working on
18. Sometimes even during a conversation, I find unrelated thoughts popping into my head.
19. When I have an important conversation coming up, I tend to go over it in my mind again and again
20. If I have an important event coming up, I can't stop thinking about it.

BIS/BAS

Each item of this questionnaire is a statement that a person may either agree with or disagree with. For each item, indicate how much you agree or disagree with what the item says. Please respond to all the items; do not leave any blank. Choose only one response to each statement. Please be as accurate and honest as you can be. Respond to each item as if it were the only item. That is, don't worry about being "consistent" in your responses. Choose from the following four response options:

1 = very true for me
2 = somewhat true for me
3 = somewhat false for me
4 = very false for me

1. A person's family is the most important thing in life.
2. Even if something bad is about to happen to me, I rarely experience fear or nervousness.
3. I go out of my way to get things I want.
4. When I'm doing well at something I love to keep at it.
5. I'm always willing to try something new if I think it will be fun.
6. How I dress is important to me.
7. When I get something I want, I feel excited and energized.
8. Criticism or scolding hurts me quite a bit.
9. When I want something I usually go all-out to get it.
10. I will often do things for no other reason than that they might be fun.
11. It's hard for me to find the time to do things such as get a haircut.
12. If I see a chance to get something I want I move on it right away.
13. I feel pretty worried or upset when I think or know somebody is angry at me.
14. When I see an opportunity for something I like I get excited right away.
15. I often act on the spur of the moment.
16. If I think something unpleasant is going to happen I usually get pretty "worked up."
17. I often wonder why people act the way they do.
18. When good things happen to me, it affects me strongly.
19. I feel worried when I think I have done poorly at something important.
20. I crave excitement and new sensations.
21. When I go after something I use a "no holds barred" approach.
22. I have very few fears compared to my friends.
23. It would excite me to win a contest.
24. I worry about making mistakes.