AN EXAMINATION OF RISK AND RESILIENCE FACTORS PREDICTING EXECUTIVE FUNCTIONING IN WOMEN FOLLOWING PSYCHOLOGICAL TRAUMA

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Psychological trauma may affect higher-order executive functions, which include selective attention, inhibition, and task-switching processes. Difficulty in these executive processes can in turn influence individuals' daily functioning and may also negatively affect the psychological treatment of post-trauma symptoms. Women may be most at risk for developing problems with executive functioning following trauma, consistent with their overall greater risk of developing post-trauma symptoms. Yet, little is understood about the influence of psychological variables, premorbid functioning, and specific trauma factors in determining post-trauma cognitive functioning in women. Additionally, individual variability in susceptibility to psychological distress and neuropsychological deficits following trauma remains an open area of study. The present study investigated the relationship between psychological and trauma factors with neuropsychological outcomes in women with trauma histories as well as individual variability in risk for poor neuropsychological outcomes. In total, 60 participants' data (age M = 29.73, SD = 10.91) were included in analyses. The final sample consisted of 33 community members recruited from the UNT Psychology Clinic and the UNT student body and 27 veterans recruited from the Veterans Affairs North Texas Healthcare System (VANTHCS). Regression and path analysis identified premorbid intellectual functioning as a predictor of better neuropsychological outcomes and anxiety and depression symptoms as risk factors for worse neuropsychological functioning. Person-centered cluster analyses focused on individual differences in outcomes identified three groups differing in psychological distress and neuropsychological functioning. Additional analyses identified differences in trauma exposure,

psychological functioning, and neuropsychological performance between subgroups of civilians and veterans and those with and without a history of PTSD.

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INTRODUCTION

Research on the sequelae of psychological trauma has revealed that experiencing significant negative life events can affect emotional and interpersonal functioning, resulting in increased depression, anxiety, guilt, and anger as well as strained familial, marital, and social relationships (Frueh, Turner, Beidel, & Cahill, 2001; Zoladz & Diamond, 2013). While psychological research has tended to focus on emotional facets of PTSD, neuropsychological trauma research has highlighted difficulty with cognitive processes among individuals who have experienced trauma. Specifically, individuals who experience trauma may have more difficulty with higher-order executive functions (EF), including selective attention, task-switching, and inhibition processes (Aupperle, Melrose, Stein, & Paulus, 2011). Difficulty in these functions may negatively influence activities of daily living and also create a barrier to cognitive therapies that are often used for treatment of post-trauma symptoms.

Women have an increased risk of experiencing certain types of trauma (Tolin & Foa, 2006), and also have an increased risk of developing maladaptive emotional processes and cognitive content following trauma, leading to a heightened probability of developing PTSD (Inslicht et al., 2013). Moreover, emerging research on the relationship between trauma and EF suggests that women's cognitive processes may be affected by trauma to a greater extent than those of men (Stricker, Keller, Castillo, & Haaland, 2015). This research is significant, as it highlights the need for a better understanding of how women are cognitively affected by trauma, and how dysfunctional cognitive processes may be implicated in psychological disorders, therapy outcome, and daily living.

Just as only a small percentage of individuals who experience trauma will develop emotional dysregulation leading to PTSD, not all of those who experience trauma will sustain

impairments in EF. Person-centered analyses such as cluster analysis or latent class analysis can provide important information about who is most at risk for poorer psychological and neuropsychological outcomes following trauma, thus providing insight into who within this sub-sample of trauma-affected individuals may require more specific psychological or neuropsychological interventions.. Furthermore, a variable-centered examination of psychological, premorbid cognitive, and trauma factors and their role in risk and resilience may lead to a better understanding of what influences may determine cognitive disruptions following trauma.

The Role of Executive Functioning in Cognition

EF encompasses complex cognitive processes that allow for effortful control and contribute to goal-directed behavior (Lezak, 1995) and includes skills related to attentional set-shifting, updating processes within working memory, and inhibitory control (Miyake et al., 2000; Tsuchida & Fellows, 2012). Attentional set-shifting involves the ability to switch between tasks, or learn from new response rules placed on a current task. Updating processes require manipulating information within working memory, and may involve either verbal or visuospatial components. Inhibitory control is the ability to suppress an automatic response in order to engage in a more controlled, task-appropriate response.

Along with measuring facets of EF through neuropsychological testing, there has been a move within the field to conceptualize EF in terms of ecological applications (Burgess et al., 2006). Specifically, EF appears to have applications to in vivo self-regulation, decision-making, and ability to complete complex tasks. It contributes to the ability to stay on task, resist temptation, problem solve, and plan (Diamond, 2013). Cognitive components of EF may interact

with emotional processing to create a type of “hot” EF, where emotional salience, motivation, and arousal influence cognitive-affective control (Zelazo et al., 2005).

Executive processes depend heavily on the prefrontal cortex (PFC). Neuroimaging studies support the notion that larger PFC volume is associated with better performance on executive tasks (Yuan & Raz, 2014) and lesions of the PFC can cause drastic impairment of EF (Alvarez & Emory, 2006). Within the PFC, the dorsolateral region is involved in working memory processes and cognitive flexibility (“cool” EF), while the ventrolateral and orbitofrontal cortexes are involved in EF tasks with an emotional or stimulus-reward component (“hot” EF) (Iordan, Dolcos, & Dolcos, 2013).

Imaging studies have revealed that, in addition to PFC involvement, frontal subcortical circuits appear to play an important role in EF (Bonelli & Cummings, 2007). Projections from the PFC connect to the basal ganglia and thalamus, forming dorsal and ventral systems, which are involved in the processing of sensory, spatially related, and emotional information. Three specific frontal-subcortical circuits appear to have relevance to higher-order information processing and decision-making. The dorsolateral circuit, involving the dorsolateral PFC, basal ganglia, and thalamus, is largely responsible for cool EF. The anterior cingulate cortex (ACC) circuit involves motivational mechanisms, while the orbitofrontal circuit integrates amygdala functions in order to process emotionally salient information to form appropriate task-relevant responses. The latter two circuits are considered ventral systems, and appear to be more relevant to hot executive processes.

Diversity in EF capability can be observed both in healthy individuals and in those with a variety of psychological disorders. In healthy adults, genetic heritability appears to largely contribute to EF development and control (Miyake & Friedman, 2012), and EF appears to show

developmental stability. Though updating, set-shifting, and inhibition all appear to improve from childhood to adulthood and later show mild decline in older adulthood, there is evidence to suggest that those who demonstrate poorer EF early in life will likely continue along this same trajectory as they progress through development.

In addition to individual variability that is manifested across healthy adults, neuropsychological and psychological disorders can greatly affect EF. Acquired brain injuries, including traumatic brain injuries and cardiovascular accidents (CVA), can disrupt typical PFC and frontal subcortical circuit functioning to such an extent that EF may be mildly to severely impaired (Zinn, Bosworth, Hoenig, & Swartzwelder, 2007; Gregorio et al., 2015; Caeyenberghs et al., 2012). The extent of the damage caused by brain injury or stroke, as well as the location of the damage, can influence specific areas of functioning, though typically impaired patients will see global deficits in higher-order thinking and decision making skills.

While brain injuries involve discrete events affecting brain function, more chronic psychological disorders may also affect EF. Major Depressive Disorder (MDD) has been linked to significant impairment in many aspects of EF, including the core processes of shifting, inhibition, and working memory updating. Furthermore, MDD appears to create inefficiencies in EF abilities involving planning and verbal fluency. Disturbances in EF abilities are hypothesized to be due to structural and functional abnormalities found in the frontal lobes of patients with MDD, including abnormalities in the dorsolateral PFC, ventrolateral PFC, and ACC (Snyder, 2013). Furthermore, a multitude of other psychological disorders may also present with EF deficits, including obsessive compulsive disorder (Morein-Zamir et al., 2016), attention deficit-hyperactivity disorder (Sjowall, Roth, Lindqvist, & Thoreel, 2013), generalized anxiety disorder (Visu-Petra, Miclea, & Visu-Petra, 2013), and the focus of the present study, PTSD.

With PTSD, as well as other disorders related to EF dysfunction, there is some question about the direction of the relationship between the disorder and the breakdown in higher-order cognition. It is possible that poorer EF may make a disorder more likely to develop, and therefore dysfunctional executive processes may be a cause rather than an effect of these disorders. Yet with PTSD, as with other disorders, it is difficult to do prospective research to parse out the directionality of this relationship. Therefore, most studies will reference a correlation between various disorders and EF, rather than a causal effect.

Trauma and Executive Functioning

Among other neuropsychological and psychological contributors to EF dysfunction, psychological trauma and resulting post-trauma symptoms, including those found in posttraumatic stress disorder (PTSD), are emerging as an area of particular focus. Within the last decade, the neurocognitive sequelae of trauma, as well as the role of cognition in the development and perpetuation of PTSD, has become a more prominent area of study (Vasterling & Verfaellie, 2009). Specifically, the neurobiological underpinnings of cognitive symptoms in PTSD (Blair et al., 2013), along with the demonstration of cognitive inefficiencies via neuropsychological testing (Marx et al., 2009; Aupperle, Melrose, Stein, & Paulus, 2011), have gained more interest in recent years.

In line with the traditional understanding of PTSD as a disorder of emotional dysregulation, it has been theorized that emotionally provocative information may elicit an atypical neurocognitive response in individuals that have experienced trauma (Buckley, Blanchard, & Neill, 2000). This hypothesis is in line with the conceptualization of hot EF, which incorporates affectively salient information with cognitive demands. Neurobiological research

has proposed a dysfunctional mechanism in those with PTSD, consisting of HPA activation resulting in glucocorticoid release along with release of norepinephrine, epinephrine, and dopamine. These hormones may work in concert to initially decrease PFC and hippocampal activity and increase amygdala activity, creating fear memories, and over time lead to significant dysregulation in these areas (Bremner, 2016). Specific areas of the PFC affected in those with PTSD include the dorsal anterior cingulate cortex (dACC) and the ventrolateral PFC (Etkin, Egner, & Kalisch, 2011).

Further evidence for dysregulation of hot EF has been uncovered using tasks with high cognitive demands, which also incorporate trauma-related affective information or risk/reward incentives (Blair et al., 2013; Caparos & Blanchette, 2014). Results from studies examining hot EF reveal that individuals who have experienced trauma may have facilitated attention to, along with more difficulty disengaging attention from, negative and more specifically trauma-related stimuli (Bar-Haim, Lamy, Pergamin, Bakermans-Kranenburg, & van Ijzendoom, 2007; Caparos & Blanchette, 2014). Within the context of neuropsychological assessment, this has most frequently been demonstrated via slowed response times to negative or trauma-related words on a modified Emotional Stroop task (Ashley, Honzel, Larsen, Justus, & Swick, 2013) and attentional bias when presented with threatening faces in a Dot Probe Task (Lin et al., 2014).

Hot EF has been further investigated in terms of response to task demands high in risk and reward, eliciting problem solving ability combined with motivational and affective salience. Results from the Iowa Gambling Task (IGT) used in combination with structural neuroimaging suggest that Operation Enduring Freedom/Operation Iraqi Freedom (OEF/OIF) veterans with PTSD perform worse on this task when compared to controls (Fogleman et. al, 2017). Within the sample, PTSD interfered with the ability to make decisions when confronted with high risk and

reward. Imaging implicated gray matter morphometry in the lateral prefrontal cortex as predicting the link between PTSD and IGT performance, again supporting the notion of dysfunction in hot EF circuitry in this population.

While tasks such as the Emotional Stroop task and IGT provide insight into how an individual with PTSD may perform in a neuropsychological testing setting, it is also important to conceptualize how deficits in hot EF may present in real-world settings. Difficulty appropriately processing affectively salient information, inhibiting responses to potentially threatening information, and making decisions based on risk and reward may perpetuate PTSD symptoms, including hypervigilance and arousal. Additionally, individuals who have difficulty appropriately attending to emotional and motivational information may struggle to successfully participate in PTSD treatment such as Cognitive Processing Therapy (CPT) and Prolonged Exposure Therapy (PE), which place a high demand on development of appropriate emotional responses and adaptive cognitive reappraisal of the traumatic experience (Vasterling & Verfaellie, 2009).

While initial theories surrounding neurocognitive functioning following trauma emphasized the role of affect in EF dysregulation, emerging evidence suggests that dorsolateral PFC circuitry involved in traditional cool EF may also be meaningfully affected by traumatic experiences. On neuropsychological tests, individuals with trauma histories may show below average to impaired performance on tasks of attention, updating in working memory, and inhibitory functioning, all tasks central to EF (Aupperle, Melrose, Stein, & Paulus, 2011). Individuals with PTSD resulting from sexual assault or combat have evidenced decreased performance on the digit span forward task of simple attention and both digit span backward and letter-number sequencing tasks of working memory (LaGarde, Doyon, & Brunet, 2010; Samuelson et al., 2006). Attentional set-shifting, as assessed in tasks such as the Trail Making

Test may be difficult for individuals with PTSD, though evidence for and against this effect is mixed (Stein et al., 2012; Leskin & White, 2007). Additionally, a relationship may exist between trauma and decreased sustained attention, as assessed by the Continuous Performance Task (CPT). Specific error patterns on this task include increased omissions and slowed response time (Vasterling et al., 2002; Shucard, McCabe, & Szymanski, 2008), in addition to increased commissions, which may be related to a breakdown in inhibitory capacity. Results from other tasks of inhibition, including the traditional Stroop task (Cui, Chen, Liu, Shan, & Jia, 2014), Go/No-Go Task (Falconer, Allen, Felmingham, Williams, & Bryant, 2013), and Attentional Network Task (ANT; Leskin & White, 2007) provide further evidence for decreased inhibition in those who have experienced significant trauma.

As with the effects of hot EF, the deficits resulting from decreased cool EF may also contribute to maintenance of PTSD symptoms and poorer response to cognitive-based therapies. A recent study examined the relationship between neurocognitive functioning and response to psychotherapy in female veterans with PTSD (Haaland, Sadek, Keller, & Castillo, 2016). Results revealed that those participants with higher learning and memory capacity had better outcomes following a 16-week group psychotherapy intervention. Furthermore, those who responded better to the intervention evidenced significant improvement in inhibition/switching capacity. The correlation between emotional symptom improvement and improvement in EF provides support for the role of EF in maintaining PTSD symptoms, and the importance of addressing EF in treatment.

Of note, emerging research on the neurocognitive effects of trauma primarily focuses on individuals who have experienced a traumatic event and meet criteria for PTSD. This research approach, separating individuals with PTSD from those without the disorder, has provided useful

information demonstrating that those with PTSD may show more deficits in both hot and cool EF. Thus, the present study provides secondary analyses to examine groups in this fashion. However, a dichotomous research approach, separating individuals into PTSD and non-PTSD groups, or even further separating into PTSD, trauma without PTSD, and no trauma groups, does not take into account the potential within-group differences for these individuals. The nature of psychopathology is likely continuous rather than categorical (Wright et al., 2013), and therefore separation into groups does not capture the full range of presentations.

It is possible that some individuals who experience trauma but do not meet criteria for PTSD may still have significant difficulty with cognitive and cognitive-affective tasks. Additionally, for those who do meet criteria for PTSD, there may be variability in how much of a cognitive effect is evidenced. Emerging literature supports this notion, finding that military deployment correlates with smaller prefrontal gray matter volume regardless of PTSD status, with trauma exposure and psychological symptoms playing a bigger role in determining neuropsychological sequelae than a dichotomous identification of "PTSD" or "No PTSD" (Butler et al., 2017). Therefore, while the present study assessed for differences between those with and without a formal diagnosis of PTSD, the main study objective was to further explore what factors may provide within-group differences across the full spectrum of PTSD symptomology and predict who exhibits better neurocognitive functioning following trauma.

Sex Differences in Post-Trauma Functioning

Trauma literature suggests women and men are differentially affected by traumatic events (Fullerton et al., 2001; Cohen & Hien, 2006). This difference begins with the frequency and type of events experienced by each sex (Tolin & Foa, 2006; Sullivan, Contractor, Gerber, &

Neumann, 2017), and is manifested in different physiological, emotional, and behavioral reactions to trauma (Inslicht et al., 2013). Emerging research also highlights potential sex differences in the neurocognitive effects of trauma (Stricker, Keller, Castillo, & Haaland, 2015), though there are gaps in the literature that the present study aims to address.

Meta-analyses reveal that men experience traumatic events at a higher rate than women (Tolin & Foa, 2006). Yet, the type of trauma each sex is more at-risk for varies. While males and females are equally likely to experience nonsexual child abuse, females are at an increased risk of childhood sexual abuse. They are also more likely than men to experience sexual assaults in adulthood. Alternatively, men are more likely to experience physical assault, combat trauma, accidents (including motor vehicle accidents), and significant illnesses or injuries.

Due to the different rates of experiencing certain types of trauma, it has been proposed that women's risk for greater post-trauma emotional and psychological symptoms, leading to a greater risk for PTSD, may be due to the increased risk of sexual abuse or assault. In other words, this theory suggests that sexual assault and abuse may create a stronger response in victims than do the traumas experienced more frequently by men. Some researchers suggest that assault/abuse events are associated with more betrayal, and this betrayal creates cognitive discrepancies for the trauma victim that may lead to more significant PTSD symptom presentation (Tang & Freyd, 2010). Yet, there is also evidence to suggest that as differences in traumatic event prevalence are accounted for, sex differences in rate of PTSD are still significant (Tolin & Foa, 2006; Cromer & Smyth, 2010). When taken together, these findings suggest that interpersonal violence experienced by women puts them at a higher risk for PTSD, and that this risk is likely due to a combination of cognitive structuring and other, more sex-specific, factors.

Examining sex differences in a mixed-sex military population provides some insight into what social and behavioral factors outside of type of trauma may account for discrepancies in post-trauma symptom severity and PTSD development. Within a military setting, men and women are exposed to similar traumatic experiences, and are each at a heightened risk of developing negative post-trauma symptoms. Yet, women in this population are still at an increased risk of developing PTSD compared to men, and it is likely that this increased risk is associated with pre-trauma, peri-trauma, and post-trauma factors. In terms of pre-trauma factors, previous exposure to interpersonal trauma, such as childhood sexual abuse or sexual assault in adulthood, may put females at an increased risk of subsequent stress exposure and compounded or complex trauma effects when compared to men (Vogt et al., 2011). Relationship concerns prior to and during deployment may increase stress levels and impact how a person perceives a traumatic event. While the effect of relationship concerns may be the same in men and women, it may be more likely to be experienced in women than in men. Additionally, social support may be more important for women to cope with trauma, and inadequate social support post-deployment may have a greater effect on women than on men. Finally, women are at a greater risk of experiencing military sexual trauma (MST) then men, thus lending further support to the hypothesis that type of trauma may have an influence on post-trauma functioning.

Results from military studies suggest that factors such as prior trauma experience, additional stressors, cognitive structuring at the time of the event, and social support following trauma each may play a role in women's increased risk of PTSD, and these findings can easily be extrapolated into civilian settings. Yet, while social factors and trauma factors appear to be related to the development of post-trauma emotional symptoms, psychological and physiological factors appear to also play a role as well. In terms of psychological risk factors, women are at a

greater risk than men for nearly all anxiety disorders (McLean, Asnaani, Litz, & Hofmann, 2011). Women diagnosed with an anxiety disorder are also more likely than men to be diagnosed with another anxiety disorder. In line with this difference in prevalence rates, women who have high trait anxiety may be more likely to perceive events as traumatic, and may be less resilient to the effects of these events (Scali et al., 2012). This difference may be attributable to a differing underlying stress response in women high in anxiety than in men.

Neuroticism represents another psychological trait with higher prevalence in females than males, and another potential predisposing factor for PTSD (Breslau & Schultz, 2013). Neuroticism is a measure of negative emotionality, or tendency to experience more negative emotions including worry, irritability, and depression. On average, women's neuroticism scores tend to be approximately half a standard deviation above those of men (Chapman, Duberstein, Sorensen, & Lyness, 2007). Both prospective longitudinal studies (Breslau & Schultz, 2013) and retrospective studies (McFarlane, 1989; Hyer et al., 1994) have linked higher neuroticism with an increased risk of PTSD development following experience of a trauma. As is likely the case with anxiety, this link may be a result of individuals with higher rates of neuroticism appraising events as more threatening, and therefore having greater stress responses to these events (Servaas, Riese, Ormel, & Aleman, 2014).

Closely related to psychological factors influencing sex differences in the emotional toll of trauma, physiological factors may also increase women's risk of developing PTSD. Differences in hormone activation in the stress response may affect how women utilize different parts of their brain to understand and cope with trauma both during and after a traumatic event (Zer-Aviv & Akirzav, 2016; Bruce et al., 2013). Specifically, the stress hormone cortisol has been shown to play an important role in fear learning during a traumatic event and fear extinction

following trauma. While fear learning plays an evolutionary adaptive role in response to a stressful stimulus, dysregulation in this pathway can lead to heightened and inappropriate fear responses associated with anxiety disorders and PTSD. Additionally, impaired fear extinction may play a key role in perpetuating dysfunctional emotional processes following trauma, thus prolonging and exacerbating PTSD symptoms. In males and females, cortisol may act differently in the initiation of fear learning, thus contributing to sex differences in PTSD development (Zorawski, Blanding, Kuh, & LaBar, 2006). Specifically, higher correlations have been found between cortisol levels and fear acquisition in men than in women, suggesting fear acquisition may work via different mechanisms in each sex.

Estrogen levels have also been proposed to play a role in this fear acquisition sex difference. Normal estrogen fluctuations occur throughout a woman's menstrual cycle, and estrogen levels also significantly increase and decrease across the reproductive lifespan. Evidence from both animal (Walf, Paris, & Frye, 2009) and human models (Glover et al., 2012) suggests that high estrogen levels may play a role in buffering against fear-learning while also increasing fear-inhibition in extinction. Low levels of estrogen, however, may predict a stronger fear-potentiated startle response and poorer fear extinction in women who have been exposed to trauma. This suggests that low estrogen may play a role in the development of post-trauma emotional dysregulation in women while also inhibiting extinction. When examined in terms of sex differences in PTSD development, these results highlight an important physiological dichotomy in the mechanisms behind PTSD development in women versus men.

Differences in hormone applications for men and women's fear learning during trauma may influence how brain regions are utilized on a more macro-level. When looking at the effect of estrogen level on emotional reactivity, differences can be seen between reactivity of both the

amygdala and hippocampus based on changes in hormonal levels (Cover, Maeng, Lebron-Milad, & Milad, 2014). This difference influences reactivity to emotional stimuli, and can be seen on neuropsychological tests of hot executive functioning, including an emotional Go-No Go task. In terms of sex differences, functional MRI research also suggests that men with PTSD may have increased activation in the left rostral dorsolateral ACC (dACC) during extinction recall when compared with women (Shvil et al., 2014). While the implications of this difference are not well understood, it does highlight the role sex, and specifically sex hormones in relation to cortisol, play in the fear pathway and eventual development of PTSD.

Women's Neurocognitive Functioning following Trauma

Just as women are at a differential risk for emotional regulation deficits associated with PTSD, women may also display difficulty with neurocognitive functioning to a greater extent than men following trauma. It is theorized that these differences may exist primarily in the effective higher-order processing of information present in both cool cognitive and hot emotional-cognitive EF. Yet, this remains an open area of research, and emerging studies are only beginning to investigate this relationship.

Much of the emerging work that has identified areas of weakness in executive processes following trauma has used mixed-sex or predominantly male military samples (Baker et al., 2012; Vogt et al., 2011), making literature on sex differences in EF limited. Some evidence for sex differences in post-trauma neurocognitive functioning emerges from additional analyses or discussion points in studies utilizing mixed-sex samples. In one early study examining the relationship between PTSD symptoms and EF, the Trail Making Task and the Attentional Network Task were used to assess a sample of young civilian adults who had experienced trauma

(Leskin & White, 2007). Though sex difference analyses were not the primary purpose of the study, researchers found that EF was related to PTSD symptoms in women, but not in men. This result suggested that PTSD symptoms might have a stronger link to neurocognitive functioning in women than in men. In the discussion of these results, the authors suggest that this sex difference may account for mixed literature on the effects of trauma on EF, and should be further examined in future research.

In another pilot study examining the relationship between trauma experience, PTSD symptoms, and EF, these factors were again examined in a mixed sex sample (Sullivan & Neumann, 2016). Both cool and hot EF were studied via the traditional and modified Emotional Stroop paradigms. Results from this study demonstrated that the relationship between trauma factors and EF was stronger in women than in men. This was true for both tasks incorporating emotionally-salient information (hot EF) and those depending on cool cognitive processing (cool EF). When taken together, these initial studies highlight the importance of examining specific EF capabilities in women who have been affected by trauma.

As some evidence for the relationship between sex and neurocognitive functioning following trauma has emerged, the need to study the specific cognitive functioning strengths and weaknesses in traumatized women has been emphasized. Some studies have approached this topic by examining early-life trauma, including neglect and abuse, in predominantly female (Gould et al., 2013) or all female samples (Navalta et al., 2006). These studies have provided evidence of inefficiencies in EF, and specifically inhibitory capacity. Furthermore, results from a study assessing neurocognitive functioning in women with PTSD resulting from intimate partner violence suggest that those with PTSD have slower processing speed across EF and non-EF tasks than those without a diagnosis of the disorder (Twamley et al., 2009). While this evidence

provides important support for the study of EF in women following trauma, each of these studies is limited in trauma scope and does little to examine risk and resilience factors predicting neurocognitive performance.

A study with a broader scope of trauma history evaluated women with PTSD in terms of cognitive functioning and functional capacity (Kaye et al., 2014). Results revealed that women with PTSD demonstrated mild, but significant cognitive inefficiencies when assessed via the MATRICS Consensus Cognitive Battery. T-scores on a trail making task, symbol coding, animal naming, letter-number span, spatial span, HVLT-R, BVMT-R, CPT, and NAB all fell within the 40 to 49 range, thus reaching the low average to average threshold. Functional capacity was similarly on the low average end when compared to individuals without PTSD. This study provided support for the fact that women with PTSD may experience some below average ability in cognitive processes. While this study lent support for the relationship between women's neurocognition and PTSD, like prior studies it also did not examine risk and resilience factors predicting performance.

In addition to those studies addressing civilian women, this area has also begun to be explored with female veterans. The first study to investigate the relationship between trauma and neurocognitive processes in an all-female veteran sample was published just two years ago (Stricker, Keller, Castillo, & Haaland, 2015), reinforcing the novelty of this important emerging area. In this study, female veterans with PTSD displayed poorer EF than did a control group of female veterans without the disorder. This was evidenced by worse performance on tasks of working memory and response inhibition/switching. The effect of this decreased performance was linked to greater overall learning difficulty, which has been hypothesized to interfere with functioning following trauma and make interventions promoting recovery more difficult.

While Kaye and Stricker's studies demonstrate differences in EF of women with and without PTSD, they, like most literature in this emerging field, studied PTSD versus non-PTSD groups without accounting for those who have experienced significant trauma but do not meet full criteria for the disorder. Additionally, prior researchers may have overlooked other differences that could account for variability in performance, including level of trauma exposure, type of trauma exposure, differences in premorbid cognitive functioning, and differences in psychiatric comorbidity. Notably, all of these factors differed between the two groups in Stricker's study. Therefore, while emerging literature indicates that some women may have greater EF deficits following trauma than others, it does little to explain risk and resilience factors for these neurocognitive differences. This is an important gap in the literature that the present study seeks to fill.

Risk and Resilience Factors predicting Post-Trauma Outcomes

The primary purpose of the present study was to examine the risk and resilience factors predicting EF capacity following trauma in women. Evidence for the presence of risk and resilience factors comes from both the limited research base examining neurocognition in women post-trauma (Stricker, Keller, Castillo, & Haaland, 2015; Kaye et al., 2014) and the broader literature on resilience to PTSD symptoms (Charney, 2004; Baker et al., 2012). Results from these trauma studies suggest that not all those who experience trauma respond similarly, with some individuals being more affected than others. The idea of resilience suggests that some individuals who experience trauma may have protective factors that predict a better outcome, whether in terms of emotional stability or EF post-trauma. When measured via self-report, many

women who have experienced high levels of trauma also endorse high resilience, which may be tied to better emotional and neurocognitive outcomes (Scali et al., 2012).

The concept of resilience in terms of emotional functioning following trauma has been examined throughout PTSD literature in both civilians and veterans, and can be useful in informing the present research. One study of over 2,000 Marines provides a preliminary evaluation of risk and resilience factors to post-trauma symptoms (Baker et al., 2012). Yet, this examination was limited in terms of its generalizability to women, as the sample was comprised completely of male participants. Additionally, the exact mechanisms of risk and resilience were not delved into within this study. Nevertheless, the large longitudinal study sought to examine the relationship between biological, genetic, neuropsychological, psychological, and combat-specific factors with post-deployment psychological functioning. Looking at biological risk, blood immune-biomarkers were found to be possible predictors of post-deployment PTSD risk. In terms of historical events impacting PTSD presentation, a combat-related TBI and early life trauma appeared to place a greater risk on participants. Perceived support was also important, as lower perceived support was correlated with a greater risk for developing aggression and other psychological effects related to PTSD.

An additional study of risk factors for PTSD looked at pre-deployment cognitive features as a risk factor for the development of the disorder (Marx et al., 2009). Findings from this analysis suggest that poorer pre-trauma visual immediate memory may be related to development of PTSD following trauma. This relationship may also be dependent on pre-deployment psychological functioning, as neurocognitive performance had a stronger effect on post-deployment PTSD symptoms in those with higher pre-deployment symptoms (who were therefore already at an increased risk). These results highlight the concept of resilience factors

providing a link between trauma and post-trauma functioning. Additionally, they emphasize the need for further research into neurocognitive performance following trauma, as this may predict functioning in other capacities. As with the Marine resilience study (Baker et al., 2012) though, the Marx et al. study has limited generalizability to women, as women represent a small percentage of the overall sample.

The aforementioned studies support the notion of resilience factors predicting emotional or general neurocognitive outcomes following trauma, albeit mostly in men. While there is only limited research on resilience factors predicting EF following trauma in women, burgeoning studies support the fact that this area is important for further investigation. Though resilience was not the primary focus of study, Stricker and colleagues' 2015 publication provides support for the idea of resilience in terms of EF, finding that not all those who experience trauma demonstrate the same deficits in working memory and inhibitory capacity. An additional preliminary study informing the present research (Sullivan & Neumann, 2016) also demonstrates that there are some individuals, specifically women, who experience a great deal of trauma and yet do not perform poorly on tests of EF. Thus, though it is a novel area of exploration, the notion that resilience likely plays a factor in EF following trauma in women is supported by early literature, and remains an area in need of further investigation.

Evidence from the broader PTSD literature base and general EF research suggests that predictors of a better outcome following trauma may lie in cognitive, personality, mood, and trauma-related factors. Pre-trauma cognitive ability may predict how individuals respond to trauma, both emotionally and neurocognitively. For example, there is evidence to suggest that higher cognitive ability before exposure to a combat trauma may be a protective factor putting an individual at a lower risk for developing PTSD symptoms (Gale et al., 2008). Better pre-trauma

performance on specific neuropsychological tasks, including those assessing visual immediate memory, may also be a protective factor predicting lower likelihood of developing PTSD (Marx et al., 2009). While it may be obvious that better pre-trauma EF likely also predicts better post-trauma EF, more general cognitive ability may have the same buffering effect. Unlike other more fluid domains of cognition, like EF, measures of crystallized intelligence administered post-trauma may provide insight into the relationship between cognitive ability and EF following trauma.

Personality factors may also affect how individuals respond to trauma, both in relation to PTSD symptoms and neurocognitive domains, including EF. Specifically, neuroticism has been linked to poorer overall functioning following PTSD. Relevant to the present population of interest, the neural underpinnings of neuroticism have been studied in women high on this personality trait. Results from fMRI assessments suggest that women who endorse high levels of neurotic traits show decreased activation of the retrosplenial and visual cortex when worrying. (Servaas, Riese, Ormel, & Aleman, 2014). These neuronal effects may impact autobiographical specificity in memory formation and visual mental imagery. Deficits in these areas may affect effective processing of trauma information, and perpetuate PTSD symptoms. In terms of neurocognitive functioning, the results of this fMRI study provide evidence that neuroticism impacts how an individual processes information, and may enhance risk for poorer EF in general, but especially when coping with a traumatic event.

Specific psychological disorders can influence EF, and potentially compound the effects of trauma on neurocognitive functioning. While trauma in itself is likely to affect EF, regardless of PTSD diagnosis, it is hypothesized that those who have more severe PTSD symptoms likely also exhibit poorer EF (Stricker, Keller, Castillo, & Haaland, 2015; Olff, Polak, Witteveen, &

Denys, 2014). Since trauma may result in EF deficits even without a formal PTSD diagnosis, PTSD symptoms may be viewed more as a risk factor for greater EF deficits following trauma rather than a definite cause. In addition to PTSD, other internalizing disorders can also contribute to difficulty with higher-order cognitive processing (Snyder, 2013). Because women are at an increased risk of experiencing internalizing symptoms, and are more likely to be diagnosed with one of these disorders, comorbid internalizing disorders are an important aspect to examine when looking at risk and resilience factors for EF after trauma. Studies have suggested that Major Depressive Disorder, for example, may mediate the relationship between trauma, PTSD, and deficits in some specific EF domains, specifically set-shifting and working memory (Olff, Polak, Witteveen, & Denys, 2014). These results emphasize the importance of examining a variety of mood disorder factors when analyzing risk and resilience in post-trauma women.

Trauma-related factors, including type of trauma (White et al., 2014), intensity of trauma (Marx et al., 2009), duration of trauma (Navalta et al., 2005), and age at which trauma occurred (Lupien, McEwen, Gunnar, & Heim, 2009; Marx et al., 2009), have been some of the most widely studied risk and resilience factors for psychological and neuropsychological functioning following a traumatic event. While the relationship between these trauma factors and EF has not been extensively examined, results from the literature can help identify how these factors may relate to risk and resilience associated with neurocognitive outcomes. In terms of type of trauma, it has been proposed that assault, and specifically sexual assault, may place an individual at an increased risk of PTSD when compared to other categories of traumatic events. One large study, examining 2,142 adults, found that over 35% of individuals who had experienced an assault met criteria for PTSD (White et al., 2014). Though these events were reported less frequently than other traumatic events by the sample as a whole, they were far more likely to result in a

diagnosis of PTSD than any other type of trauma. Since women are at an increased risk of experiencing sexual abuse and assault, and since this type of trauma leads to poorer overall functioning following trauma, it is possible that the assault trauma category may act as a risk factor for poorer EF in women following a traumatic event.

Just as the category of trauma may act as a risk or resilience factor predicting EF outcome, intensity of the traumatic event may also provide insight into who performs best neurocognitively post-trauma. Intensity of a stressor has been studied in relation to neuropsychological performance within an examination of risk and resilience factors in military members (Marx et al., 2009). In terms of reaction time, a raw measure of cognitive speed and efficiency, intensity of a traumatic event may actually predict better performance, evidenced by faster reaction speed. This enhanced simple reaction speed may be related to overall behavioral reactivity, which is likely to increase as a stressor becomes more salient. While this observation suggests that intensity of a traumatic event may affect neuropsychological functioning, it does little to explain more complex tasks of EF, such as inhibition or attention. Additionally, a sample comprised of approximately 6% women (Marx et al., 2009) may not generalize well to how females respond to intense stressful events.

Literature on prolonged or repeated stressors suggests that duration of trauma and time since the experience of trauma may also impact neuropsychological outcomes. Duration of childhood sexual abuse has been found to have a strong correlation to memory function, as assessed by the Memory Assessment Scale (MAS), in college-aged women (Navalta et al., 2005). While this result is not specific to EF, it is likely that a longer duration of a traumatic event may impact this area of neurocognition in a similar manner. Time since traumatic experience may also be related to neurocognitive performance. Specifically, more chronically

presenting PTSD symptoms that continue to be evidenced once a significant amount of time has passed after the trauma appear to be related to attentional capacity (Marx et al., 2009). The relationship between these longer-lasting post-trauma symptoms and EF appears to be more significant than that between EF and acute PTSD symptoms.

Also within the realm of temporal factors, age at which a trauma is experienced may put an individual more at risk, or predict better resilience, in post-trauma outcomes. Neurobiological theories from both human and animal studies suggest that the effect of stressful events may vary by age at the time of the event, partially determined by which brain areas are developing or declining at that age (Lupien, McEwen, Gunnar, & Heim, 2009). Childhood trauma, such as child abuse, has been associated with a decrease in glucocorticoid level, thus affecting how the stress response is enacted throughout the lifespan. In childhood and beyond, though many areas of the brain continue to be at-risk for experiencing the effects of trauma, certain areas may be more susceptible depending on the age of an individual. For example, the hippocampus may be most vulnerable to the effects of extreme stress when a child is very young, while the amygdala may be more at risk during later childhood. Frontal lobe areas, which play the largest role in EF, may be most susceptible to the impact of trauma during adolescence and early adulthood, as this is a time that the PFC is experiencing a rapid increase in volume. Older adults experience a decline in functioning of particular brain areas as a part of normal aging. Stress during this decline may lead to maintenance or manifestation effects, which could lead to an increase in risk for PTSD.

While this literature on the effects of age on stress-related disorders provides important insight into how EF may be influenced throughout the lifespan, highlighting adolescence and early adulthood as a particularly vulnerable time, the authors acknowledge that this relationship

may look different for males and females. Hormonal fluctuations of estrogen in women at different ages may differentially affect how their brain is affected by stressful life events, and therefore more investigation is warranted into how age at which a trauma is experienced provides risk or resilience for post-trauma cognitive difficulties.

Post-Trauma Functioning in Civilians versus Veterans

The present study examined risk and resilience factors related to neurocognitive functioning after trauma in two sub-groups: civilian women and female veterans. While women in each of these groups are likely to be affected by trauma in a different way than men, civilian women and female veterans may also experience different traumatic events, have different emotional responses, and ultimately have different neurocognitive profiles from one another.

In the general populations of female civilians and female veterans, veterans are at an increased risk of poor general health, chronic health conditions, and increased mental health conditions when compared to civilian women (Lehavot et al., 2012). Both deployed and non-deployed female military veterans report poorer mental health than civilian women, with deployed women having the worst mental health outcomes (Hoglund & Schwartz, 2014). Differences in trauma rates, types, and outcomes only add to these differences. The percentage of female veterans who will experience trauma across their lifetime is high, with estimates ranging from 81 to 93 percent (Zinzow et al., 2007). Additionally, the type of trauma they will experience is unique to their position as military members, with 30 to 45 percent experiencing MST and 4 to 31 percent having combat exposure. Female veterans who have experienced MST are at an elevated risk of additional trauma experiences. A 2011 study (Kelly, Skelton, Patel, & Bradley) found that over 95 percent of their sample had at least one trauma in addition to MST,

with sexual abuse as adult civilians representing the most common additional trauma. In a study comparing sexual abuse and assault across the lifetime, rates of adulthood sexual assault were higher in female veterans than in civilians (Schultz, Bell, Naugle, & Polunsky, 2006). Although rates of childhood sexual abuse did not differ, veterans reported longer durations of abuse.

The outcome of traumatic experiences in female veterans is an elevated risk of poor psychological and physical health. With regard to psychological functioning, 27 percent of female veterans will be diagnosed with PTSD within their lifetime, versus 10 to 12 percent of civilian women (Escalona et al., 2004). While sexual assault places women at an increased risk of poor psychological outcomes, MST presents a particular risk. When compared to women who have experienced sexual assault as civilians, those with MST have increased rates of depression, alcohol abuse, and reduced social functioning associated with avoidance symptoms (Suris, Lind, Kashner, & Borman, 2007).

In addition to increased negative psychological outcomes in female veterans, this population is also more likely to evidence poorer physical health. Female veterans experience more chronic health conditions, including chronic pain, than civilians (Lehavot et al., 2012). Within this group, female veterans with PTSD and/or major depressive disorder are most at risk, as they are more likely to report chronic health conditions and healthcare utilization than veterans without these conditions (Dennis et al., 2009). Specific traumatic experiences may place female veterans at a greater risk of physical health complaints than civilian women. MST in female veterans has been associated with pelvic and back pain, headaches, chronic fatigue, and menstrual problems. Furthermore, it may be related to increased cardiovascular risk factors, including obesity and sedentary lifestyles (Suris & Lind, 2008). Intimate partner violence, which may also be more prevalent in female veterans than in civilians (O'Campo et al., 2006), is

associated with increased smoking and heavy drinking, additional major cardiovascular risk factors (Dichter, Cerulli, & Bossarte, 2011).

Of particular interest in the present study is how these two groups of women function neurocognitively following trauma. While studies have examined neuropsychological functioning following trauma in civilian and female veterans separately, few if any studies have examined between-group differences. Thus, a secondary aim of the present study was to evaluate differences in trauma experiences, psychiatric comorbidities, emotional functioning, and neurocognitive functioning in civilian and veteran subgroups.

The Present Study

Executive functioning affects how an individual is able to carry out goal-directed behavior in their daily life. In the context of trauma, EF may help determine how individuals respond to therapy and are able to recover both from cognitive and emotional symptoms resulting from trauma. Thus, the present study sought to build on early work in neuropsychology emphasizing the relationship between trauma, psychological sequelae, and associated neurocognitive functioning (Vasterling & Verfaellie, 2009; Aupperle, Melrose, Stein, & Paulus, 2011) and expand it to study women in both civilian and veteran groups. The primary goal of the study was to examine how risk and resilience factors predict EF in women following trauma. A two-fold approach was taken to address the primary study aim. First, a person-centered approach was used to delineate risk and resilient subtypes, who have experienced similar frequency and severity of trauma, but differ in terms of post-trauma psychological functioning. Next, a variable-centered approach examined factors predicting neuropsychological outcomes following trauma. Although factors affecting psychological outcomes post-trauma have been studied (Suliman,

Stein, & Seedat, 2014), and literature has begun to emerge examining how these factors influence neurocognition (Marx et al., 2009; Navalta et al., 2005), this was the first study to the author's knowledge to specifically investigate contributors to risk and resilience predicting EF outcomes in post-trauma women.

Along with the overarching goal of the study, additional aims involved investigating differences in neuropsychological test performance in women with and without PTSD as well as investigating psychological and neurocognitive functioning following trauma in civilian and veteran samples. The literature supports differences in neuropsychological functioning between those who do and do not have PTSD (Stricker, Keller, Castillo, & Haaland, 2015; Kaye et al., 2014). Additionally, because veterans and civilians are likely to experience different types and frequencies of trauma (Zinzow et al., 2007), they may also vary in terms of psychological functioning, risk and resilience factors, and resulting neuropsychological test performance. The present study sought to study these relationships to add to the literature on trauma types, PTSD, and neuropsychological abilities in women, both civilian and veteran.

Based on the goals of the present research, the following primary study aims were proposed, each coupled with specific hypotheses:

Primary Aim 1. Delineate risk and resilient subtypes within the total post-trauma sample.

> Hypothesis 1a. It is predicted that a 'resilient' subtype of cases within the sample will demonstrate better EF performance and higher self-reported resilience, as well as less psychological disturbance, despite elevated trauma experiences.
>
> Hypothesis 1b. A 'risk' subtype will also emerge that displays elevated trauma experiences along with worse EF performance and greater psychological disturbance.

Primary Aim 2. Determine which variables best predict cognitive functioning, specifically EF, in women who have varying experiences of trauma.

Hypothesis 2a. It is hypothesized that number of trauma experiences, negative affect (neuroticism), and symptoms of GAD, MDD, and PTSD will negatively predict performance on EF tasks.

Hypothesis 2b. Alternatively, pre-morbid cognitive ability and self-reported resilience (CD-RISC) will predict better EF task performance.

Furthermore, the following secondary aims were proposed, along with specific hypotheses:

Secondary Aim 1 (Study Aim 3). Evaluate differences in trauma experiences, emotional functioning, and neuropsychological functioning in trauma-exposed women with and without PTSD.

Hypothesis 3a. It is hypothesized that women who have a formal diagnosis of PTSD will exhibit poorer psychological functioning than those without a diagnosis of the disorder. They will also differ in terms of trauma factors (type, age).

Hypothesis 3b. Those with PTSD will evidence poorer results on the CPT-3, IGT, and DKEFS Color-Word Interference Test than women who have not been diagnosed with the disorder.

Hypothesis 3c. Women who meet CAPS-5 criteria for PTSD, regardless of prior diagnosis, will exhibit poorer psychological functioning and different trauma factors (type, age) than those without presence of the disorder.

Hypothesis 3d. Those who meet CAPS-5 criteria for PTSD will also exhibit poorer performance on the CPT-3, IGT, and DKEFS Color-Word Interference Test than those not meeting current diagnostic standards.

Secondary Aim 2 (Study Aim 4). Investigate differences between civilian and veteran women in terms of trauma exposure, mood symptoms, and neurocognitive functioning.

Hypothesis 4a. It is predicted that female veterans will endorse more traumatic experiences than civilian women, with specific elevations in combat-related trauma and military sexual trauma.

Hypothesis 4b. Furthermore, it is hypothesized that female veterans will have elevated depression, anxiety, neuroticism and PTSD symptoms when compared to civilian women.

Hypothesis 4c. Due to the close relationship between mood functioning and EF, it is also predicted that female veterans will perform worse on the DKEFS color-word interference task, IGT, and CPT-3 than civilian women.

METHOD

Participants

Participants were recruited from the University of North Texas Psychology Clinic, the UNT SONA System, and the Veterans Affairs North Texas Healthcare System (VANTHCS). Inclusion criteria was history of a traumatic experience. A formal diagnosis of PTSD was not a requirement for participation. Participants were excluded for the following: history or presence of a seizure, diagnosed traumatic brain injury (TBI), other neurological disorders, or vision problems that could interfere with testing, including colorblindness. Those with a current substance use disorder were also excluded, as well as those currently experiencing an ongoing traumatic event, such as an abusive relationship.

A total of 66 women completed the present study. Of this total, six participants' data was excluded due to criterion A not being met on the CAPS-5. Thus, the final sample was comprised of 60 participants: 33 from the community ("Civilian" group) and 27 from the VA ("Veteran" group). Demographic information is provided in Table 1. Participants ranged in age from 18 to 55 (M = 29.73, SD = 10.91) and were diverse in terms of ethnicity. Mean education was 14.20 years (SD = 1.58), equivalent to some college or an associate degree. Psychiatric diagnoses, including diagnosis of PTSD, were assessed via self-report in civilians. For veterans, psychiatric diagnoses were recorded from the participants' Computerized Patient Record System (CPRS) records. The most prevalent disorder in the sample was major depressive disorder (50%), followed by PTSD (48.3%), generalized anxiety disorder (25%), and bipolar disorder (16.7%).

Measures

Trauma Experience

Trauma history was assessed via the Trauma History Questionnaire (THQ; Green, 1996) paired with a clinical interview. The THQ provides information on lifetime history of traumatic experiences, including type of experience, age of occurrence, intensity of experience, and frequency of occurrence. The THQ has high test-retest reliability (.91), as well as high content validity, established by the instrument's consistency with the DSM's criterion A stressor category (Hooper, Stockton, Krupnick, & Green, 2011). This questionnaire was paired with a clinical interview (CAPS-5) to elaborate on subjective experience of the traumatic event. For the present study, internal consistency of the THQ was $a = .70$.

PTSD Symptoms

To assess symptoms associated with PTSD, the PTSD Checklist-5 was administered. The PCL-5 gathers information on current post-traumatic symptoms, and is consistent with current DSM-5 criteria for PTSD. The PCL-5 has demonstrated test-retest reliability (.75 - .88), and internal consistency (a =.90) with traumatized samples (Wilkins, Lang, & Norman, 2011). Within the present study, internal consistency of the PCL-5 was $a = .94$.

Along with the PCL-5, the Clinician-Administered PTSD Scale for DSM-5 (CAPS-5; Weathers et al., 2013) standard clinical psychiatric interview was administered. The CAPS-5 was used to elaborate on symptom experience and intensity and conceptualize whether a formal diagnosis of PTSD could be made. The CAPS-5 is a 30-item structured interview, which is considered the gold standard in PTSD assessment. The CAPS-5 aids a trained clinician to make a current or lifetime diagnosis of PTSD, as well as assess PTSD symptoms over the past month.

Self-Reported Resilience

Resilience, broadly defined, was assessed using the Connor-Davidson Resilience Scale (CD-RISC; Connor & Davidson, 2003). The CD-RISC is a self-report measure of personality features comprising resilience in the presence of stressful events. The intra-class correlation of the CD-RISC is high (.87), and its criterion validity when compared to the Brief Resilience Scale is .59 (Windle, Bennett, & Noyes, 2011). Its internal consistency in the present study was α = .93.

Mood and Personality Features

Mood symptoms and personality features related to trauma may provide a link between trauma, resilience, and cognitive functioning. Thus, the Beck Depression Inventory, Second Edition (BDI-2; Beck et al., 1961) and Beck Anxiety Inventory (BAI; Beck, Epstein, Brown, & Steer, 1988) were administered. Both the BDI-2 and BAI are psychometrically-sound self-report measures of mood. Average internal consistency for the BDI-2 is .86 and for the BAI is .92. (Beck, Steer, & Garbin, 1988). Internal consistency in the present study was α = .93 for the BDI-2 and α = .90 for the BAI.

Neuroticism represents a transdiagnostic feature that is also linked with PTSD features. To assess personality, the Eysenck Personality Questionnaire Revised – Abbreviated (EPQR-A; Ogle, Rubin, & Siegler, 2014) was administered. The EPQR-A has satisfactory internal reliability (.76), and provides a valid brief evaluation of multiple personality traits, including extraversion and neuroticism (Lewis & Maltby, 1995). Internal consistency of the EPQR-A in the present study was α = .75.

Crystallized Cognitive Skills

Academic cognitive skills have been linked to executive functioning, and high pre-trauma cognitive skills may predict better long-term neuropsychological outcomes. To assess crystallized cognitive skills, most likely to be consistent with pre-trauma functioning, the Wechsler Test of Adult Reading (WTAR; Wechsler, 2001) was administered. The WTAR has high reliability (.73) compared to other estimates of full scale IQ (Strauss, Sherman, & Spreen, 2006), and can properly gauge crystallized, or pre-morbid, intelligence.

Executive Functioning

Executive Functioning includes the cognitive capacity to focus attention and inhibit responses. EF was assessed using standard neurocognitive tests of attention, inhibition, task switching, and decision-making in the face of reward and punishment. A computerized version of the Continuous Performance Test, Conners CPT-3 (Conners, 2014), assessed sustained attention and inhibition. The CPT-3 requires participants to quickly respond to letter stimuli as they appear on the screen, while inhibiting responses to the letter "x." Primary outcome measures include omission errors, commission errors, and response time, with score dimensions assessing inattentiveness, impulsivity, sustained attention, and vigilance. Median split-half reliability of the CPT-3 was estimated at .92 for norm samples, while test-retest reliability was .67.

The DKEFS Color-Word Interference Test (Delis et al., 2001) assessed inhibition and task-switching. This test replicates the inhibition demands of the traditional Stroop task (Stroop, 1935) with an additional switching subtest. Stroop tasks, including the DKEFS Color-Word Interference Test, are considered a prototypical assessment of inhibition (Miyake et al., 2000). They have been repeatedly validated over the past century and found to be psychometically

sound in both single item presentations and multiple item presentations, such as the one in the DKEFS Test (MacLeod, 1991).

In the Color-Word Interference Task, participants view four separate pages with words: a word reading task, a color naming task, a color-word interference task, and an inhibition/switching task. On the word reading task, participants are asked to quickly read a page of words written in black letters that say “red,” “blue,” and green.” On the color naming task, red, blue, and green blocks are presented to the participant, and they are again instructed to say the names of these colors as quickly as possible. The primary measures of EF on this task are the color-word interference and inhibition/switching portions. On the color-word interference task, participants view words (red, green, blue) written in incongruent ink. They are asked to say the color of the ink, not what the letters say. On the inhibition/switching task, participants alternate between reading the color of the ink and the letters, depending on the presence or absence of a box around the word. Primary outcome measures of the DKEFS Color-Word Interference Task assess simple processing speed for the color naming and word reading tasks, and EF for the latter two tasks.

To assess problem solving in response to reward and punishment, the Iowa Gambling Task was administered (Bechara et al., 1994). The Iowa Gambling Task is a computerized measure of decision making that presents participants with four decks of cards: A’, B’, C’, and D’. Participants are instructed to pick a card from any deck that they wish, while trying to accumulate monetary rewards for advantageous choices. While play money is awarded following some picks, other decks produce punishments, decreasing the amount of money in their bank. Decks A’ and B’ produce high immediate gains with even higher long-term losses, thus they are labeled as disadvantageous decks. Alternatively, decks C’ and D’ result in small immediate gains

which accumulate to higher net gains overall.

Primary outcome measures of the IGT relate to net gains on each segment of the test across five total segments, along with percentage of time choosing from each deck and total money accumulated at the end of the test. Cold EF is associated with rational evaluation of risks and benefits within the test, as well as the ability to hold and retrieve information on these risks and benefits from memory. Alternatively, the IGT assesses hot EF through emotional and affective responses to the risks and rewards associated with the options (Seguin, Arseneault, & Tremblay, 2007). Structural Equational Modeling (SEM) suggests that the IGT measures attentional capacity throughout all 100 trials, with EF emphasized in trials 40 through 100 (Gansler, Jerram, Vannorsdall, and Schretlen, 2011).

Procedure

Recruitment on the UNT campus included flyers placed in the UNT Psychology Clinic waiting room and a blurb describing the study on the SONA website. Potential participants from the Psychology Clinic called the researcher, and were screened for inclusion and exclusion criteria prior to participation. Undergraduate students were able to sign up for the study via the SONA system. At VANTHCS, potential participants were identified through participation in a prior research database study, by physician referral, or though membership in a trauma therapy group. Potential participants were mailed letters explaining the study, which were followed up with phone calls subsequent to a two-week period.

Once a participant was identified via flyer, letter, or phone call, an in-person informed consent meeting was established. During the informed consent meeting, the purpose, procedures, and potential risks and benefits of the study were reviewed with participants. Upon completion

of the informed consent process, participants began testing by completing the hand-written questionnaires: the THQ, BDI-2, BAI, EPQR-A, PCL-5, and CD-RISC. Following completion of these questionnaires, a CAPS-5 structured interview was administered to garner additional information about the participant's trauma history and probability of meeting diagnosis for PTSD. Neuropsychological testing then took place in the following order: WTAR, DKEFS Color-Word Interference Task, CPT-3, and IGT.

Following testing, all participants received referrals to sources of psychological support. For the Civilian sample, these referrals included: The National Sexual Assault Online Hotline, the Safe Online Helpline, the National Suicide Prevention Lifeline, and the University of North Texas Psychology Clinic. Veterans were referred to the Veterans Crisis Line. While SONA participants received course credit for the study, other participants were not compensated.

RESULTS

Power Analysis

Recent research with female veterans matched on IQ and demographics (Stricker et al., 2015) found medium to large effects sizes for differentiating those with (n = 56) versus without PTSD (n = 53) in terms of neurocognitive functioning (d's = .57 – .88). Additional preliminary analyses, based on a non-clinical general population sample (Sullivan et al., 2016), found small to medium effect sizes for the variable-centered results (d's = .26 – .56), and very large effect sizes for the person-centered results (d's = 2.7 – 3.9). Thus, it is reasonable to expect, at minimum, moderate effects sizes for the proposed research using a clinic-based and veteran-based sample. Using linear multiple regression (random model, exact test, one-tail) with 6 predictors to estimate required sample size for alpha at .05 and power at .95, a total sample of N = 60 was required. The present sample met this power analysis aim.

Missing and Incomplete Data

Six participants were excluded from the present study for not meeting criterion A of the DSM-5 PTSD criteria, thus indicating they did not experience the diagnostic definition of a traumatic event. Furthermore, eight participants (seven civilians and one veteran) had incomplete data on the IGT or CPT-3 due to computer errors. Because psychological questionnaire data was present for each of these participants, and other measures of neuropsychological functioning were also available, pair-wise exclusion was used rather than list-wise deletion. The eight individuals with incomplete data did not differ from the rest of the sample in terms of age, education, or premorbid cognitive ability. Furthermore, their scores on psychological measures (BDI, BAI, EPQR-A, THQ, PCL-5, CD-RISC, and CAPS) were not significantly different from

the remainder of the sample, nor were their scores on neuropsychological measures.

Descriptive Values

Table 1 describes demographics for the total sample, including age, education, and prior history of psychiatric disorders. Table 2 lists scores on psychological measures, while Table 3 describes trends in primary or "criterion A" trauma. Mean scores on neuropsychological measures for the total sample as well as subgroups can be found in Table 4. Regarding PTSD diagnosis, 47 individuals' (78.33%) current PTSD diagnoses as assessed by the CAPS-5 were consistent with prior diagnosis or lack of diagnosis. In other words, if these individuals had been diagnosed with PTSD they continued to meet criteria and if they did not have a prior diagnosis they continued to not meet criteria for the disorder. Three participants (5%) had a history of PTSD but did not currently meet criteria, while ten (16.67%) currently met CAPS-5 criteria without having a prior diagnosis.

Analysis of Hypotheses

Hypothesis 1

> Hypothesis 1a: It is predicted that a 'resilient' subtype of cases within the sample will demonstrate better EF performance and higher self-reported resilience, as well as less psychological disturbance, despite elevated trauma experiences.
>
> Hypothesis 1b: A 'risk' subtype will also emerge that displays elevated trauma experiences along with worse EF performance and greater psychological disturbance.

Hierarchical cluster analysis, based on the variables CAPS-5 severity score, BAI total score, BDI-2 total score, PCL-5 total score, THQ score, and EPQR-A total score, revealed three distinct groups: Group 1 (moderate symptoms), Group 2 (severe symptoms), and Group 3 (mild symptoms). A three-cluster solution was chosen as favorable over a four-cluster solution, due to

a four-cluster solution creating very small individual clusters resulting in low power in subsequent analyses. Further, a three-cluster solution was chosen over a two-cluster solution because the dichotomous nature of the two-cluster solution is not ideal to explore individual differences across a spectrum as this study aimed to do. ANOVA analyses were performed to examine differences in emotional and neuropsychological functioning between clusters. T-tests were then performed to examine bivariate group differences. Because psychological and trauma measures were used as criteria to separate groups, analyses were used more as confirmatory descriptions of groups rather than to represent primary unique findings. Group means and ANOVA results are displayed in Table 5. They are displayed graphically in Figure 1. Tables 6 through 8 show results from t-tests.

ANOVA revealed a difference between groups in terms of number of traumatic experiences endorsed ($F(2,57) = 7.34$, $p < .01$, $h^2=.21$). While the mild symptoms group had the lowest frequency of traumatic events ($M = 4.92$, $SD = 2.39$), the number of traumas did not differ between the moderate symptoms and severe symptoms group. In terms of emotional functioning, the mild symptoms group evidenced minimal depression, anxiety, PTSD symptoms, and neuroticism, each significantly lower than both the moderate symptoms and severe symptoms groups. Additionally, self-reported resilience was higher than the moderate symptoms and severe symptoms groups.

Along with elevated number of traumatic events ($M = 8.05$, $SD = 4.05$), the moderate symptoms group had a moderate elevation of depressive symptoms and anxiety symptoms, each of which was higher than that seen in the mild symptoms group and lower than the severe symptoms group. Their PTSD symptom elevation, as measured by the CAPS-5 and the PCL-5, as well as their neuroticism score also fell between the scores of the other two groups. The

moderate symptoms group's average self-reported resilience was between those of the severe symptoms group and mild symptoms group, but was only significantly different from the mild symptoms group.

The severe symptoms group was marked by elevated traumatic experiences (M = 9.66, SD = 3.71), along with severe symptoms of depression, anxiety, and PTSD, each greater than the other two groups. Self-reported resilience was lower than the mild symptoms group.

The moderate symptoms and severe symptoms clusters emerged as groups of interest. Because the two groups did not differ in terms of number of traumatic experiences on the THQ, but differed significantly on measures of emotional functioning, they were considered to be resilient and risk groups, respectively. Further exploration revealed no differences in percentage of civilians and veterans between these groups (c^2 (1) = 4.27, p = .075, r = .27). Additionally, a Sample X Cluster MANOVA suggested that veteran or civilian status did not influence cluster differences on psychological measures (Table 9). Thus, the groups could be examined in terms of risk and resilience for psychological and neuropsychological effects of trauma without separating based on veteran status. Results from further analyses on these groups can be found under Hypothesis 2a and Hypothesis 2b.

Differences in scores on neuropsychological tests were first examined via ANOVA across the three clusters, followed by t-tests to parse out differences between individual groups. In terms of premorbid intellectual functioning, the moderate symptoms group (M = 104.84, SD = 11.21) had a score significantly higher than the mild symptoms (M = 90.25, SD = 11.55) group (t(29) = 3.49, p < .01, d = 1.28). On neuropsychological measures, the three groups differed in terms of DKEFS Color Naming score (F(2,57) = 3.28, p < .05, h^2=.10), with the moderate symptoms group (M = 11.00, SD = 2.13) outperforming the severe symptoms group (M = 8.83,

SD = 3.73) (*t*(45.35) = 2.56, *p* < .05, *d* = .71).

Hypothesis 2

> Hypothesis 2a: It is hypothesized that number of trauma experiences, negative affect (neuroticism), and symptoms of GAD, MDD, and PTSD will negatively predict performance on EF tasks.

Initial ANOVA analyses explored whether those with different types of primary trauma or different age of primary trauma differed in terms of neuropsychological function. Results revealed no differences between groups based on these trauma factors. Correlation analyses were then performed to explore the relationship of other trauma factors, personality factors, and mood symptoms with performance on neuropsychological measures. Tables 10 to 12 display results from these correlations for the DKEFS Color Word Interference Task, IGT, and CPT-3, respectively. While mood and trauma measures did not correlate with performance on the DKEFS or CPT-3, BAI total score was significantly negatively correlated with Net 5 score on the IGT (*r* = -.317, *p* < .05). Item analysis revealed that item 2 (feeling hot; *r* = -.295, *p* < .05) and item 6 (dizziness; *p* = -.267, *p* < .05) had significant negative correlations with IGT Net 5 score, with item 4 (inability to relax) approaching significance (*p* = -.251, *p* = .060).

Regression analyses were then performed to further explore the predictive nature of risk and resilience factors in relation to neurocognitive functioning. Due to the high multicollinearity between PCL-5 and CAPS-5 Severity variables (VIF > 5), separate regression models were run incorporating the CAPS-5 severity score and PCL-5 total score. A series of stepwise regressions were performed using risk factor predictors of BDI-2 score, BAI score, THQ score, EPQRA score and either PCL-5 or CAPS-5 severity score, and dependent variables of DKEFS Color Naming, DKEFS Color-Word Interference, DKEFS Interference/Switching, IGT Net 5, and

CPT-3 Comissions. While risk factors did not predict performance across DKEFS and CPT-3 measures, BAI score (R^2 = .10; F(1,55) =6.14, p < .05; b = -.32) best predicted performance on IGT Net 5.

To further explore the contribution of psychological risk factors to neuropsychological test performance, two path analyses were run. The first model, displayed as Figure 2, used CAPS-5 severity score as a predictor while the second model, displayed as Figure 3, analyzed PCL-5 score. Additional predictor risk variables in each model were BDI-2 score, BAI score, and THQ score. Because EPQR-A did not add a significant contribution to the model, it was omitted from path analyses. A composite, summed variable was used for DKEFS Color-Word Interference performance. Because of the heavy contribution of processing speed to each subtest of this task, the summed variable can best be described as an attention/processing speed measure with some EF demand. IGT performance was represented by Net 5 score, which had the greatest relationship with predictor variables in initial regression analyses. Because CPT-3 variables did not relate to psychological questionnaires, it was not included in the path models.

In the first path analysis, BDI-2 score negatively predicted DKEFS Color-Word Interference task performance, while BAI score and number of traumatic experiences, represented by THQ score, negatively predicted IGT Net 5 score. The second path model replicated these results, though PCL-5 score positively predicted IGT Net 5 task performance.

Total sample regression analyses and path analyses were followed with regressions analyzing the relationship between risk factors and neuropsychological functioning only in those individuals in the moderate symptoms and severe symptoms clusters. Due to the smaller sample size of this subgroup, and the resultant lower power associated with path analyses, only linear regressions were performed. Because these two clusters evidenced a high number of traumatic

experiences and elevated symptoms, regressions sought to determine how risk factors predicted neurocognitive performance in this subsample that had been particularly affected by trauma. Results from stepwise regressions, presented in Table 13, revealed mood symptoms predicted poorer performance across DKEFS and IGT measures. BDI-2 score best predicted performance on the DKEFS Color Naming, Color-Word Interference, and Interference/Switching tasks, while BAI score was the best predictor of IGT Net 5 performance. Notably, when CAPS-5 severity was entered into the model in place of PCL-5 score, it became the most significant predictor of DKEFS Interference/Switching score ($R^2 = .07$ $F(1,46) = 4.77$, $p < .05$; b = -.31).

Additional Subgroup Analyses for Hypothesis 2a

In addition to examining the total sample and a subset of clusters, subgroups differing in veteran/civilian status and PTSD diagnosis were assessed. Due to the smaller size of these subsamples, correlations were used in lieu of regression. Tables 14 to 16 display results from correlation analyses evaluating the relationship between risk factors and neuropsychological performance for veterans. Tables 17 to 19 display results from the civilian group. In the veteran group, PCL-5 score ($r = .447$, $p < .05$), BDI-2 score ($r = .523$, $p < .01$), BAI score ($r = .399$, $p < .05$), and EPQR-A score ($r = .462$, $p < .05$) each demonstrated positive correlations with CPT-3 response time, though an outlier may have influenced this relationship. In the civilian group, CAPS-5 severity score was positively correlated with commission errors ($r = .436$, $p < .05$) and negatively correlated with response time ($r = -.537$, $p < .01$) on the CPT-3. BAI score was negatively correlated with response time on the CPT-3 ($r = -.398$, $p < .05$).

Further analyses assessed relationships between mood and trauma reports with neuropsychological functioning in those with and without a history of diagnosed PTSD (PTSD

history and no PTSD history groups). Tables 20 to 22 and Tables 23 to 25 display results from these analyses. In the PTSD history group, BDI-2 score was negatively correlated with Color Naming (r = -.377, p < .05) score on the DKEFS Color Word Interference Test, while CAPS-5 severity was negatively correlated with the Color Word Interference score (r = -.433, p < .05). The relationship between BDI-2 score and Word Reading approached significance (r = -.367, p = .050). For the IGT, the PTSD history group's PCL-5 score was positively correlated with Net 1 score (r = .391, p < .05), while the BAI was negatively correlated with Net 5 score (r = -.404, p < .05). CPT Response time was correlated with scores on the PCL-5 (r = .413, p < .05), BDI-2 (r = .401, p < .05), and EPQR-A (r = .485, p < .01).

Alternatively, in the no PTSD history group, CAPS-5 severity score correlated with color naming scaled score (r = .373, p < .05). CAPS-5 severity (r = -.402, p < .05), PCL-5 score (-.428, p < .05), THQ score (r = -.365, p < .05), and BAI score (r = -.559, p < .01) each evidenced negative correlations with Net 3 score on the IGT. On the CPT-3, CAPS-5 severity (r = .438, p < .05) and BAI score (r = .409, p < .05) each correlated with perseverations, though outliers may have influenced this relationship.

Hypothesis 2b: Alternatively, pre-morbid cognitive ability and self-reported resilience (CD-RISC) will predict better EF task performance.

Further analyses explored the relationship between resilience factors (premorbid cognitive ability and self-reported resilience) and EF task performance. Correlations revealed that scaled score on the WTAR, a measure of premorbid intelligence, was significantly correlated with better performance on the DKEFS Word Reading (r = .334, p < .01), Color Word Interference (r = .309, p < .05), and Interference/Switching tasks (r = .274, p < .05), as well as

the Net 5 score on the IGT ($r = .575$, $p < .05$). No relationship existed between the CD-RISC and neuropsychological task performance.

Regression models then assessed how the resiliency factor of premorbid intelligence predicted neuropsychological test performance within the total sample and within the high-trauma clusters (moderate symptoms and severe symptoms). For the neuropsychological measures that did not have a significant predictor in prior risk factor analyses (Hypothesis 2a), simple linear regressions assessed the predictive value of WTAR scaled scores. For the IGT Net 5, which had BAI score as a significant predictor, a hierarchical multiple regression was employed to first explore the relationship between premorbid intelligence and IGT performance and then to determine whether the BAI has predictive value beyond that of the WTAR. Because CD-RISC did not relate to neuropsychological outcomes in correlation analyses, it was not incorporated into regression models. As seen in Table 26, premorbid intellectual functioning predicted performance on the DKEFS Color-Word Interference task, DKEFS Interference/Switching Task, and IGT Net 5. Furthermore, BAI score continued to predict IGT Net 5 score beyond the predictive value of WTAR score.

Path analyses revealed similar results with regard to premorbid intelligence. WTAR score positively predicted both IGT and DKEFS performance. Contrary to hypotheses, CD-RISC was negatively associated with IGT Net 5 score, though effect size was low and it was a poor predictor when compared to other risk and resilience factors.

Further hierarchical regression was performed on data from moderate and severe symptoms clusters, eliminating the mild symptoms cluster with minimal traumatic experiences and subsequent symptoms. The first step of the regressions involved an evaluation of WTAR's contribution to neuropsychological performance, while the second step examined the effect of

the most salient risk factor beyond that of premorbid intelligence. Because there were no significant predictors of CPT-3 commission errors, a simple linear regression was run for this variable. Results are displayed in Table 27. In this sub-group of the sample, WTAR score predicted performance on the DKEFS Color Word Interference Task. Adding prominent risk factors to the model explained a significantly higher percentage of the variance in performance for DKEFS Color Naming ($F(2,45) = 5.19$, $p = .009$), DKEFS Color-Word Interference ($F(2,45) = 5.10$, $p = .010$), DKEFS Interference/Switching ($F(2,45) = 3.54$, $p = .037$), and IGT Net 5 ($F(2,42) = 7.34$, $p = .002$).

Additional Subgroup Analyses for Hypothesis 2b

Correlation analyses by sub-group (civilian/veteran) were then performed. In the civilian group, WTAR scaled score was positively correlated with performance on the DKEFS Color Naming ($r = .377$, $p < .05$), Word Reading ($r = .473$, $p < .01$), Color Word Interference ($r = .408$, $p < .05$), and Interference/Switching ($r = .504$, $p < .01$) tasks. In the veteran group, WTAR scaled score was positively correlated with total money won on the IGT ($r = .456$, $p < .05$).

Additional analyses assessed PTSD History and No PTSD History groups. In the PTSD History group, WTAR scaled score was positively correlated with both DKEFS Word Reading ($r = .411$, $p < .05$) and Color Word Interference ($r = .481$, $p < .01$). CD-RISC score was negatively correlated with CPT-3 Perseverations ($r = -.397$, $p < .05$) and WTAR scaled score was negatively correlated with CPT-3 Omissions ($r = -.476$, $p < .05$). No correlations existed between resilience factors and IGT scores. In the No PTSD History group, WTAR scaled score positively correlated with IGT Net 5 score ($r = .452$, $p < .05$). No other correlations were present between resilience factors and neuropsychological test performance in this group.

Hypothesis 3

> Hypothesis 3a: It is hypothesized that women who have a formal diagnosis of PTSD will exhibit poorer psychological functioning than those without a diagnosis of the disorder. They will also differ in terms of trauma factors (type, age)

In total, 29 participants had a prior diagnosis of PTSD, while 31 did not. Table 28 compares those with PTSD versus those without a diagnosis of the disorder. The PTSD history group was older than the no PTSD history group ($t(49.95) = 4.98$, $p < .001$, $d = 1.29$), and had more members with a diagnosis of bipolar disorder ($c^2(1) = 4.82$, $p < .05$, r = .28). They were more likely to be veterans than civilians ($c^2(1) = 17.04$, $p < .001$, r = .53). No differences in education, ethnicity, or other psychiatric diagnoses was present. Table 29 compares the groups in terms of scores on psychological measures. The PTSD history group had higher scores on the BDI-2, BAI, EPQRA, THQ, PCL-5 and CAPS-5 than the no PTSD history group. They did not differ on resilience scores as assessed by the CD-RISC.

Regarding trauma factors, a chi square test revealed differences between PTSD history and no PTSD history groups in terms of primary (Criterion A) traumatic event ($c^2(6) = 13.45$, $p < .05$, r = -.31). All six participants with combat as a primary traumatic event also had a history of PTSD. None of the participants with a primary traumatic event of witnessing serious injury/abuse or learning about a traumatic death had previously been diagnosed with PTSD. Furthermore, groups differed in terms of age in which the primary traumatic event was experienced ($c^2(2) = 8.68$, $p < .05$, r = .38). The majority of participants in the PTSD history group had experienced their criterion A event while in adulthood (68.97%). Only 32.26% of participants in the no PTSD history group had experienced their primary traumatic event while in adulthood, versus 19.35% in adolescence and 48.39% in childhood.

> Hypothesis 3b: Those with PTSD will evidence poorer results on the CPT-3, IGT, and DKEFS Color-Word Interference Test than women who have not been diagnosed with the disorder.

With regard to neuropsychological functioning, no differences were present in premorbid intellectual functioning between the groups. Furthermore, they did not differ across measures on the CPT-3 or IGT. While no significant differences were noted on the DKEFS Color-Word Interference Test, scaled score on the color word interference task approached significance (PTSD history $M = 8.72$, no PTSD history $M = 10.19$; $t(58) = 1.78$, $p = .080$, $d = .46$).

> Hypothesis 3c: Women who meet CAPS-5 criteria for PTSD, regardless of prior diagnosis, will exhibit poorer psychological functioning and different trauma factors (type, age) than those without presence of the disorder

Thirty-six participants (60%) met CAPS-5 criteria for PTSD, based on Criterion A stressor combined with presence, severity, and duration of symptoms across diagnostic clusters. Groups with a diagnosis of PTSD based on CAPS-5 criteria (PTSD present) and without a diagnosis of PTSD based on CAPS-5 criteria (PTSD absent) were compared in terms of demographic features, trauma histories, psychological functioning, and neurocognitive functioning. Table 30 displays demographic information and prior psychiatric diagnoses. Of those in the PTSD present group, 23 were veterans (63.9%) and 13 were civilians (36.1%), which represented a significant difference ($c^2(1) = 12.97$, $p < .001$, r = .47). The PTSD present group was older than the PTSD absent group, but the two groups did not differ in terms of education. The majority of participants (72.2%) in the PTSD present group had a prior diagnosis of the disorder. The PTSD present group also had a higher proportion of individuals with a diagnosis of bipolar disorder.

Table 31 describes performance on psychological measures, where the PTSD present group evidenced more elevated scores on the BDI-2, BAI, EPQR-A, THQ, PCL-5, and CAPS-5

severity. No differences were present on the CD-RISC. With regard to category of the Criterion A stressor, those in the PTSD present group were more likely to have experienced a recent trauma (c^2 (2) = 11.39, $p < .01$, $r = .42$). In total, 63.89% endorsed a primary traumatic experience in adulthood versus 29.17 in the PTSD absent group. Those in the PTSD absent group were more likely to have experienced a remote childhood trauma, with 58.33% indicating their primary traumatic event occurred before the age of 13 versus 16.67% in the PTSD present group. The two groups also differed in terms of type of Criterion A stressor (c^2 (6) = 15.97, $p < .05$, $r = -.46$). The majority of participants in the PTSD present group had experienced sexual violence as a Criterion A stressor (72.22%), followed by combat (13.89%), and serious injury/abuse (8.33%).

> Hypothesis 3d: Those who meet CAPS-5 criteria for PTSD will also exhibit poorer performance on the CPT-3, IGT, and DKEFS Color-Word Interference Test than those not meeting current diagnostic standards.

PTSD present and PTSD absent groups did not differ in terms of premorbid intellectual functioning. No between-group differences were present on the IGT, CPT-3, or DKEFS Color-Word Interference Test.

> Hypothesis 4a: It is predicted that female veterans will endorse more traumatic experiences than civilian women, with specific elevations in combat-related trauma and military sexual trauma.

Table 32 displays demographic information for the civilian and veteran groups. Groups differed in terms of age and education, with the veteran group an average 16.37 years older than the civilian sample and having an average of over 1 year more of education.

Table 33 explores trauma categories by group. Overall, civilian and veteran groups differed in terms of type of primary trauma experienced (c^2 (6) = 15.06, $p < .05$, $r = -.34$). As would be expected, veterans were more likely to endorse combat and MST as their primary

stressors than were civilians due to the military-nature of these traumas, while civilians were more likely to have a primary trauma of witnessing death, witnessing serious injury/abuse, or learning about a traumatic death of a loved one, each outside a combat setting.

While the CAPS-5 assessed primary traumatic events, also known as Criterion A stressors, the THQ provided information on each traumatic event experienced across the participant's lifetime. The civilian sample reported an average of 6.52 traumatic events (*SD* = 3.62), while the veteran sample endorsed an average of 10.26 events (*SD* = 3.44). The veteran sample were more likely to report accidents, natural disasters, exposure to chemicals, situations where they feared they may be killed or seriously injured, situations where they saw dead bodies, combat, rape, and other forms of sexual assault.

> Hypothesis 4b: It is hypothesized that female veterans will have elevated depression, anxiety, and PTSD symptoms when compared to civilian women.

The civilian and veteran groups were assessed in terms of history of psychiatric diagnosis as well as presence of symptoms of depression, anxiety, neuroticism, and PTSD. While the groups did not differ in number of participants diagnosed with MDD, the veteran group had more participants diagnosed with bipolar disorder, GAD, and PTSD.

Table 34 contains mean scores on symptom inventories. Compared to the civilian group, the veteran group had more elevated BDI-2 scores, BAI scores, and PTSD symptoms as assessed by the PCL-5 total score and CAPS-5 severity score. Groups did not differ in terms of neuroticism.

> Hypothesis 4c: Due to the close relationship between mood functioning and EF, it is also predicted that female veterans will perform worse on the DKEFS color-word interference task, IGT, and CPT-3 than civilian women.

The veteran and civilian groups did not differ on a measure of premorbid intelligence ($t(58) = 1.98$, $p = .053$, $d = .51$). While the two groups did not differ in terms of completion time

on DKEFS Color-Word Interference tasks, the veteran group made more errors on an interference/switching task than the civilian group ($t(39.69) = -2.61$, $p < .05$, $d = .68$). On the IGT, the veteran group had a lower net score in the first section of the test than the civilian group ($t(3893) = -2.70$, $p < .05$, $d = .73$), indicating this group took longer to establish good and bad decks. No differences were noted on the CPT-3.

DISCUSSION

The present study examined the relationship between mood, personality, cognitive factors, and trauma factors predicting women's neuropsychological outcomes following traumatic experiences. Neuropsychological functioning following trauma, and specifically post-trauma EF, has become more prevalently researched over the past decade (Vasterling & Verfaellie, 2009; Caparos & Blanchette, 2014, Fogleman et. al, 2017; Aupperle et. al, 2011). In recent years, women's post-trauma neuropsychological functioning has amassed attention within the field, as women are more at risk for increased prevalence of mood symptoms, PTSD diagnoses, and perhaps also poorer cognitive functioning subsequent to trauma (Navalta et al., 2006; Twamley et al., 2009). Though early studies have examined trauma, PTSD, and EF in civilian (Kaye et al., 2014) and veteran (Stricker et. al, 2015) women, no study to date has analyzed what factors predict intragroup differences in neurocognitive outcomes in women affected by trauma.

The primary inclusion criterion in this study was past experience of a traumatic event, and participation did not require a formal diagnosis of PTSD. This approach was unique, as many prior studies focus only on those with diagnoses of the disorder (Stricker et. al, 2015; Fogleman et. al, 2017). Additionally, the present sample was comprised of veteran and civilian women, which allowed for assessment of those with a wide range of traumatic experiences, presenting symptoms, and neuropsychological capacities. Primary hypotheses were that anxiety symptoms, depression symptoms, PTSD symptoms, increased trauma exposure, lower premorbid intellectual functioning, and poorer perceived resilience would predict worse performance on EF measures in a mixed sample of veteran and civilian women. Furthermore, risk and resilient subtypes would emerge differing in mood, trauma, and neurocognitive factors. Secondary

analyses examined differences in psychological functioning, traumatic experiences, and neuropsychological outcomes between groups of civilians and veterans, those with and without PTSD diagnoses, and those with and without clinically elevated PTSD symptoms. Veterans and those with history and presence of PTSD were predicted to have poorer functioning across all measures. The following findings emerged.

Primary Findings

Individuals Similar in Level of Trauma may Differ in Psychological Outcomes as well as Neuropsychological Test Performance

Cluster analysis based upon psychological measures divided the total sample into three distinct groups. Based on selection criteria for these groups, one had low trauma exposure as well as low psychological distress. The other two groups had similar exposure to trauma, similar percentages of veterans and civilians, and a comparable level of premorbid intelligence; however, they differed in psychological symptom elevation. While the two groups with high trauma exposure did not differ in self-reported resilience, the moderate symptoms group's score trended lower than the severe symptoms group. Because of their differences in psychological symptom elevation and trends toward differences in resilience, these two groups were considered to represent resilient and risk subtypes within the sample.

The separation of groups similar in trauma exposure but differing in post-trauma psychological functioning has its roots in literature on post-trauma subtypes. Prior studies have used latent class analysis (LCA) to identified highly traumatized groups who differ in PTSD symptom severity (Sullivan et al., 2017), oftentimes resulting in three-class trauma typology solutions (Hagan et al., 2015; McFeeters, Boyda, & O'Neill, 2015). Unlike the present study, though, many of these prior studies have used LCA with the goal of differentiating PTSD from

other disorders, such as borderline personality disorder (Cloitre et al., 2014), or to observe differences in type of trauma exposure within a subset of individuals (Armour et al., 2014; Adams et al., 2016). The present study added an additional dimension to prior classification approaches by separating individuals similar in trauma exposure and different in post-trauma psychological functioning. In this way, differences in neuropsychological functioning between groups divergent in psychological symptoms, but each still with significant distress, could be evaluated.

This led to the most substantial finding of cluster differences: risk and resilience groups differed in processing speed on the DKEFS Color Naming task. This finding supports the notion of an underlying difference in post-trauma cognitive functioning between groups who also differ in post-trauma psychological functioning. While the two groups did not evidence discrepancies on EF tasks, it is likely that subcortical processes, such as attention and processing speed, may be susceptible to subtle but sustained changes in psychological functioning. This result is consistent with prior findings of processing speed deficits across EF tasks for women with PTSD resulting from IPV (Twamley et al., 2009). Furthermore, it is supported by recent imaging studies that have revealed subcortical volume reduction in those exposed to trauma, with a negative correlation between CAPS-5 clinical score and volume (O'Doherty et al., 2017). Most importantly, this difference highlights the need for person-centered research approaches to inform treatment for those with trauma exposure, as those with the greatest psychological distress may warrant the most need for neuropsychological assessment and intervention.

Pre-Trauma Cognitive Ability may Act as a Resilience Factor, while Elevated Psychological Symptoms may be a Risk Factor for Poorer Neuropsychological Functioning

Correlation, regression, and path analyses were employed to explore the primary

hypothesis that risk and resilience factors would be associated with performance on EF tasks. Premorbid intelligence, as assessed by the WTAR, most consistently demonstrated positive correlations with EF task performance and predicted neuropsychological scores in regression and path models. This was true in the full sample and across correlations exploring the civilian and veteran groups, as well as groups with and without a history of PTSD. Furthermore, it held true across performance on tasks assessing attention, problem solving in the presence of reward and punishment, inhibition, and switching.

Prior studies have found that higher pre-trauma cognitive functioning may predict better functioning after trauma, both in terms of emotional outcomes and neurocognitive outcomes (Gale et. al, 2008; Marx et. al, 2009). This idea is also supported within the concept of cognitive reserve, which holds that those with protective factors, such as higher pre-morbid cognitive functioning and educational exposure, may cope better with exposure to brain changes (Stern, 2009). This concept has been previously studied in OEF/OIF veterans who have been exposed to blasts while deployed. Results suggested that those with higher pre-trauma cognitive functioning, as assessed by the WTAR, performed better on two measures of EF: Mazes and Categories subtests (Moncrief, 2015). Additionally, a study of intellectual functioning's relationship with post-trauma neuropsychological test performance examined Vietnam veterans (Vasterling et al., 2002), with a finding that higher premorbid intelligence is related to better neurocognitive outcomes. Notably, in Vasterling's 2002 study, as in the current study, psychological symptom severity continued to be associated with post-trauma neurocognitive functioning when intellectual functioning was controlled for. Thus, the present results support prior established literature, and emphasize the effects that pre-trauma functioning can have on post-trauma EF.

While WTAR score was associated with neuropsychological measure performance across

the total sample and most subgroups, it did not predict neuropsychological scores within the moderate and severe symptom clusters. This is perhaps due to the fact that little variation existed within and between these groups in terms of WTAR scaled score. Future studies may seek to examine differences in neuropsychological test performance in those with elevated PTSD symptoms as a function of a greater range of premorbid intelligence.

Overall, contrary to hypotheses, self-reported resilience was not a good predictor of neuropsychological functioning. While it negatively predicted IGT Net 5 performance in path analyses, this effect size was low. Because CD-RISC score evidenced significant negative correlations with other predictors in path analyses, including PCL-5 score, EPQRA score, and both BDI-2 and BAI scores, it is possible that a suppression effect artificially reduced significance between this resilience measure and neuropsychological performance. However, due to limited significant results from correlations, it is more likely that the CD-RISC was not an adequate measure of resilience to neuropsychological effects of trauma in this sample. Since the CD-RISC was developed to assess general psychological resilience and not any specific factors that may predict better neuropsychological functioning following trauma (Conner & Davidson, 2003), it does not ask questions related to pre-trauma cognitive ability, academic factors, complexity of cognitive demands at work, or other cognitive factors that may account for differences in neuropsychological outcomes. The limitations and future directions section explores this finding and possible solutions for future research in more detail.

In terms of risk factors, elevated anxiety was associated with poorer performance on the IGT, a measure of hot EF. Specifically, self-reported anxiety on the BAI evidenced a negative correlation with Net 5 score on the IGT and, when combined with WTAR score, predicted 14% more of the variance in Net 5 score than premorbid intelligence alone. Net 5 is a measure of how

well an individual is able to choose beneficial decks to make a profit after 80 initial trials with reward and punishment feedback. Therefore, it is both a measure of sustained attention and affective EF. Results suggest that in those who have experienced trauma, elevated anxiety may be associated with poorer problem solving in the face of affective demands. While the relationship between anxiety level and Net 5 score was more prevalent in those with PTSD, those without a diagnosis of the disorder evidenced a significant relationship between BAI score and Net 3 IGT score. Thus, regardless of diagnosis of PTSD, those with a trauma history and elevated anxiety appeared to have increased difficulty with problem solving in the face of affective activation.

Prior research identifies a correlation between elevated anxiety symptoms and reduced EF and highlights the notion that this relationship may be bidirectional. Research on the relationship between psychopathology and neuropsychological functioning suggests that poorer EF may lead to increased rumination (Zetsche et al., 2012; Demeyer et al., 2012), worry (Snyder et al., 2014) and ineffective use of emotional regulation strategies (Andreotti et al., 2013), thus exacerbating anxiety symptoms. This effect may be especially important to note in those who have a history of trauma, as post-trauma psychological functioning may be mediated by ability to manage emotions and reduce rumination.

In the alternate direction, anxiety may lead to attentional bias toward threat (Heeren et al., 2013), as demonstrated on a hot EF task closely related to that presented within the IGT. Reduction in the ability to sort affective information presented in threatening or punishment situations, as was the case with the IGT, may again be of particular concern to those with trauma histories coupled with elevated anxiety (Bar-Haim et al., 2007). Therefore, the relationship observed in the present study between anxiety and affective problem solving has support within

prior literature, implications for future research and treatment modalities, and real-word implications. Treatment providers should be aware of this relationship, as it could affect PTSD treatments with high cognitive demands. Furthermore, researchers, treatment providers, and individuals who have experienced trauma should be aware of the effect elevated anxiety in conjunction with traumatic experiences may have on affective problem solving in daily functioning.

While anxiety represented the greatest risk factor for decreased performance on the IGT in the total sample and those with the most elevated symptom presentations (moderate and severe symptoms clusters), path analyses accounting for the total sample and regression models examining the moderate and severe symptoms clusters suggested depression may also affect neuropsychological performance. In path analyses examining the total sample, BDI-2 score negatively predicted DKEFS Color-Word Interference subtest performance as a whole, with a medium effect size. In moderate and severe symptom clusters, BDI-2 score represented the best predictor of performance across three DKEFS measures, accounting for 7 to 12% of performance variance across measures. When looking at each Color-Word Interference task score individually, it appears that BDI-2 had the greatest predictive value for the color naming subtest, a test of simple attention and processing speed. Its predictive value was reduced for each of the measures that incorporated EF. This result again echoes deficits in subcortical functioning associated with high psychological distress post-trauma, and identifies depression as a significant contributor to these changes.

Depression rates are elevated in women who have experienced trauma (Golding, 1999), and a high comorbidity exists between PTSD and depression in women (O'Campo et al., 2006). Furthermore, there is ample literature to support the notion that depression is associated with

reduced attention and processing speed (Tsourtos, Thompson, & Stough, 2002; Cohen et al., 2001; Hammar, Lund, & Hugdahl, 2003; Lampe, Sitskoom, & Heeren, 2004). It is reasonable to expect, then, that women who have experienced trauma and are experiencing elevated depression symptoms may be at risk for reduced proficiency in these domains. Therefore, as literature begins to describe the relationship between women's trauma experiences and resulting neurocognitive functioning, anxiety and depression may emerge as specific risk factors predicting poorer functioning.

Notably, when anxiety and depression effects were accounted for in path analyses, PCL-5 score positively predicted IGT Net 5 performance, instead of negatively predicting it as would be expected. This suggests that comorbid symptoms and not pure PTSD symptoms accounted for associations between trauma and executive functioning. This finding supports the notion that trauma affects psychological functioning in a complex way and that emotional dysregulation also displays a complicated relationship with neuropsychological task performance dependent on multiple factors. Overall, it supports the idea that inclusion of persons with complex presenting symptoms and comorbidities should be included in trauma research. Additionally, it highlights the need for person-centered analyses to delve further into various symptom presentations and their relationship with neuropsychological outcomes.

Risk and Resilience Factors may be Differentially Associated with Neuropsychological Functioning in Subgroups (Veterans, Civilians, With PTSD, Without PTSD)

Pre-trauma cognitive ability, depression, and anxiety were the most prevalent resilience and risk factors identified through correlational analyses and regression models; however, other factors demonstrated relationships with neurocognitive functioning when examined within subgroups of the overall sample (those with and without PTSD and veterans/civilians). In those

with PTSD, depression severity was correlated with increased response time on two measures of the DKEFS as well as response time on the CPT-3. Additionally, self-report PTSD symptoms and personality factors were associated with slowed response time on the CPT-3.

This result echoes the predictive value of depression score discussed in the above analysis of moderate and severe symptom clusters, as many within these clusters had a diagnosis of PTSD. Additionally, it highlights slowed response times across other measures relating to PTSD severity. Prior literature on neurocognitive functioning in women with PTSD has found slowed response time across EF measures and neuropsychological measures not assessing EF (Twamley et al., 2009), and a correlation between PTSD severity and neurocognitive performance (Vasterling et al., 2002). Therefore, this result is quite similar to results in the PTSD literature base. Additionally, in terms of resilience factors, CD-RISC score was negatively correlated with CPT-3 perseverations in the PTSD group, suggesting those with PTSD who view themselves as having increased resilience and coping factors were less likely to exhibit repetitive anticipatory responses. While self-reported resilience did not show many significant correlations within the present results, this finding highlights that it may relate in some ways to neurocognitive performance, though it is by no means a primary predictor.

In those without PTSD, cumulative traumatic experiences and symptoms of PTSD as assessed via self-report and structured interview each evidenced negative correlations with performance on the IGT. These results suggest that even without a formal diagnosis of PTSD, those with elevated symptoms associated with poor post-trauma emotional functioning may also be at risk for decreased EF. This finding highlights the need to assess and provide treatment for those with emotional and neurocognitive difficulty following a traumatic experience, whether or not that person has a history of PTSD diagnosis or meets PTSD criteria. It also provides support

for future research into the emotional and cognitive functioning of those with a significant trauma history but without a diagnosis of the disorder.

Because of the high percentage of veterans within this sample also having a prior history of PTSD, trends between these groups were similar. Elevated PTSD symptoms, depression, anxiety, and characterological negative affect each were correlated with increased response time on the CPT-3. This reiterates the fact that veterans, and especially those with PTSD, may have increased difficulty with processing speed in attention tasks as negative mood and affect become more distressing.

Alternatively, the civilian group demonstrated unique patterns in terms of risk factors. In this group, PTSD symptom severity was correlated with increased commission errors, as would be expected. However, increased PTSD symptom severity and anxiety were both negatively correlated with response time. In other words, civilian participants with elevated distress related to post-trauma symptoms and anxiety were quick to respond to stimuli, but impulsive in their responding. Thus, while both veteran and civilian groups had reduced neurocognitive functioning as psychological distress increased, this distress was evidenced in different ways; it appeared as slowed processing speed in veterans and quick, impulsive responding in civilians. While the trends appearing in both civilians and veterans are reflective of relatively small sample sizes and therefore may not be representative of the larger population of either group, differences presented here emphasize the need to understand veteran and civilian women's unique responses to trauma, including psychological and neuropsychological manifestations of distress.

Differences Exist in Trauma Histories and Overall Psychological Functioning in those with and without PTSD

Results from secondary analyses revealed significant differences between subgroups

within the overall sample. Notably, there was significant overlap between veterans, those with a history of diagnosed PTSD, and those with present PTSD symptom elevations. Therefore, when talking about between-group differences, many similar patterns emerged among these three groups because of overlap in participants.

Differences emerged between groups with and without PTSD in terms of psychiatric comorbidities and present psychological functioning, supporting hypotheses. Between-group analyses revealed those with PTSD were more likely than those without it to have a diagnosis of bipolar disorder. Research on the comorbidity between the two disorders suggests that those with bipolar disorder are more likely to be diagnosed with PTSD, with estimates of comorbidity ranging from 16% to over 40% (Reddy et al., 2017). Therefore, the relationship observed in the present sample is consistent with literature, and provides insight into a subgroup of those who may be especially vulnerable to psychological effects following trauma.

Furthermore, those with a diagnosis of PTSD had more psychological distress than those without PTSD. Though groups with and without PTSD did not differ in terms of formal diagnosis of major depressive disorder or generalized anxiety disorder, those with PTSD endorsed elevated distress on measures of depression symptoms, anxiety symptoms, and PTSD symptoms. It has been hypothesized that women with pre-trauma mood and anxiety disorders are more likely to develop PTSD following trauma (Scali et al., 2012), yet the directionality of cause and effect is unclear in the present study. It is possible that having a diagnosis of PTSD has contributed to increased depression and anxiety, or that the symptoms assessed on mood inventories closely mimic those evidenced in PTSD. While the direction of the relationship between depression, anxiety, and PTSD symptoms is not clear, it is apparent that those with a formal diagnosis of PTSD are more likely to have more elevated symptoms overall. This result

has implications in the treatment of PTSD as well as management of co-occurring psychological symptoms that may be present. Essentially, treatment providers should be aware of the full range of symptoms those women with PTSD are experiencing, and design a treatment program to address the full spectrum of mood, anxiety, and PTSD manifestations.

In terms of trauma history, those with a history of PTSD had more distinct traumatic events than those with a trauma history but without a diagnosis of PTSD. Those with PTSD had experienced on average three more traumatic events across their lifetimes than those without PTSD, demonstrating the nature of repeated and compounding trauma. The experience of multiple traumatic events across one's lifetime is more prevalent than experience of just one trauma (Kessler et al., 1995), and the experience of one traumatic event is associated with risk for experiencing further trauma (Banyard et al., 2001; Casey & Nurius, 2005). Experiencing multiple traumatic events places an individual at an increased risk of negative mental health outcomes, including greater risk of PTSD and worsened PTSD severity (Briere, Agee, & Dietrich, 2016), but also including increased risk of depression, lower functional independence and increased risk of somatic disorders (Agorastos et al., 2014; Karam et al., 2014; Afari et al., 2014). In this way, the present results are consistent with the literature on cumulative traumas, and highlight the need for special focus on those who have experienced multiple traumas and may be at risk for PTSD and comorbid psychological distress.

Additionally, those with PTSD experienced different types of traumatic events than those without PTSD, though these results largely echo differences between civilian and veteran participants. For example, combat placed participants at a high risk for PTSD, with all six veteran participants in the study with combat experience also having a prior diagnosis of the disorder. Established PTSD literature supports the notion that combat exposure places veterans at

an increased risk of developing PTSD, and thus this result is consistent with predictions (Hoge & Warner, 2014). None of the participants who had a primary trauma of learning about the death of a loved one had a diagnosis of PTSD. Within the general population, this category of traumatic experience is also less associated with PTSD than other events, such as sexual or interpersonal violence (Smith, Summers, Dillon, & Cougle, 2016). Age of primary trauma also differed between those with and without PTSD; those with a diagnosis of PTSD were more likely to have experienced their most significant traumatic event while in adulthood. This result differs from literature supporting the lasting effects of childhood trauma (Ogle, Rubin, & Siegler, 2013), but may relate more to type of trauma reported in this particular sample, because many participants experienced interpersonal trauma, combat, or sexual violence in adulthood.

While hypotheses predicting intergroup differences in terms of psychological functioning and traumatic events were largely supported, differences in neuropsychological functioning between those with and without PTSD were not evidenced in the data. Scores across measures of inhibition, task-switching, sustained attention, and affective decision making were average both in those with and without history of PTSD. A difference in speed of performance on an inhibition task (DKEFS Color-Word Interference) was the only measure that approached significance, with the PTSD group's performance falling on the low end of the average range. There are a number of reasons why between group differences may not have been observed, each of which is addressed in the limitations and future directions section below.

Differences Exist in Trauma History, Psychological, and Neurocognitive Functioning in Civilians and Veterans

Because of the significant overlap between those with a history of PTSD and those with elevated present symptoms of the disorder, between group differences between those with and

without elevated symptoms were similar to the differences described above. Overlap also accounted for similarities in analyses comparing veteran and civilian participants, but with some notable differences. Veteran participants were more likely to be diagnosed with bipolar disorder, generalized anxiety disorder, and PTSD than civilian participants, highlighting greater risk for psychiatric distress in this group. They also had more elevated present distress, as evidenced by greater scores on depression, anxiety, and PTSD inventories.

Additional analyses explored differences in primary traumatic event, as well as lifetime experience of trauma, between veteran and civilian groups. On average, veterans experienced nearly four more traumatic events across their lifetimes than civilians. While their primary events were more likely to be MST or combat due to the military-nature of these traumas, they were also more likely to have experienced accidents, natural disasters, exposure to chemicals, situations where they feared for their lives or saw dead bodies, rape, and other forms of sexual assault than civilians. These results emphasize the increased lifetime risk of trauma in female veterans when compared to civilians, and highlight the need for post-trauma services in this population.

Along with poorer psychological functioning, veterans evidenced lower scores on two indices of neuropsychological performance. First, they made more errors on an inhibition/switching task, suggesting decreased executive control on tasks of increased complexity. Additionally, they took longer to establish patterns to inform decision making on a task high in affective load. While these two differences highlight only minor divergence between veterans and civilians, they point to areas for potential neurocognitive intervention. Veteran services may supplement assessments of psychological functioning with screeners evaluating

cognitive functioning to inform diagnosis and treatment of post-trauma female military members and veterans.

Limitations and Future Directions

Although the present study offered results and discussion points informing research on post-trauma psychological and neuropsychological functioning in women, limitations existed that may have affected analyses and reduced generalizability of results. Regarding statistical analyses, due to the exploratory nature of this study, adjustments were not made to alpha values resulting from multiple correlations. Had adjustments been made, it is possible that some of the smaller values would have lost significance. While the sample size was sufficient to offer moderate power, a larger overall sample with increased representation of both civilians and veterans would have increased power and decreased likelihood of type 2 error. Furthermore, the civilian group in the present study was largely homogenous due to the large representation of college students, while the veteran group had a large percentage of women with PTSD. Future studies specifically aiming to evaluate differences between civilian and female veterans post-trauma may seek to recruit a more representative community sample that is similar in terms of age, education, and ethnicity to the veteran sample. Additionally, future studies examining veteran and civilian differences may balance percentage of participants with PTSD in each group.

Because the focus of the present study was within-group differences in terms of risk and resilience factors predicting post-trauma neuropsychological outcomes, a control group of individuals without a significant traumatic experience was not included. However, use of a control group would have allowed for comparison of scores on psychological and

neuropsychological measures between those with and without trauma experiences, to better understand the effect of trauma on functioning. Future studies may incorporate a control group together with post-trauma PTSD and post-trauma without PTSD groups to assess a broad spectrum of outcomes.

Overall, participants performed relatively well on neuropsychological measures. Therefore, when talking about differences between groups or predictive nature of risk and resilience factors, it is important to note that differences may be subtle. However, though participants performed generally well on the measures used, they may evidence difficulty in concentration, decision making, and inhibition in functional tasks within their daily lives. While the present study assessed EF using standardized neuropsychological tasks, these tasks were low on ecological validity, and participants' functioning in real-world settings was not evaluated. Both self-report measures of EF and more ecologically valid tasks may have provided more information on real-world functioning of those women who have had traumatic experiences. Additionally, more difficult neuropsychological tasks with higher sensitivity to inefficiencies in attention and EF may have more accurately assessed participants' functioning. Future studies may utilize ecologically valid in vivo or virtual reality tasks to better assess how EF affects real-world activities.

The veterans within this study were largely recruited from trauma therapy groups at the VA. While this sampling method was good for identifying women who had experienced significant trauma, it also limited the veteran group to women who were involved in treatment to reduce symptoms. Throughout the course of therapeutic treatment, patients' symptoms sometimes become more pronounced as they work through their trauma experiences. As treatment successfully progresses, it may be effective in not only reducing emotional symptoms

related to PTSD, but also in improving neurocognitive outcomes. If early in treatment, these participants may have had elevated distress compared to baseline levels or, if later in treatment, they may have previously had poorer functioning that was not observed in this study. Future research may seek to identify women with a recent history of trauma or a new diagnosis of PTSD who have not yet received treatment, reducing within-sample variability in treatment effects.

Although the present study assessed a range of factors that may predict neuropsychological functioning post-trauma, other factors were not assessed. The self-report resilience measure provided a broad assessment of a vast concept, and specific resilience factors were not assessed in depth. For example, social support at the time of the trauma and subsequent to the trauma may predict better psychological functioning and also better neuropsychological functioning. Additionally, biological factors, including hormones and chromosome biomarkers, may make an individual more susceptible to PTSD symptoms. In terms of risk factors, anxiety acted as a predictor of poorer executive functioning. Item analysis revealed that three items were of particular importance: dizziness, feeling hot, and inability to relax. In a veteran population with elevated physical complaints, it is possible that the source of this correlation is physical rather than psychological dysfunction, and physical correlates of poorer outcomes may be considered. Future studies may seek to evaluate these and other risk and resilience factors in the study of post-trauma neuropsychological functioning.

Overall, findings highlight the need for intervention to reduce psychological distress and improve cognitive functioning in those individuals most at risk for poor neuropsychological outcomes following trauma. Yet, no evidence-based therapies exist to effectively target and improve neuropsychological functioning in those with trauma histories. The best avenue currently available to treat these individuals is evidence-based treatment for PTSD, including

prolonged exposure and cognitive processing therapy that can reduce post-trauma symptoms and thus also lead to an improvement in cognitive complaints. Future research may focus on the effectiveness of these therapies in improving neuropsychological outcomes.

Finally, effort and engagement were not assessed in the present study. Within the informed consent process, it was explained to veterans that results from the study would not be placed in their medical record and would in no way affect benefits. Similarly, civilians were explained confidentiality of records. In this way, the researcher sought to reduce incentive for intentional malingering. Additionally, performance on neuropsychological tests was generally consistent with what would be expected with adequate engagement. However, it is still possible that participants did not put forth full effort on tasks. Future research may aim to incorporate stand-alone and embedded effort measures within studies of neuropsychological functioning post-trauma.

Conclusions

In a sample of veteran and civilian women reporting significant traumatic experiences, premorbid intellectual functioning was the best predictor of neuropsychological test performance. Level of anxiety also played a role in the ability to respond to risk and reward stimuli and detect patterns to make beneficial choices (hot EF), with higher anxiety hindering performance. On a person-centered basis, not all those who had experienced elevated trauma evidenced similar resulting symptoms, with risk and resilience groups emerging. These groups, who differed in level of anxiety, depression, and PTSD symptoms, also exhibited differences on a task of attention and processing speed, demonstrating a subcortical inefficiency associated with higher symptom presentation. Depression symptoms appeared to provide a link between

psychological presentation and tasks related to subcortical functioning in those with especially elevated trauma and moderate to severe symptoms, with anxiety continuing to predict affective problem solving.

Examinations of subgroups within the overall sample revealed differences between those with PTSD and those without, as well as between civilians and veterans, in terms of trauma history, psychological functioning, and neuropsychological test performance. As would be expected, those with PTSD had higher levels of psychological symptoms, including depression and anxiety, than those with trauma histories but without diagnoses of PTSD. They did not, however, differ on neuropsychological functioning. Veterans and civilians differed in type of trauma experienced and veterans displayed elevated psychological symptoms. Furthermore, veterans exhibited more errors and a longer time to establish patterns on two tests of EF.

Together, results highlight the need to assess those who have experienced trauma in terms of psychological and neuropsychological functioning and provide intervention to those who may be most at risk, including those with elevated depression and anxiety. They establish pre-trauma intellectual functioning as a significant predictor of post-trauma neurocognitive functioning, identifying the need for interventions to aid those with lower premorbid cognitive functioning and higher potential to express deficits after trauma. While no evidence-based interventions exist to effectively improve neurocognitive functioning, therapies that reduce overall distress and incorporate real-world strategies to aid in tasks high in cognitive demands may be especially beneficial to those subgroups most at risk. Finally, examination of between-group differences identified differences between veterans and civilians in terms of trauma exposure, psychological symptoms, and neuropsychological functioning. Future research may seek to continue

delineating the psychological and neuropsychological needs of these two groups and target interventions specific to each of their needs.

Table 1

*Descriptive Statistics: Total Sample (*n = *60)*

		Range	Mean (SD)
Demographic	Age	18 - 55	29.73 (10.91)
	Education	12 – 18	14.20 (1.58)
		Frequency	Percentage
Ethnicity	White/Caucasian	28	46.7%
	Black/African American	20	33.3%
	Hispanic	8	13.3%
	Asian	2	3.3%
	Other	2	3.3%
Psychiatric Diagnosis	Major Depressive Disorder	30	50%
	Bipolar Disorder	10	16.7%
	Generalized Anxiety Disorder	15	25%
	PTSD	29	48.3%

Table 2

Scores on Psychological Measures: Total Sample

Measure	Range	Mean (SD)
WTAR	68 - 124	99.68 (12.98)
BDI	1 – 53	25.22 (12.47)*
BAI	3 – 53	22.98 (12.69)**
EPQR-A	8 – 24	16.32 (4.12)
THQ	2 – 18	8.20 (3.98)
PCL-5	5 – 74	40.78 (18.99)
CD-RISC	16 - 96	60.62 (17.77)
CAPS-5	0 – 63	31.48 (15.30)***

*BDI-2 Ranges: 10 (16.7%) Minimal; 8 (13.3%) Mild; 22 (36.7%) Moderate; 20 (33%) Severe. **BAI Ranges: 9 (15%) Minimal; 14 (23.3%) Mild; 19 (31.7%) Moderate; 18 (30%) Severe. ***N = 36 (60%) meet CAPS-5 Criteria for PTSD

Table 3

Category of Criterion A Trauma: Total Sample

Category	Frequency	Percentage
Sexual Violence	35	58.3%
Military Sexual Trauma	10	16.7%
Combat	6	10%
Serious Injury/Abuse	7	11.7%
Life Threat	3	5%
Witnessing Death/Serious Injury/Abuse	6	10%
Learning About Traumatic Death	3	5%

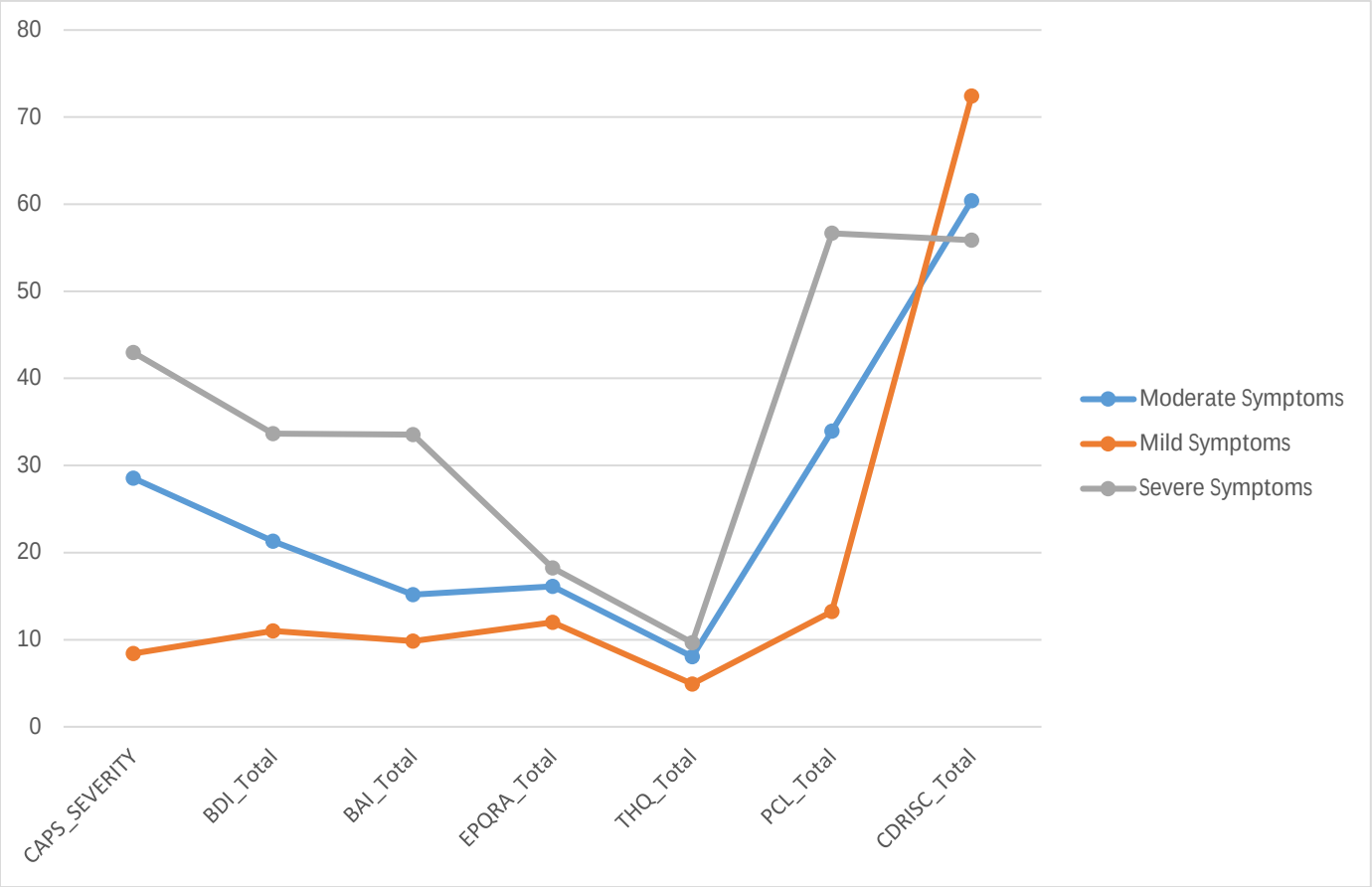

Figure 1. Psychological questionnaire scores across mild, moderate, and severe symptom clusters.

Table 4

Scores on Neuropsychological Measures: Total Group & Subgroups

	Measure	Total Sample Mean	PTSD History Group Mean	No PTSD History Group Mean	PTSD Present Group Mean	PTSD Absent Group Mean	Veteran Group Mean	Civilian Group Mean
DKEFS Color Word Interference Test (Scaled Scores)	Color Naming	9.58	8.97	10.16	9.33	9.96	9.56	9.61
	Word Reading	10.23	9.90	10.55	10.19	10.29	10.37	10.12
	CW Interference	9.48	8.72	10.19	9.14	10.00	8.67	10.15
	CW Interference Errors	9.85	9.59	10.10	9.78	9.96	9.41	10.21
	Interference/Switching	9.45	9.07	9.81	9.44	9.46	8.81	9.97
	Interference/Switching Errors	10.43	10.24	10.61	10.19	10.79	9.70	11.03
Iowa Gambling Task (T Scores)	Net 1	48.70	46.78	50.43	48.00	49.67	45.04	51.77
	Net 2	49.93	52.48	47.63	51.09	48.33	52.38	47.87
	Net 3	47.56	48.96	46.30	47.12	48.17	46.92	47.87
	Net 4	45.91	45.81	46.00	45.33	46.71	45.00	48.10
	Net 5	43.39	41.22	45.33	42.18	45.04	41.12	46.68
	Total Net	46.35	46.70	46.03	46.12	46.67	45.31	45.29
	Total Money (raw)	-741.67	-714.26	-766.33	-756.36	-721.46	-949.81	-567.10
Continuous Performance Test – 3 (T Scores)	Omissions	54.12	50.11	58.29	51.34	58.29	50.96	57.17
	Comissions (T score)	55.24	54.80	55.70	55.52	54.83	53.81	56.62
	Response Time (T score)	49.48	47.96	51.04	48.57	50.84	47.81	51.08
	Detectability (T score)	53.37	53.33	55.45	53.91	55.06	53.74	54.98
	Perseverations (T score)	54.70	52.90	56.57	56.21	52.44	54.15	55.24

Table 5

Psychological Measure Results of Mild Symptoms, Moderate Symptoms, and Severe Symptoms Clusters

	Mild Symptoms Group Mean (SD)	Moderate Symptoms Group Mean (SD)	Severe Symptoms Group Mean (SD)	ANOVA F value	p value, h^2
THQ	4.92 (2.39)	8.05 (4.05)	9.66 (3.71)	7.34	$p < .01$**, $h^2 = .21$
CAPS-5 Severity	8.42 (5.13)	28.53 (7.07)	42.97 (8.97)	85.89	$p < .001$**, $h^2 = .75$
PCL-5	13.25 (7.46)	56.66 (9.69)	33.95 (6.47)	123.39	$p < .001$**, $h^2 = .81$
BDI	11.00 (6.27)	21.32 (8.58)	33.66 (9.71)	30.98	$p < .001$**, $h^2 = .52$
BAI	9.83 (5.86)	15.16 (5.06)	33.55 (8.27)	61.14	$p < .001$**, $h^2 = .68$
EPQR-A	12.00 (3.22)	16.11 (3.74)	18.24 (3.29)	14.14	$p < .001$**, $h^2 = .33$
CD-RISC	72.42 (13.51)	60.42 (12.93)	55.86 (20.07)	4.07	$p < .05$*, $h^2 = .13$
WTAR	90.25 (11.55)	104.84 (11.21)	100.21 (12.86)	5.39	$p < .01$**, $h^2 = .16$

* significant at $p < .05$. ** significant at $p < .01$

Table 6

Comparison between Scores on Self-Report Measures for Moderate Symptoms and Severe Symptoms Clusters

	t-value	Significance (p)	Effect size (h^2)
THQ	-1.413	.164	.42
CAPS-5	-5.91	<.001**	1.79
PCL-5	-9.74	<.001**	2.76
BDI	-4.50	<.001**	1.35
BAI	-9.16	<.001**	2.56
EPQR-A	-2.08	<.05*	.60
CD-RISC	.88	.385	.27

* significant at p < .05. ** significant at p < .01

Table 7

Comparison between Scores on Self-Report Measures for Mild Symptoms and Severe Symptoms Clusters

	t-value	Significance (p)	Effect size (h^2)
THQ	-4.08	<.001**	1.52
CAPS-5	-12.47	<.001**	4.73
PCL-5	-13.87	<.001**	5.02
BDI	-7.44	<.001**	2.77
BAI	-8.53	<.001**	3.17
EPQR-A	-5.56	<.001**	1.92
CD-RISC	2.61	<.05*	.97

* significant at p < .05. ** significant at p < .01

Table 8

Comparison between Scores on Self-Report Measures for Moderate Symptoms and Mild Symptoms Clusters

	t-value	Significance (p)	Effect size (h^2)
THQ	2.42	<.05*	.94
CAPS-5	8.52	<.001**	3.26
PCL-5	8.18	<.001**	2.96
BDI	3.59	<.01**	1.37
BAI	2.69	<.05*	.97
EPQR-A	3.14	<.01**	1.67
CD-RISC	-2.47	<.05*	.91

* significant at p < .05. ** significant at p < .01

Table 9

Results from a MANOVA exploring Veteran/Civilian x Cluster Effects on Self-Report Measures

	F value	Significance (p)	Effect size (h^2)
CAPS-5	.19	.658	.00
PCL-5	.05	.824	.00
BDI	1.88	.177	.04
BAI	.03	.864	.00
EPQR-A	.44	.509	.01
THQ	.01	.91	.00
CD-RISC	.01	.914	.00

Table 10

Correlations between Psychological Measures & DKEFS Color Word Interference Scaled Scores: Pearson Values & Significance

	CAPS-5 Severity	PCL-5	THQ	BDI-2	BAI	EPQR-A	CD-RISC	WTAR
Color Naming	-.09	-.12	.05	-.25	-.08	.02	.21	.19
p value	*.514*	*.360*	*.695*	*.056*	*.540*	*.023*	*.102*	*.152*
Word Reading	.01	-.10	.07	-.23	<.01	.03	.17	.33**
p value	*.967*	*.432*	*.615*	*.084*	*.999*	*.850*	*.195*	*.009*
Color Word Interference	-.06	-.10	-.02	-.16	-.04	.01	.03	.31*
p value	*.670*	*.432*	*.875*	*.231*	*.752*	*.933*	*.817*	*.016*
Color Word Interference Errors	-.141	-.01	-.09	-.10	-.19	-.17	-.03	.22
p value	*.283*	*.951*	*.512*	*.467*	*.156*	*.188*	*.822*	*.096*
Interference/Switching	-.04	-.02	-.06	-.10	-.10	.04	.05	.27*
p value	*.783*	*.893*	*.663*	*.467*	*.434*	*.737*	*.715*	*.034*
Interference/Switching Errors	-.08	-.07	-.15	<.01	-.06	-.07	-.10	.04
p value	*.556*	*.619*	*.239*	*.981*	*.634*	*.614*	*.428*	*.761*

* significant at $p < .05$. ** significant at $p < .01$

Table 11

Correlations between Psychological Measures & IGT Performance (T scores): Pearson Values & Significance

		CAPS-5 Severity	PCL-5	THQ	BDI-2	BAI	EPQR-A	CD-RISC	WTAR
Net 1		-.04	.08	-.15	-.10	-.08	-.04	.14	-.22
	p value	*.743*	*.568*	*.273*	*.473*	*.580*	*.777*	*.294*	*.097*
Net 2		.23	.15	.05	.21	.26	.10	-.15	.19
	p value	*.088*	*.263*	*.696*	*.205*	*.055*	*.466*	*.294*	*.159*
Net 3		-.08	-.18	-.11	-.08	-.14	.02	.15	.11
	p value	*.541*	*.184*	*.401*	*.552*	*.294*	*.864*	*.265*	*.409*
Net 4		-.08	-.15	-.13	-.02	-.09	-.04	<.01	.20
	p value	*.563*	*.261*	*.344*	*.884*	*.498*	*.767*	*.978*	*.146*
Net 5		-.13	-.11	-.19	-.01	-.32*	-.15	-.27*	.28*
	p value	*.354*	*.406*	*.153*	*.947*	*.016*	*.252*	*.045*	*.039*
Total Net		-.02	-.07	-.16	.03	-.11	-.03	-.07	.24
	p value	*.881*	*.613*	*.222*	*.852*	*.416*	*.822*	*.620*	*.069*
Total Money (Raw)		.02	<.01	-.15	.11	-.01	-.07	-.10	.19
	p value	*.907*	*.994*	*.273*	*.399*	*.922*	*.592*	*.471*	*.158*

* significant at $p < .05$. ** significant at $p < .01$

Table 12

Correlations between Psychological Measures & CPT T Scores: Pearson Value & Significance

	CAPS-5 Severity	PCL-5	THQ	BDI-2	BAI	EPQR-A	CD-RISC	WTAR
Omissions	-.07	.02	<.01	-.11	-.10	-.07	.04	-.06
p value	*.602*	*.891*	*.981*	*.431*	*.461*	*.594*	*.790*	*.657*
Comissions	.090	-.05	-.05	-.07	-.01	.05	-.11	.02
p value	*.512*	*.721*	*.736*	*.622*	*.928*	*.693*	*.428*	*.870*
Response Time	-.08	.09	-.08	.16	<.01	.18	-.23	-.19
p value	*.554*	*.518*	*.556*	*.246*	*.982*	*.198*	*.092*	*.162*
Detectability	<.01	-.14	-.04	-.16	-.17	-.12	.07	.07
p value	*.987*	*.302*	*.771*	*.374*	*.223*	*.394*	*.622*	*.639*
Perseverations	.22	.16	.15	.06	.16	-.15	-.06	.19
p value	*.102*	*.240*	*.269*	*.666*	*.239*	*.288*	*.687*	*.165*

* significant at p < .05. ** significant at p < .01

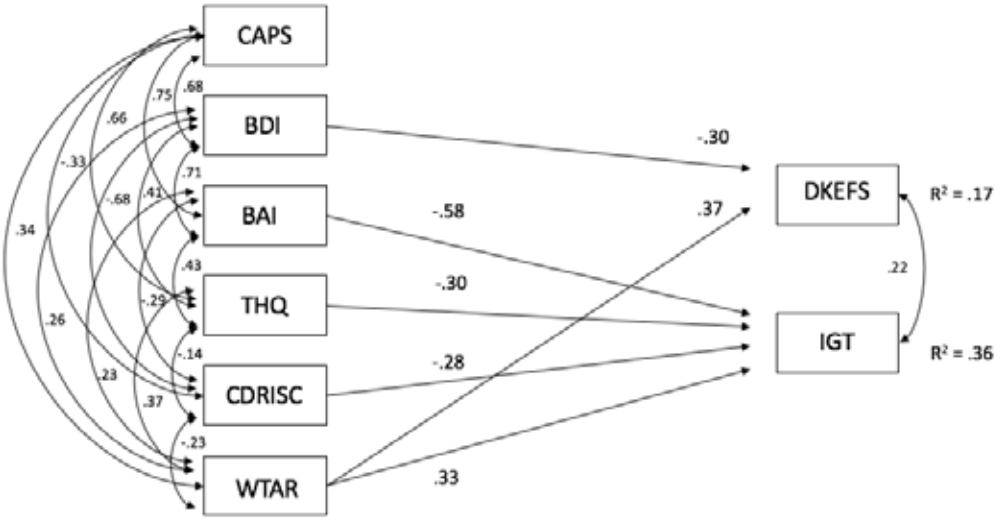

Figure 2. Path model assessing risk and resilience factors and neuropsychological test performance (including CAPS-5).

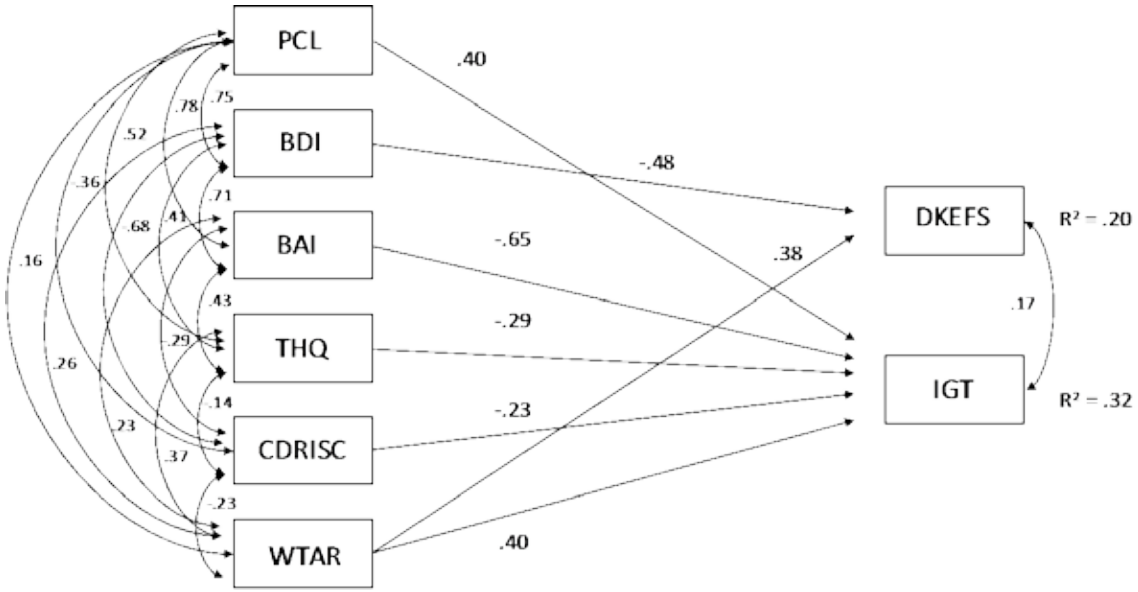

Figure 3. Path model assessing risk and resilience factors and neuropsychological test performance (including PCL-5).

Table 13

Moderate and Severe Symptoms Clusters

Measure	Predictor variable	b	t	p	R^{2-}	Adjusted R^2
DKEFS Color Naming	BDI-2	-.37	-2.70	.010*	.14	.12
	BAI	.13	.76	.454	-	-
	EPQR-A	.20	1.30	.202	-	-
	THQ	.10	.73	.468	-	-
	PCL-5	-.09	-.52	.608	-	-
DKEFS Color-Word Interference	BDI-2	-.30	-2.17	.035*	.09	.07
	BAI	.05	.26	.798	-	-
	EPQR-A	.01	.06	.953	-	-
	THQ	<-.01	-.02	.987	-	-
	PCL-5	.04	.23	.821	-	-
DKEFS Interference/Switching	BDI-2	-.30	-2.10	.041*	.09	.07
	BAI	-.12	-.65	.518	-	-
	EPQR-A	.02	.11	.912	-	-
	THQ	-.07	-.45	.653	-	-
	PCL-5	-.10	-.56	.576	-	-
IGT Net 5	BDI-2	.17	1.06	.297	-	-
	BAI	-.44	-3.19	.003**	.19	.17
	EPQR-A	-.13	-.92	.365	-	-
	THQ	-.19	-1.34	.186	-	-
	PCL-5	-.07	-.35	.728	-	-
CPT-3 Comissions	No significant predictors.					

[a] Predictors entered into regression model were: PCL-5 score, BDI-2 score, BAI score, THQ score, and EPQR-A score. * significant at $p < .05$. ** significant at $p < .01$

Table 14

Veteran Group - Correlations between Psychological Measures & DKEFS Scaled Scores: Pearson Values & Significance

	CAPS-5 Severity	PCL-5	THQ	BDI-2	BAI	EPQR-A	CD-RISC	WTAR
Color Naming	-.19	-.12	-.03	-.30	-.07	-.07	.36	.02
p value	*.347*	*.567*	*.900*	*.134*	*.717*	*.748*	*.063*	*.919*
Word Reading	<-.01	-.05	-.06	-.20	0.07	<-.01	.22	.18
p value	*.990*	*.801*	*.766*	*.323*	*.724*	*.996*	*.261*	*.378*
Color Word Interference	.06	0.04	0.02	-.09	-.05	-.08	0.02	.37
p value	*.775*	*.829*	*.927*	*.667*	*.805*	*.689*	*.933*	*.061*
Color Word Interference Errors	-.26	-.26	-.29	-.06	-.21	-.34	-.06	.36
p value	*.195*	*.188*	*.146*	*.773*	*.286*	*.080*	*.766*	*.064*
Interference/Switching	<.01	.07	<.01	.04	-.15	<-.01	<.01	.21
p value	*.992*	*.724*	*.983*	*.860*	*.464*	*.992*	*.972*	*.296*
Interference/Switching Errors	.21	.13	.19	.15	.05	-.17	-.17	.24
p value	*.292*	*.521*	*.351*	*.442*	*.823*	*.408*	*.394*	*.233*

* significant at p < .05. ** significant at p < .01

Table 15

Veteran Group - Correlations between Psychological Measures & IGT Performance (T Scores): Pearson Values & Significance

		CAPS-5 Severity	PCL-5	THQ	BDI-2	BAI	EPQR-A	CD-RISC	WTAR
Net 1		.12	.16	-.03	-.03	.01	.05	.13	-.14
	p value	*.558*	*.437*	*.889*	*.875*	*.950*	*.809*	*.520*	*.497*
Net 2		.16	<.01	-.06	.06	.27	-.10	-.01	.26
	p value	*.442*	*.990*	*.763*	*.786*	*.186*	*.623*	*.964*	*.200*
Net 3		<-.01	-.17	-.06	-.08	-.03	.04	.23	.19
	p value	*.985*	*.411*	*.762*	*.698*	*.872*	*.834*	*.254*	*.353*
Net 4		-.04	-.20	-.09	-.06	-.13	.05	.05	.30
	p value	*.840*	*.318*	*.671*	*.770*	*.518*	*.825*	*.794*	*.139*
Net 5		-.04	<.01	-.16	.14	-.28	-.09	-.30	.34
	p value	*.852*	*.984*	*.437*	*.501*	*.173*	*.670*	*.143*	*.089*
Total Net		.09	-.06	-.12	.03	-.05	<.01	.01	.35
	p value	*.662*	*.789*	*.565*	*.892*	*.826*	*.995*	*.957*	*.076*
Total Money (Raw)		.13	-.02	-.14	.16	<-.01	-.03	-.07	.46*
	p value	*.538*	*.914*	*.506*	*.426*	*.973*	*.873*	*.739*	*.019*

* significant at p < .05. ** significant at p < .01

Table 16

Veteran Group - Correlations between Psychological Measures & CPT T Scores: Pearson Values & Significance

	CAPS-5 Severity	PCL-5	THQ	BDI-2	BAI	EPQR-A	CD-RISC	WTAR
CPT Omissions	.08	.14	-.16	.19	.04	.30	-.28	-.26
p value	*.691*	*.489*	*.440*	*.350*	*.860*	*.126*	*.164*	*.190*
CPT Comissions	-.12	-.25	-.09	-.14	-.09	.10	.11	-.06
p value	*.545*	*.208*	*.645*	*.491*	*.657*	*.632*	*.572*	*.784*
CPT Response Time	.28	.45*	.19	.52**	.40*	.46*	-.27	-.01
p value	*.158*	*.020*	*.341*	*.005*	*.039*	*.015*	*.178*	*.944*
CPT Detectability	-.19	-.35	.01	-.28	-.34	-.22	<.01	.03
p value	*.348*	*.078*	*.951*	*.151*	*.083*	*.267*	*.984*	*.875*
CPT Perseverations	.11	-.02	.12	.15	<-.01	.01	-.21	.19
p value	*.580*	*.906*	*.566*	*.467*	*.980*	*.969*	*.297*	*.349*

* significant at p < .05. ** significant at p < .01

Table 17

Civilian Group - Correlations between Psychological Measures & DKEFS Scaled Scores: Pearson Values & Significance

	CAPS-5 Severity	PCL-5	THQ	BDI-2	BAI	EPQR-A	CD-RISC	WTAR
Color Naming	<.01	-.14	.15	-.22	-.09	.14	-.02	.38*
p value	*.990*	*.443*	*.397*	*.209*	*.608*	*.445*	*.902*	*.030*
Word Reading	-.02	-.19	.15	-.30	-.08	.04	.12	.47**
p value	*.898*	*.298*	*.410*	*.087*	*.643*	*.828*	*.491*	*.005*
Color Word Interference	.03	.10	.17	-.09	.09	.18	-.04	.41*
p value	*.850*	*.573*	*.351*	*.601*	*.625*	*.314*	*.814*	*.018*
Color Word Interference Errors	.03	.03	.18	-.05	-.11	.03	-.05	.19
p value	*.849*	*.891*	*.316*	*.793*	*.560*	*.883*	*.764*	*.289*
Interference/Switching	.11	.04	.09	-.12	.06	.19	.03	.50**
p value	*.549*	*.823*	*.638*	*.511*	*.750*	*.286*	*.874*	*.003*
Interference/Switching Errors	-.11	-.03	-.25	.07	.04	.25	-.18	.01
p value	*.562*	*.867*	*.169*	*.696*	*.829*	*.164*	*.316*	*.959*

* significant at $p < .05$. ** significant at $p < .01$

Table 18

Civilian Group - Correlations between Psychological Measures & IGT Performance (T Scores): Pearson Values & Significance

		CAPS-5 Severity	PCL-5	THQ	BDI-2	BAI	EPQR-A	CD-RISC	WTAR
Net 1		.16	-.05	.11	.14	.08	-.03	-.05	-.19
	p value	*.393*	*.780*	*.558*	*.468*	*.668*	*.878*	*.784*	*.339*
Net 2		.08	.13	-.14	.19	.10	.25	-.26	<.01
	p value	*.651*	*.496*	*.452*	*.318*	*.580*	*.179*	*.159*	*.996*
Net 3		-.12	-.14	-.13	-.04	-.23	.02	-.02	.07
	p value	*.518*	*.442*	*.486*	*.825*	*.217*	*.905*	*.921*	*.695*
Net 4		-.05	-.07	-.11	.06	-.03	-.09	-.09	.18
	p value	*.785*	*.701*	*.555*	*.741*	*.873*	*.643*	*.921*	*.344*
Net 5		-.07	-.17	-.10	-.02	-.29	-.17	-.36*	.32
	p value	*.705*	*.363*	*.588*	*.923*	*.118*	*.351*	*.047*	*.076*
Total Net		-.02	-.10	-.13	.12	-.11	-.03	-.26	.22
	p value	*.936*	*.579*	*.482*	*.539*	*.564*	*.887*	*.164*	*.239*
Total Money (Raw)		.07	-.04	-.03	.21	.08	-.06	-.24	.05
	p value	*.696*	*.821*	*.879*	*.259*	*.660*	*.733*	*.187*	*.808*

* significant at p < .05. ** significant at p < .01

Table 19

Civilian Group - Correlations between Psychological Measures & CPT T Scores: Pearson Values & Significance

	CAPS-5 Severity	PCL-5	THQ	BDI-2	BAI	EPQR-A	CD-RISC	WTAR
CPT Omissions	-.03	.06	.24	-.16	-.10	-.19	.17	.06
p value	*.865*	*.760*	*.228*	*.407*	*.614*	*.330*	*.385*	*.770*
CPT Comissions	.43*	.19	.16	.08	.12	.06	.07	.17
p value	*.023*	*.339*	*.413*	*.701*	*.535*	*.777*	*.730*	*.375*
CPT Response Time	-.40*	-.19	-.28	-.21	-.40*	-.19	-.24	-.37
p value	*.034*	*.334*	*.157*	*.290*	*.036*	*.325*	*.210*	*.051*
CPT Detectability	.31	.10	-.02	.04	.06	.06	.16	.16
p value	*.109*	*.607*	*.935*	*.837*	*.782*	*.771*	*.407*	*.409*
CPT Perseverations	.36	.27	.29	.05	.27	-.24	.03	.24
p value	*.062*	*.158*	*.135*	*.814*	*.165*	*.215*	*.888*	*.221*

* significant at $p < .05$. ** significant at $p < .01$

Table 20

PTSD History Group - Correlations between Psychological Measures & DKEFS Scaled Scores: Pearson Values & Significance

	CAPS-5 Severity	PCL-5	THQ	BDI-2	BAI	EPQR-A	CD-RISC	WTAR
Color Naming	-.22	-.21	.05	-.38*	-.04	-.03	.35	.22
p value	*.263*	*.273*	*.805*	*.044*	*.830*	*.897*	*.061*	*.245*
Word Reading	-.17	-.30	.07	-.37	.01	-.09	.24	.41*
p value	*.380*	*.116*	*.709*	*.050*	*.954*	*.628*	*.211*	*.027*
Color Word Interference	-.01	.01	.08	-.10	.05	-.03	.06	.48**
p value	*.945*	*.957*	*.692*	*.624*	*.795*	*.878*	*.758*	*.008*
Color Word Interference Errors	-.43	-.30	-.30	-.13	-.28	-.31	<-.01	.26
p value	*.019*	*.110*	*.115*	*.497*	*.141*	*.101*	*.977*	*.167*
Interference/Switching	-.09	<.01	-.02	.01	-.03	.06	.02	.35
p value	*.651*	*.973*	*.913*	*.960*	*.871*	*.744*	*.903*	*.060*
Interference/Switching Errors	-.08	-.13	-.05	-.05	-.15	-.19	-.11	.15
p value	*.678*	*.493*	*.795*	*.805*	*.452*	*.323*	*.585*	*.431*

* significant at $p < .05$. ** significant at $p < .01$

Table 21

PTSD History Group - Correlations between Psychological Measures & IGT Performance (T Scores): Pearson Values & Significance

		CAPS-5 Severity	PCL-5	THQ	BDI-2	BAI	EPQR-A	CD-RISC	WTAR
Net 1		.29	.39*	-.04	.08	.17	.03	.02	-.25
	p value	*.147*	*.044*	*.858*	*.696*	*.397*	*.886*	*.932*	*.218*
Net 2		.13	-.07	.02	.08	.18	-.10	-.02	.21
	p value	*.51*	*.71*	*.92*	*.70*	*.38*	*.62*	*.92*	*.29*
Net 3		.09	-.12	<.01	-.01	.07	-.06	.06	.15
	p value	*.664*	*.559*	*1.000*	*.976*	*.727*	*.773*	*.761*	*.443*
Net 4		.11	-.04	-.10	.10	.07	<-.01	-.15	.14
	p value	*.598*	*.864*	*.617*	*.638*	*.738*	*.990*	*.469*	*.495*
Net 5		-.13	-.08	-.28	.04	-.40*	-.16	-.31	.20
	p value	*.510*	*.689*	*.160*	*.862*	*.037*	*.431*	*.112*	*.317*
Total Net		.16	.01	-.16	.10	.01	-.08	-.17	.21
	p value	*.441*	*.946*	*.429*	*.608*	*.971*	*.694*	*.406*	*.300*
Total Money (Raw)		.17	.04	-.21	.20	.05	-.12	-.16	.26
	p value	*.385*	*.859*	*.293*	*.324*	*.806*	*.541*	*.441*	*.185*

* significant at p < .05. ** significant at p < .01

Table 22

PTSD History Group - Correlations between Psychological Measures & CPT T Scores: Pearson Values & Significance

	CAPS-5 Severity	PCL-5	THQ	BDI-2	BAI	EPQR-A	CD-RISC	WTAR
CPT Omissions	.02	.19	-.05	.22	-.01	.20	-.27	-.48*
p value	*.936*	*.339*	*.790*	*.254*	*.963*	*.307*	*.173*	*.010*
CPT Comissions	-.20	-.25	-.01	-.05	-.04	.04	.04	-.05
p value	*.306*	*.208*	*.946*	*.802*	*.824*	*.848*	*.838*	*.813*
CPT Response Time	.31	.41*	.01	.40*	.18	.49**	-.33	-.22
p value	*.106*	*.029*	*.944*	*.034*	*.372*	*.009*	*.085*	*.268*
CPT Detectability	-.18	-.21	.16	-.04	-.26	-.10	-.15	-.09
p value	*.356*	*.276*	*.407*	*.860*	*.185*	*.614*	*.434*	*.660*
CPT Perseverations	.10	<-.01	.29	.24	-.11	.01	-.40*	.09
p value	*.601*	*.987*	*.139*	*.212*	*.567*	*.946*	*.037*	*.635*

* significant at p < .05. ** significant at p < .01

Table 23

No PTSD History Group - Correlations between Psychological Measures & DKEFS Scaled Scores: Pearson Values & Significance

	CAPS-5 Severity	PCL-5	THQ	BDI-2	BAI	EPQR-A	CD-RISC	WTAR
Color Naming	.37*	.27	.35	.15	.12	.28	-.13	.27
p value	*.039*	*.137*	*.054*	*.417*	*.527*	*.126*	*.487*	*.142*
Word Reading	.32	.23	.19	.02	.12	.24	.04	.30
p value	*.079*	*.214*	*.315*	*.933*	*.511*	*.200*	*.828*	*.097*
Color Word Interference	.17	.21	.08	-.04	.10	.19	-.07	.22
p value	*.369*	*.250*	*.662*	*.834*	*.606*	*.295*	*.694*	*.228*
Color Word Interference Errors	.15	.10	.21	.01	-.02	<.01	-.09	.21
p value	*.407*	*.586*	*.268*	*.944*	*.916*	*.984*	*.643*	*.265*
Interference/Switching	.14	.09	.01	-.11	-.07	.10	.05	.25
p value	*.459*	*.621*	*.972*	*.569*	*.691*	*.591*	*.811*	*.180*
Interference/Switching Errors	.01	.10	-.23	.16	.15	.13	-.14	-.06
p value	*.952*	*.581*	*.218*	*.404*	*.423*	*.487*	*.470*	*.732*

* significant at p < .05. ** significant at p < .01

Table 24

No PTSD History Group - Correlations between Psychological Measures & IGT Performance (T Scores): Pearson Values & Significance

		CAPS-5 Severity	PCL-5	THQ	BDI-2	BAI	EPQR-A	CD-RISC	WTAR
Net 1		-.14	-.07	-.13	-.14	-.20	<.01	.30	.13
	p value	*.466*	*.727*	*.502*	*.474*	*.279*	*.999*	*.113*	*.487*
Net 2		.01	.14	-.27	.08	.05	.19	-.32	.04
	p value	*.965*	*.471*	*.148*	*.658*	*.781*	*.323*	*.083*	*.830*
Net 3		-.40*	-.43*	-.37*	-.31	-.56**	.02	.28	.03
	p value	*.028*	*.018*	*.047*	*.094*	*.001*	*.930*	*.137*	*.887*
Net 4		-.24	-.30	-.17	-.13	-.29	-.07	.17	.26
	p value	*.197*	*.109*	*.364*	*.488*	*.126*	*.697*	*.362*	*.158*
Net 5		.05	.01	.02	.13	-.11	-.07	-.26	.45*
	p value	*.810*	*.956*	*.907*	*.504*	*.575*	*.723*	*.164*	*.012*
Total Net		-.21	-.21	-.24	-.09	-.32	-.01	.05	.27
	p value	*.256*	*.263*	*.197*	*.638*	*.084*	*.960*	*.775*	*.146*
Total Money (Raw)		-.15	-.07	-.14	.02	-.13	-.04	-.02	.10
	p value	*.418*	*.730*	*.475*	*.913*	*.496*	*.844*	*.935*	*.586*

* significant at p < .05. ** significant at p < .01

Table 25

No PTSD History Group - Correlations between Psychological Measures & CPT T Scores: Pearson Values & Significance

	CAPS-5 Severity	PCL-5	THQ	BDI-2	BAI	EPQR-A	CD-RISC	WTAR
CPT Omissions	.05	.11	.20	-.12	-.03	-.09	.13	.12
p value	*.790*	*.590*	*.309*	*.538*	*.870*	*.667*	*.530*	*.543*
CPT Comissions	.38	.16	-.06	-.07	.05	.09	.18	.11
p value	*.052*	*.441*	*.783*	*.743*	*.797*	*.652*	*.366*	*.574*
CPT Response Time	-.16	.02	-.03	.14	.01	.08	-.20	-.14
p value	*.434*	*.942*	*.900*	*.485*	*.979*	*.709*	*.317*	*.480*
CPT Detectability	.26	-.01	-.13	-.20	-.01	-.08	.27	.27
p value	*.186*	*.977*	*.521*	*.328*	*.968*	*.693*	*.166*	*.181*
CPT Perseverations	.44*	.35	.28	.09	.41*	-.17	.05	.31
p value	*.022*	*.076*	*.161*	*.652*	*.034*	*.384*	*.805*	*.112*

* significant at p < .05. ** significant at p < .01

Table 26

Total Sample - Regression Results[a]

Measure	Predictor variable	b	t	p	R^2	Adjusted R^2	ΔR^2
DKEFS Color Naming	WTAR	.19	1.45	.152	.04	.02	-
DKEFS Color-Word Interference	WTAR	.31	2.47	.016*	.10	.08	-
DKEFS Interference/Switching	WTAR	.27	2.17	.034*	.08	.06	-
IGT Net 5 (Model 1)	WTAR	.28	2.12	.039*	.08	.06	-
IGT Net 5 (Model 2)	WTAR	.35	2.87	.006**	.22	.19	.14
	BAI	-.39	-3.15	.003			
CPT-3 Comissions	WTAR	.02	.17	.870	<.01	-.02	-

[a]Simple linear regression run for DKEFS Color Naming, DKEFS Color-Word Interference, DKEFS Interference/Switching, and CPT-3 Comissions. Hierarchical multiple regression run for IGT Net 5. * significant at $p < .05$. ** significant at $p < .01$

Table 27

Moderate and Severe Symptoms Clusters: Regression Results[a]

Measure	Predictor variable	b	t	p	R^2	Adjusted R^2	ΔR^2
DKEFS Color Naming Model 1	WTAR	.20	1.35	.183	.04	.02	-
DKEFS Color Naming Model 2	WTAR	.23	1.68	.099	.19	.15	.15
	BDI-2	-.39	-2.88	.006**			
DKEFS Color-Word Interference Model 1	WTAR	.28	1.96	.056	.08	.06	-
DKEFS Color-Word Interference Model 2	WTAR	.30	2.25	.029*	.19	.15	.11
	BDI-2	-.33	-2.44	.019*			
DKEFS Interference/Switching Model 1	WTAR	.20	1.35	.184	.04	.02	-
DKEFS Interference/Switching Model 2	WTAR	.22	1.59	.120	.14	.10	.10
	BDI-2	-.31	-2.26	.029*			
IGT Net 5 Model 1	WTAR	.27	1.84	.073	.08	.05	-
IGT Net 5 Model 2	WTAR	.26	1.96	.056	.26	.22	.18
	BAI	-.43	-3.25	.002**			
CPT-3 Comissions	WTAR	-.04	-.26	.796	<.01	-.02	-

[a]Multiple hierarchical regression run for DKEFS Color Naming, DKEFS Color-Word Interference, DKEFS Interference/Switching, and IGT Net 5. Hierarchical multiple regression run for CPT-3 Comissions. * significant at $p < .05$. ** significant at $p < .01$.

Table 28

Descriptive Statistics: PTSD History Group and No PTSD History Group

Demographic Factor		PTSD History Group (n = 29) Mean (SD)	No PTSD History Group (n = 31) Mean (SD)	p value, Effect Size
Age		35.90 (10.68)	23.97 (7.51)	p <.001**, d = 1.29
Education		14.45 (1.74)	13.97 (1.40)	p = .273, d = .30
Ethnicity		c^2 (4) = 2.58		p = .631, r = .21
Veteran Status		21 (72.4%)	6 (19.4%)	p <.001**, r = .53
Psychiatric History	MDD	17 (58.6%)	13 (41.9%)	p = .196, r = .17
	GAD	7 (24.1%)	8 (25.8%)	p = .881, r = .02
	Bipolar Disorder	8 (27.6%)	2 (6.5%)	p <.05*, r = .28

* significant at p < .05. ** significant at p < .01

Table 29

Comparison of Psychological Measures: PTSD History Group and No PTSD History Group

Measure	PTSD History Group (n = 29) Mean (SD)	No PTSD History Group (n = 31) Mean (SD)	t statistic (p value, d)
WTAR	101.83 (12.92)	97.68 (12.92)	1.24 (p = .219, d = .32)
BDI-2	30.62 (11.50)	20.16 (11.31)	3.55 (p <.001**, d = .92)
BAI	28.86 (12.00)	17.48 (10.84)	3.86 (p <.001**, d = 1.00)
EPQRA	17.45 (3.86)	15.26 (4.13)	2.12 (p <.05*, d = .55)
THQ	9.83 (3.64)	6.68 (3.72)	3.32 (p <.01**, d = .86)
PCL-5	49.31 (17.03)	32.81 (17.40)	3.71 (p <.001**, d = .96)
CAPS-5 Severity	39.90 (13.28)	23.61 (14.61)	4.84 (p <.001**, d = 1.17)
CD-RISC	58.41 (19.14)	62.68 (16.43)	.93 (p = .357, d = .24)

* significant at p < .05. ** significant at p < .01

Table 30

Descriptive Statistics: PTSD Present Group and PTSD Absent Group

Demographic Factor		PTSD Present Group (n = 36) Mean (SD)	PTSD Absent Group (n = 24) Mean (SD)	p value, Effect size
Age		32.89 (11.10)	25.00 (8.86)	p <.01**, d = .79
Education		14.39 (1.76)	13.92 (1.25)	p = .261, d = .31
Ethnicity		c^2 (4) = 5.82		p = .213, r = -.24
Veteran Status		23 (63.9%)	4 (16.7%)	p <.001**, r = .47
Psychiatric History	PTSD	26 (72.2%)	3 (12.5%)	p <.001**, r = .59
	MDD	20 (55.6%)	10 (41.7%)	p = .292, r = .14
	GAD	8 (22.2%)	7 (29.2%)	p = .543, r = -.08
	Bipolar Disorder	9 (25.0%)	1 (4.2%)	p <.05*, r = .27

* significant at p < .05. ** significant at p < .01

Table 31

Comparison of Psychological Measures: PTSD Present Group and PTSD Absent Group

Measure	PTSD Present Group (n = 36) Mean (SD)	PTSD Absent Group (n = 24) Mean (SD)	t statistic (p value, d)
WTAR	102.11 (12.52)	96.04 (13.07)	1.81 (p = .076, d = .47)
BDI-2	30.17 (10.97)	17.79 (10.96)	4.28 (p <.001**, d = 1.13)
BAI	28.50 (12.23)	14.71 (8.13)	5.25 (p <.001**, d = 1.33)
EPQRA	17.17 (3.65)	15.04 (4.51)	2.01 (p <.05*, d = .52)
THQ	9.97 (3.81)	5.54 (2.48)	5.02 (p <.001**, d = 1.38)
PCL-5	51.53 (13.21)	24.67 (14.43)	7.44 (p <.001**, d = 1.94)
CAPS-5 Severity	41.36 (9.04)	16.67 (9.73)	10.06 (p <.001**, d= 2.63)
CD-RISC	58.22 (19.09)	64.21 (15.25)	1.29 (p = .204, d = .35)

* significant at p < .05. ** significant at p < .01

Table 32

Descriptive Statistics: Civilian and Veteran Groups

Demographic Factor		Civilian Group (n = 33)	Veteran Group (n = 27)	p value, Effect size
Age		M = 22.36, SD = 5.18	M = 38.74, SD = 9.15	p <.001**, d = 2.20
Education		M = 13.64, SD = 1.25	M= 14.89, SD = 1.70	p <.05, d = .84
Ethnicity		c^2 (4) = 2.70		p = .609, r = -.284
Psychiatric History	PTSD	8 (24.2%)	21 (77.8%)	p <.001**, r = .53
	MDD	13 (39.4%)	17 (63%)	p = .07, r = .24
	GAD	12 (36.4%)	3 (11.1%)	p <.05*, r = -.29
	Bipolar Disorder	2 (6.1%)	8 (29.6%)	p <.05*, r = .32

* significant at p < .05. ** significant at p < .01

Table 33

Comparison of Traumatic Event Frequencies: Civilian and Veteran Groups

		Civilian Group Frequency (%)	Veteran Group Frequency (%)	p value, Effect Size
Category of Criterion A Trauma	Sexual Violence	18 (54.5%)	17 (63%)	p = .602, r = .085
	Military Sexual Trauma (MST)	0 (0%)	10 (37%)	p < .001, r = .494
	Combat	0 (0%)	6 (22.2%)	p < .01, r = .369
	Serious Injury/Abuse	4 (12.1%)	3 (11.1%)	p = 1.00, r = -.016
	Life Threat	2 (6.1%)	3 (11.1%)	p = 1.00, r = -.054
	Witnessing Death/Serious Injury/Abuse	6 (18.2%)	0 (0%)	p = .497, r = -.168
	Learning About Traumatic Death	3 (9.1%)	0 (0%)	p = .120, r = -.242
THQ Item (Lifetime History)	Mugging	5 (15.2%)	6 (22.2%)	p = .490, r = .091
	Robbery	8 (24.2%)	11 (40.7%)	p =.184, r = .176
	Break-in while not home	4 (12.1%)	8 (29.6%)	p =.107, r = .218
	Break-in while home	2 (6.1%)	5 (18.5%)	p =.139, r = .193
	Accident (work/car)	7 (21.2%)	17 (63.0%)	p <.01**, r = .424
	Natural Disaster	6 (18.2%)	14 (51.9%)	p <.01**, r = .355
	Man-made disaster	5 (12.1%)	5 (18.5%)	p =.506, r = .089
	Chemical Exposure	0 (0%)	12 (44.4%)	p <.001**, r = .553
	Serious Injury	8 (24.2%)	6 (22.2%)	p =.857, r = -.024
	Fear of being injured/killed	13 (39.4%)	20 (74.1%)	p <.01*, r = .347

(table continues)

		Civilian Group Frequency (%)	Veteran Group Frequency (%)	p value, Effect Size
THQ Item (Lifetime History) *(cont.)*	Witnessing serious injury/someone being killed	12 (36.4%)	15 (55.6%)	p =.142, r = .192
	Seeing dead bodies	9 (27.3%)	15 (55.6%)	p < .05, r = .287
	Loved one killed by drunk driver	6 (18.2%)	9 (33.3%)	p =.193, r = .174
	Spouse/partner/child die	6 (18.2%)	7 (25.9%)	p =.483, r = .094
	Serious or life-threatening illness	4 (12.1%)	6 (22.2%)	p =.317, r = .135
	Receiving news of illness, injury, traumatic death of loved one	25 (75.8%)	20 (74.1%)	p = .883, r = -.019
	Exposure to Combat	1 (3.0%)	12 (44.4%)	p<.001**, r = .500
	Rape	17 (51.5%)	24 (88.9%)	p<.01**, r = .400
	Other sexual assault	18 (54.5%)	22 (81.5%)	p<0.5*, r = .284
	Other sexual contact	7 (21.2%)	12 (44.4%)	p =.061, r = .248
	Attack with weapon	6 (18.2%)	6 (22.2%)	p =.703, r = .050
	Attack without weapon	10 (30.3%)	5 (18.5%)	p =.294, r = -.135
	Abuse by family member	11 (33.3%)	10 (37.0%)	p =.769, r = .039
	Other	16 (48.5%)	10 (37.0%)	p =.382, r = -.115

* significant at p < .05. ** significant at p < .01

Table 34

Comparison of Psychological Measures: Civilians and Veterans

Measure	Civilian Group Mean (SD)	Veteran Group Mean (SD)	t statistic (p value, d)	p value, d
WTAR	96.76 (12.86)	103.26 (12.44)	1.98	p = .053, d = .51
BDI-2	21.64 (11.83)	29.59 (12.02)	3.57	p <.05*, d = .67
BAI	19.61 (12.63)	27.11 (11.70)	2.37	p <.05*, d = .62
EPQRA	15.73 (3.74)	17.04 (4.51)	1.23	p = .223, d = .32
THQ	6.52 (3.62)	10.26 (3.44)	4.08	p <.001**, d=1.06
PCL-5	34.94 (18.98)	47.93 (16.71)	2.78	p <.01**, d = .73
CAPS-5 Severity	24.39 (14.70)	38.48 (13.48)	3.50	p <.01**, d = 1.00
CD-RISC	63.33 (13.84)	57.30 (21.44)	1.26	p = .213, d = .33

* significant at p < .05. ** significant at p < .01

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