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An Intergenerational Examination of Posttraumatic Stress Disorder and Intelligence

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B. A., University of Arkansas, 2006

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Abstract

An Intergenerational Examination of Posttraumatic Stress Disorder and Intelligence

By Dorthie Sue Cross Mokdad

Individuals with posttraumatic stress disorder (PTSD) perform poorer on tests of intelligence than do individuals without PTSD, but the meaning of this finding is controversial. What continues to spark debate is whether either or both variables have a causal impact on the other and whether other factors better explain group differences. One such set of factors may be intergenerational effects of trauma on both cognitive development and risk for PTSD. This dissertation is comprised of three chapters. The first chapter presents three candidate theories to explain the relationship between intelligence and PTSD, namely a *social resource theory*, a *plasticity theory*, and a *heritability theory*. The second chapter presents a quantitative and qualitative review of 29 cross-sectional and 9 prospective studies, demonstrating significant deficits of intelligence, with perhaps more pronounced difference in analytical and analogical reasoning in individuals with PTSD. Results suggested that intelligence may be both an influencing factor prior to traumatic exposure and be impacted following it, supporting both the heritability and the plasticity theories. The third and final chapter presents findings from a cross-sectional study of mother-child pairs, examining intergenerational influences of maternal trauma, PTSD, and parenting (i.e., child abuse potential, overreactivity, and laxness) on the relationship of intelligence to PTSD in children. Participants were 48 mothers and 55 children (29 girls, 26 boys), ages 8 to 12 years, recruited from clinic waiting rooms of a public hospital. Mothers with PTSD performed poorer on a measure of analogical reasoning but, unexpectedly, demonstrated an advantage for nonverbal recognition, even accounting for trauma and other psychopathology. For children, lower verbal intelligence was associated with more frequent symptoms of PTSD, even accounting for child trauma and psychopathology; however maternal factors (i.e., maternal intelligence, trauma, PTSD, and parenting) did not predict child outcomes. Although evidence of an intergenerational impact of intelligence on risk for PTSD was not directly observed in this study, likely due to small sample size, other studies should consider the role of parenting on the relationship between children's intelligence and symptoms of PTSD.

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Dedication

To Ruthie, Stephanie, Heidi, and Teddy, my sisters and brother, my best friends, for being the most wonderful group of people with whom to share this life. You are the true diamonds of Arkansas.

To my father for your quiet strength, self-sacrifice, and steady moral compass. I have always and will always so sincerely look up to you.

To Ali, my husband, for accepting and embracing me as a whole person and for demonstrating genuine kindness toward all people. You are a rising and joyful sun, warming all those fortunate enough to be near you.

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Both Kerry Ressler, MD, PhD, and Bekh have opened amazing doors for me, allowing me the chance to join the Grady Trauma Project as a graduate practicum student, hiring me as a graduate fellow, hiring me as the project coordinator for Moms and Kids project, securing funding for me both internally and externally, and writing numerous letters of reference over the many years I have known them. Now, Kerry and Bekh sit on my dissertation committee, supporting me once again. I cannot express how deeply grateful I am for everything they have done for me.

The remaining members of my dissertation committee have been great sources of support and influence, even outside of their roles on this committee. Linda Craighead, PhD, has demonstrated strength and clarity in her role as Director of Clinical Training, spearheading important and positive changes to the program, while also managing the emotions of many hysterical graduate students—myself included. Under her leadership, graduate school—and the path to this dissertation—became clearer.

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This dissertation reflects the incredible support I am fortunate enough to have all around me. I am so thankful for each and every person—mentioned here or not—who has helped me on this journey. I could not have done it alone.

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Chapter I: Background and Theoretical Development

Exposure to traumatic events is not uncommon in the general population, affecting approximately 60 to 65% of individuals over the lifespan (Breslau et al., 1998; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Despite high rates of exposure, the development of posttraumatic stress disorder (PTSD) is much less common, occurring in 5-6% of men and 10-14% of women (Breslau, 2002). Efforts to identify risk factors for the development of PTSD following trauma are increasing and are largely driven by the recognition that PTSD is a costly illness, both to the individual and to society. An individual diagnosed with PTSD is more likely to be unemployed, utilizing health care services, abusing substances, and involved in the criminal justice system (Donley et al., 2012; McFarlane & Papay, 1992; Nandi et al., 2004; Richardson, Elhai, & Pedlar, 2006). Furthermore, individuals with PTSD are more likely to engage in partner maltreatment, as well as other forms of interpersonal violence (Marshall, Panuzio, & Taft, 2005), such that PTSD in one individual potentially begets PTSD in others. In a more concrete estimate of the impact of PTSD, Harrison, Satterwhite, and Ruday (2010) estimate that the cost of treating just PTSD among veterans returning from Iraq and Afghanistan to be over $200 million annually. This estimate does not include disability compensation or secondary costs. Identifying risk factors for PTSD may ostensibly lead to earlier intervention through empirically-based allocation of mental health resources for high-risk individuals following trauma, which may result in shortened illness duration and reduced impairment.

PTSD is the result of an ability to recover psychologically following exposure to a traumatic event and is characterized by intrusive re-experiencing, avoidance of reminders, and physiological hyperarousal. See Table 1 for a list of symptoms based on the *Diagnostic and Statistical Manual of Mental Disorders-IV-TR* (American Psychiatric Association, 2000). Traumatic experiences include situations during which an individual is threatened with the possibility of death or serious physical injury or threat to physical integrity (e.g., sexual assault), witnesses others in such situations, or is confronted with the news that others have experienced these situations (e.g., learning that a loved one was murdered). After experiencing a traumatic event, most people associate certain characteristics of the environment with the potential for future harm, and as a result, demonstrate a heightened fear response to those characteristics. This is classical conditioning. Over time, however, some individuals learn that all trauma-related cues do not signal actual threat, and their fear responses are extinguished. In individuals who develop PTSD, however, extinction processes are impaired. Cognitive-behavioral theories of PTSD emphasize the role of failed extinction, persistent cognitive and behavioral avoidance, and overly-inclusive fear structures in the development and maintenance of the disorder (Foa & Kozak, 1993).

Despite the fact that PTSD results from the experience of an event external to the individual, the risk of developing the disorder is influenced by both external factors (e.g., social support; Dalgleish, Joseph, Thrasher, Tranah, & Yule, 1996) and internal factors (e.g., genetic vulnerability; Afifi, Asmundson, Taylor, & Jang, 2010), including strong evidence for a genetic model of risk for not only the development of PTSD, but also for exposure to assaultive trauma (Stein, Jang, Taylor, Vernon, & Livesley, 2002). Stein et

al. (2002) hypothesized that underlying traits (e.g., neuroticism) can predispose an individual both to certain behaviors that may increase risk for trauma (e.g., irritability and increased physical aggression) and to poor subsequent coping (e.g., avoidance). Notably, the genetic risks for PTSD and traumatic exposure appear to be separate (Roy-Byrne, Arguelles, Vitek, Keane, & Pitman, 2004).

One such underlying factor that may be related to increased risk of PTSD following trauma is a deficit in cognitive abilities (e.g., memory and intelligence) for individuals with PTSD relative to individuals without PTSD (Brewin, Andrews, & Valentine, 2000; Brewin, Kleiner, Vasterling, & Field, 2007; Johnsen & Asbjørnsen, 2008).

Intelligence

Broadly speaking, intelligence is the ability or affords the ability to adapt to one's environment—to purposefully, rationally, and effectively problem-solve as one engages the world (Sternberg, 1997). More narrowly speaking, much of the current conceptualization and measurement of intelligence continues to draw on the works of Spearman and Cattell, among others. Spearman (1927) noted that an individual who performed well on one task tended to perform well on other tasks, even if the tasks themselves were not similar. Spearman theorized that a common factor was shared for performance on many, seemingly unrelated cognitive tasks, and he termed this factor the general intelligence factor, or *g*. *g* itself is comprised of a multitude of lower-level, specific abilities, or *s*. Multiple tests of different *s*, therefore, could be administered to approximate *g*. Cattell (1963) would later conceptualize intelligence as being comprised of two principal factors—fluid intelligence (*gf*) and crystallized intelligence (*gc*).

According to Cattell, *gf* is largely a product of biology and declines over time, and *gc* is more environmentally-influenced through educational opportunity and cultural experiences and may increase as one ages.

Relationship between Intelligence and Risk for PTSD

Intelligence may be an influencing factor in resilience against adversity and the development of psychopathology more generally. Fergusson and Lynskey (1996) found in a New Zealand birth cohort that two factors, high intelligence at age 8 and less exposure to family adversity, predicted resilience at age 19, which they defined as having fewer delinquent friends and engaging in less novelty seeking. In addition, several other studies report that relatively lower intelligence in childhood predicts later development of a wide range of disorders, including schizophrenia, depression, and anxiety disorders (Batty, Mortensen, Nybo Anderson, & Osler, 2005; Hatch et al., 2007; Kandel et al., 1988; Koenen et al., 2009; Martin et al., 2007; Zammit et al., 2004).

High intelligence may generally confer upon an individual an enhanced capacity for adapting to stress. Koenen et al. (2009) proposed that the relationship between intelligence and psychopathology can be explained using the cognitive reserve theory. This theory points to the fact that equivalent brain disease and damage do not result in equivalent levels of dysfunction (Cosentino & Stern, 2013). Thus, some other factor, such as cognitive reserve, must be buffering against these insults. Cognitive reserve refers to the brain's ability to actively compensate to reduce the impact of disease or injury and may be partly related to factors such as brain volume or synaptic density (Stern, 2009). According to this theory, cognitive reserve can deplete over time, allowing for pre-existing problems to become more obvious. Intelligence, itself, may be an important

aspect to or indicator of cognitive reserve (Plassman et al., 1995; Schmand, Smit, Geerlings, & Lindeboom, 1997).

The idea that PTSD is related to intelligence—that individuals with PTSD demonstrate poorer performance on tests of intelligence relative to those without PTSD—is supported by a number of studies, though the meaning of the relationship is controversial. What continues to spark the most debate is whether either or both variables have a causal impact on the other. Does PTSD negatively impact a person's performance on measures of intelligence, or does lower pre-exposure intelligence increase risk for developing PTSD? Several reviews have addressed the relationship between PTSD and cognitive functioning. Two meta-analytic reviews focused primarily on memory found that PTSD is associated with relative impairments in verbal memory and, to a lesser extent, visual memory (Brewin et al., 2007; Johnsen & Asbjornsen, 2008). Three reviews have addressed intelligence as parts of larger reviews of risk factors for PTSD (Brewin et al., 2000; Buckley, Blanchard, & Neill, 2000; Moore, 2009), and the authors of these reviews conclude that intelligence predicts, perhaps causally so, the development of PTSD.

Beyond a broad invocation of the cognitive reserve theory, exactly why individuals with PTSD perform worse on measures of intelligence—and whether PTSD is associated with a particular pattern of intellectual deficits—remains unclear. Three potentially promising theories are presented here: a social resource theory, a plasticity theory, and a heredity theory.

But first, a very basic explanation of the relative poor performances of individuals with PTSD is that some of the symptoms, such a difficulty concentrating, hypervigilance,

poor sleep, and intrusive memories, result in state-based impairments that can impact test performance in such a way that reflects no true underlying trait deficit. In other words, once the symptoms resolve, differences in test performance should disappear completely. Although such symptoms do impact cognitive performance (e.g., Hauri, 1997), prospective studies of group differences in intelligence prior to symptom onset (e.g., Macklin et al., 1998) make this explanation difficult to defend. The following theories may better characterize the complex and multi-dimensional nature of the relationship between PTSD and intelligence.

Social Resource Theory

Because both exposure to trauma and lower average intelligence are overrepresented in populations lacking social and economic resources, the relationship between intelligence and PTSD may be, at least in part, the result of an individual's environment, rather than a direct relationship between the two variables. Socioeconomic factors appear to impact intelligence in ways that are not predicted by genetic theory alone. Estimates of intelligence are lower and may be less heritable under strained socioeconomic conditions (Tucker-Drob, Rhemtulla, Harden, Turkheimer, & Fask, 2011; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003), and aspects of intelligence can be enhanced through better nutrition or impaired at even low-level exposure to environmental toxins such as lead (Eysenck & Schoenthaler, 1997; Koller, Brown, Spurgeon, & Levy, 2004). Moreover, intelligence can be deliberately increased as a result of more favorable educational placement (Härnqvist, 1968), and some researchers posit that gains in population intelligence scores over the last century are largely attributable to widespread access to early and more rigorous schooling (Blair, Gamson,

Thorne, & Baker, 2005). Although research supports a strong genetic contribution to intelligence, there nonetheless appears to be enough "wiggle room" to allow for influence by other variables (e.g., shared environment), particularly in low-SES individuals.

PTSD is likewise related to SES in that frequency of traumatic exposure and risk of PTSD tend to be higher in low-income, urban populations (Breslau, 2002). This increased trauma is not limited to violence. Even natural disasters result in more significant negative impact and barriers to recovery in low-SES individuals (Laska & Morrow, 2006). For survivors of Hurricane Katrina, for example, communities remained devastated years after the actual natural disaster and, along with discrimination and other factors, contributed to sustained mental health problems (Weems et al., 2010). Notably, some studies find a higher rate of PTSD among African Americans and other minority groups relative to white groups; however, these differences may be accounted for by SES, trauma severity, the experience of multiple traumas, and living in an urban environment (Breslau et al., 1998; Green et al., 1990; Kulka et al., 1990).

The resource theory, like the cognitive reserve theory, does not offer strongly specific predictions of particular patterns of deficits that may be observed in individuals with PTSD, though theory supports a greater relative impact of poverty on verbal intelligence compared to nonverbal intelligence. Crystallized intelligence, often operationalized as verbal intelligence, is considered more environmentally-influenced (Cattell, 1963). Therefore, if the relationship between intelligence and PTSD is due, in part, to the impact of SES, one might predict that individuals with PTSD would show greater relative deficits for verbal intelligence.

Plasticity Theory

A wide range of early environmental insults to brain development may impact both intelligence and the risk for PTSD. Therefore, according to the following theory, relative differences in intelligence between people with and without PTSD may be the result of early alterations to a shared substrate or functional system. Because the developing brain is particularly sensitive to environmental input, the impact of early adversity may be especially critical (Cicchetti & Tucker, 1994; Nelson & Carver, 1998). In the bioecological model of intelligence (Ceci, 1996; Davidson & Downing, 2000), how a particular biologically-predisposed cognitive ability develops is shaped by a child's context, which includes environmental factors and the child's individual traits. That ability, in turn, influences the child's context in an ongoing, iterative process such that relatively small changes either the context or the ability result in a cascade of other small changes, eventually leading to large changes in the development of that ability. In this way, even relatively minor insults to a developing brain may result in more significant and long-lasting consequences.

Several researchers posit that early exposure to extreme stress can result in alterations to normal brain development, which subsequently lead to lower intelligence (Cicchetti & Walker, 2001; De Bellis, 2001; Perry, 1997; Sanchez, Ladd, & Plotsky, 2001; Sapolsky, Uno, Rebert, & Finch, 1990). Koenen, Moffitt, Caspi, Taylor, and Purcell (2003) found in a study of children exposed to domestic violence that witnessing domestic violence accounted for 10.6% of the variance in intelligence for monozygotic (MZ) and dizygotic (DZ) twins, independent of genetic effects, and as severity of violence increased, children's intelligence decreased. In addition, neuroimaging studies of maltreated children have found structural differences in maltreated children compared

to their non-maltreated peers. De Bellis et al. (1999) found that for maltreated children with PTSD, duration of maltreatment was negatively correlated with intracranial volume; notably, intracranial volume was also positively correlated with intelligence in this sample. The results of a later study, which found evidence of neuronal loss in maltreated children, suggest that lower intracranial volume may be the result of such neuronal loss (De Bellis, Keshavan, Spencer, & Hall, 2000). Differences in intelligence for PTSD patients could be a result of the phenomena described in these studies (Mash & Barkley, 2003). In this respect, one could argue that early insults result in diminished cognitive reserve, leaving the individual more vulnerable to negative sequelae following future insults. This view is consistent with research that demonstrates that exposure to assaultive trauma in childhood increases the risk for PTSD following trauma in adulthood (Breslau, Chilcoat, Kessler, & Davis, 1999).

Studies suggest that neuronal loss may be partly caused by chronic exposure to high levels of glucocorticoids (Tata & Anderson, 2010). For example, prenatal exposure to elevated levels of cortisol is associated with lower intelligence at age seven years independent even of postnatal family environment (LeWinn et al., 2009). Many studies demonstrate an association between high cortisol levels and hippocampal volume (Sapolsky, Uno, Rebert, & Finch, 1990; Uno et al., 1994). The hippocampus is important for memory encoding, storage, and retrieval, as well as for spatial reasoning (Squire & Cave, 1991). Deficits in such abilities are likely to negatively impact performance on tests of intelligence.

However, implicating cortisol as a possible agent in the relationship between intelligence and PTSD appears to contradict a plethora of research demonstrating *lower*

levels of glucocorticoids in individuals with PTSD relative to healthy controls. PTSD is associated with increased sensitization of the HPA axis (i.e., a more immediate "off switch"), which results in lower levels of cortisol, as well as other hormones (Yehuda et al., 1995). Pregnant mothers with PTSD show low cortisol levels, as do their eventual infants (Yehuda et al., 2005). For children, however, a different pattern is reported. Following trauma, cortisol levels are positively correlated with PTSD symptom severity in children (Cicchetti & Rogosch, 2001; De Bellis et al., 1999; Delahanty, Nugent, Christopher, & Walsh, 2005). These disparate findings may suggest that the role of cortisol in the risk for PTSD is different across development, and the nature of the relationship between PTSD and intelligence could be different at different points in time for an individual, depending on the timing and chronicity of stress.

Glucocorticoids, of course, do not behave in a vacuum. In a review of hypocortisolism studies, Fries, Hesse, Hellhammer, and Hellhammer (2005) point to evidence that stress is initially associated with an increase in glucocorticoid release via HPA axis activation, but after chronic activation, the system will become increasingly sensitized and will begin to down-regulate, leading to reduced glucocorticoid release. Such down-regulation is associated with increased inflammatory response, which is, itself, associated with poor cognitive functioning (e.g., poor attention, executive function, and verbal memory) in adults (Marsland et al., 2006).

Beyond the cortisol debate, the idea that trauma changes brain functioning continues to find support. A translational model based on stress-induced adult rats found that exposure to stress resulted in gene methylation specific to the hippocampus that may help to explain persistent hippocampal dysfunction in trauma-exposed humans (Roth,

Zoladz, Sweatt, & Diamond, 2011). Additionally, in a longitudinal study of veterans before, 1.5 months after, and 1.5 years after deployment to Afghanistan, van Wingen et al. (2012) found that exposure to combat resulted in reduced midbrain activity and integrity 1.5 months after returning from combat, which was related to deficits in sustained attention. Moreover, combat exposure was related to reduced functional connectivity between the midbrain and lateral prefrontal cortex, and, although midbrain activity and integrity returned to baseline after 1.5 years, functional connectivity remained reduced. Clearly, trauma can have a lasting effect on brain functioning, and the plasticity theory suggests that such exposure may impact brain functioning in a way that simultaneously impairs performance on intelligence tests and also leaves an individual more vulnerable to PTSD. Furthermore, risk and resilience may be continuously evolving constructs with factors like intelligence and cognitive reserve ebbing and flowing over the lifespan.

Because hippocampal changes are implicated in a large number of studies examining the impact of stress on cognitive functioning, it is reasonable to expect that if individuals with PTSD demonstrate relative deficits in cognitive abilities related to hippocampal functioning (e.g., memory and spatial reasoning), such a finding would offer support for the plasticity theory; however, because poverty, itself, may produce the kind of stress response capable of impacting hippocampal function (Hanson, Chandra, Wolfe, & Pollak, 2011), findings supporting relative deficits in abilities like memory and spatial reasoning, in conjunction with weaker findings for verbal deficits, may be stronger—and more specific—support for the plasticity theory.

Heritability Theory

Both intelligence (Plomin & Kovas, 2005) and PTSD (Afifi et al., 2010; Binder et al., 2008) are highly heritable. This theory, like the previous theory, points to structural and functional differences in the brains of individuals with and without PTSD; however, unlike the previous theory which interprets differences as indicators of trauma- and PTSD-induced alterations, this theory posits that that differences observed between individuals pre-date the occurrence of trauma and PTSD and may represent a risk for both developing PTSD and for performing poorly on measures of intelligence.

Studies demonstrate that some potential sources of relative cognitive deficits are not attributable to PTSD, but rather pre-existing differences in brain structure and function (Gilbertson et al., 2002; McNally, 2003). For example, hippocampal volume is equivalent in PTSD discordant MZ twins, suggesting that reduced hippocampal volume is not trauma- or PTSD-induced, but rather that it represents pre-existing deficits that may indicate increased risk for PTSD and lower intelligence. In fact, in the general population, normal variations in the volume of several brain structures, including the hippocampus, are correlated with intelligence in the absence of trauma (Andreasen et al., 1993). Furthermore, one study found that risks for general psychopathology and low intelligence were distinctly and separately heritable in youth, but a specific risk for both low intelligence and psychopathology together was noted for some youth (Jacobs, Rijsdijk, Derom, Danckaerts, Thiery, Derom, et al., 2002). Thus, it is possible that intelligence and PTSD are related as a result of pre-existing, heritable variations in brain structures or systems. In keeping with the cognitive reserve model, proponents of the heritability theory might argue that although neurocognitive insults may, indeed, reduce reserve, the initial reserve, itself, is largely genetically determined.

This theory does not necessarily offer specific predictions for potential patterns of deficits. If relative deficits—whether for verbal or spatial abilities or for memory—exist prior to traumatic exposure, the theory is supported.

The Role of Memory

Much like general intelligence, memory has been implicated as a neurocognitive marker of PTSD, and the literature appears to indicate greater consensus. Buckley et al.'s (2000) review supported the idea that PTSD is associated with broad deficits in memory, and Elzinga and Bremner's (2002) review pointed particularly to relative deficits in declarative and working memory with likely origins in dysfunction of the hippocampus and prefrontal cortex, respectively.

The relationship between memory and intelligence remains a topic of debate, particularly regarding whether working memory and general intelligence are separable (see Ackerman, Beier, & Boyle, 2005, and Oberauer, Schulze, Wilhelm, & Suss, 2005, for competing views). Nevertheless, major theories of intelligence, including the Cattell-Horn-Carroll theory of cognitive abilities, incorporate short-term memory as one of the broad abilities comprised by *g* (McGrew, 2009).

Although this dissertation focuses primarily on intelligence, it will be important to consider the impact of memory on potential group differences in intelligence in light of the robust findings regarding deficits in memory in individuals with PTSD and the theoretical and empirical overlap of memory and intelligence.

Intergenerational Risk for Traumatic Exposure and PTSD

A robust body of literature supports the idea that the offspring of parents with PTSD are at higher risk for a range of psychopathology, including PTSD, other anxiety

disorders, depression, and suicidality (Rosenheck & Fontana, 1998; Yehuda, Bell, Bierer, & Schmeidler, 2008; Yehuda, Halligan, & Bierer, 2001). Moreover, even when controlling for trauma to the offspring themselves, parental PTSD continues to be an important predictor of PTSD in the next generation (Yehuda, Halligan, & Bierer, 2001).

The mechanisms of transmission are potentially many and include genetic, epigenetic, and behavioral pathways. Clearly, genetic risk in one generation will confer risk in the next, and several studies highlight that risk is not a straight path, but rather a coming together of factors, potentially as a result of epigenetic process (Afifi et al., 2010; Binder et al., 2008; Xie et al., 2010). Parental PTSD predicts low cortisol and increased glucocorticoid sensitivity in offspring (Yehuda et al., 2000; Yehuda, Halligan, & Grossman, 2001; Yehuda, Schmeidler, Wainberg, Binder-Brynes, & Duvdevani, 1998), and this effect is more pronounced for the offspring of mothers with PTSD, relative to fathers with PTSD (Yehuda, Bell, Bierer, & Schmeidler, 2008).

Despite the stronger effect for mothers regarding offspring HPA functioning, PTSD in both mothers and fathers has been shown to impact children (Davidson, Smith, & Kudler, 1989; Jordan et al., 1992; Rosenheck & Fontana, 1998; Ruscio, Weathers, King, & King, 2002; Yehuda et al., 2001), potentially via parenting style and behavior. Male veterans with PTSD demonstrate serious deficits in parenting skills (Jordan et al., 1992) and more emotional numbing toward their children (Ruscio et al., 2002). Women with PTSD are more likely to engage in an overreactive parenting style (Ortigo, Guarnaccia, Ortigo, Castleberry, Johnson, Pierre, Bradley, & Ressler, 2008), and women who have experienced abuse as children demonstrate increased child abuse potential (Smith, Cross, Winkler, Jovanovic, & Bradley, in press). Animal models of

intergenerational stress demonstrate that maternal grooming behavior impacts the genetic expression of factors important to stress response, including hippocampal functioning, in the offspring (Francis, Diorio, Liu, & Meaney, 1999; Liu, Diorio, Day, Francis, & Meaney, 2000). Considering both human and animal studies, PTSD in one generation may increase trauma in the next and at the same time impair coping strategies and biological stress response, leaving that generation more vulnerable to the development of PTSD.

The Role of Intelligence in Intergenerational Transmission of PTSD

Intelligence may be an important factor in the intergenerational transmission of the risk for trauma and PTSD both in parents and children. Like with PTSD, parents with relative deficits in intelligence may both pass on low intelligence to their children via genetic heritability and engage in parenting behaviors that limit child cognitive development. Previous research indicates that impaired memory performance is related to harsh reactive parenting (Deater-Deckard, Sewell, Petrill, & Thompson, 2010) and that impaired verbal intelligence performance is related to child abuse potential (Ammerman & Patz, 1996). In addition, parental intelligence is negatively related both to parenting stress and to child behavior problems (Kwon, 2007). Children of parents with relative deficits in intelligence may simultaneously be exposed to more stress and trauma and inherit an increased risk for developing PTSD, i.e., low intelligence. Nevertheless, the role of intelligence in the intergenerational transmission of risk is not understood and has not been addressed in the literature. Such a role, if any, should be explored.

Current Projects: Purpose and Structure

The remainder of this dissertation is divided into two chapters dedicated to addressing several questions that have arisen through the preceding examination of the general body of literature regarding intelligence and PTSD. The next chapter attempts to answer or more fully explore the following questions: 1) Does intelligence differ between individuals with and without PTSD? 2) If so, what is the magnitude of that difference? 3) Do other factors (e.g., SES, trauma severity, working memory, etc.) better explain such a group difference? And, 4) does that difference precede traumatic exposure?

The third and final chapter focuses on a cross-sectional study of mother-child pairs, and the primary goal of the study is to examine whether factors in one generation impact the relationship between PTSD and intelligence in the next? Separate analyses for mothers and children will also be conducted. Examining the impact of trauma across generations may further enrich current understanding of PTSD and intelligence and, depending on the results, may broaden the temporal scope of the debate regarding causality.

Chapter II: Literature Review

The goals of the review are, first, to quantitatively review retrospective studies measuring PTSD and intelligence in order to obtain an estimate of group differences, as well as allow for additional examination of potential moderators, and, second, to qualitatively review prospective studies as part of an overall effort to examine causality.

The quantitative review examines group differences—and the magnitude of differences—in intelligence between individuals with and without PTSD and also explores other potentially important factors via moderator analyses. Moderators for the quantitative review were chosen if they had been consistently implicated as risk factors for PTSD (Brewin, Andrews, & Valentine, 2000), if they could affect the way intelligence was measured (e.g., using varying number of IQ subtests), or if they could result in confounding interpretations (e.g., the use of traumatized versus non-traumatized comparison groups).

The qualitative review focuses on prospective studies—studies measuring intelligence prior to traumatic exposure and PTSD symptom onset—to attempt to address causality. Does trauma or PTSD negatively impact a person's performance on measures of intelligence, or does lower pre-exposure intelligence increase risk for developing PTSD?

The answer to this question is important not only theoretically, but also practically. If intelligence is, indeed, a pre-trauma risk factor for PTSD, it becomes an inexpensive indicator of the potential need for mental health resources and early intervention as described above. If, on the other hand, PTSD leads to impaired

performance on measures of intelligence, the focus shifts toward treating those individuals already diagnosed in hopes of restoring previous levels of functioning.

Method

This review focuses on both retrospective studies, which constitute the bulk of the meta-analysis, and prospective studies. The prospective studies are largely addressed qualitatively. Retrospective studies were reviewed using meta-analytic techniques described in Lipsey and Wilson (2001) and Borenstein, Hedges, Higgins, and Rothstein (2011). Meta-analytic techniques supplement qualitative analyses and allow for statistical aggregation of findings across studies. Such an aggregation is made possible by creating a standardized mean effect size based on the effect sizes of individual tests in the studies comprising the review. The effect size statistic is used to describe both the magnitude and the direction of an effect and is less affected by factors such as sample size than other often-used statistics (e.g., *p*-values).

Because most relevant studies report data in the form of raw group differences (i.e., means and standard deviations), the effect size *d* was determined to be the most appropriate statistic, and for the minority of studies reporting correlation coefficients, the *r* statistics were converted to *d* via Fischer's *Z* transformation and included in meta-analysis. Because studies of smaller samples tend to display an upward bias (i.e., inflated effect size), all effect sizes included in the meta-analysis were adjusted using Hedge's *g*, based on Borenstein et al.' s (2011) recommendation. For the actual meta-analysis, adjusted effect sizes were combined, weighted by their respective variances, yielding a single effect size representing the magnitude of the observed difference in intelligence between individuals with and without PTSD.

Search Strategy

All studies were located via systematic search of relevant literature published since 1990. Electronic databases (specifically *Google Scholar*, *PsycInfo*, and *PubMed*) were the primary search tool, using the following search terms: *PTSD*, *posttraumatic*, *trauma*, *cognitive*, *intelligence*, and *IQ*. In addition, reference sections of recent articles, as well as reviews on adjacent topics (e.g., memory deficits in PTSD), were used to identify additional studies. Most studies included in the review examine intelligence as a primary variable of interest; however, some studies included a measure of intelligence as part of an examination of another variable. These studies were still included in the broader review as long as intelligence was not used to match groups (i.e., participants were not chosen in such a way that intelligence would be deliberately equal) and also in the meta-analysis if an effect size could be computed. Despite careful searches, some appropriate studies were likely missed and, thus, not included in this review; however, it is unlikely that any omission represents a systematic bias, and to test for potential sampling bias, Orwin's (1983) Fail Safe *N* will be computed. Orwin's formula is an estimate of the number of unpublished or otherwise excluded studies with effect sizes of zero needed to reduce the observed mean effect size to a chosen criterion value (e.g., a Cohen's *d* of .20, representing the lower bounds of a small effect; Cohen, 1988). This formula is more conservative than Rosenthal's (1979) formula.

Inclusion/ Exclusion Criteria.

Studies were included in the meta-analysis if they were published in English, if PTSD was assessed formally with a questionnaire or clinical interview, if a measure of intelligence was included, if participants were not matched on measures intelligence, if

results were based on samples not already reported in another included study (i.e., if samples were independent), and, finally, if enough data were available to calculate an effect size. Geyskens, Krishnan, Steenkamp, and Cunha (2009) addresses the problem of interdependent effect sizes (in this case multiple studies reporting on the same sample) by suggesting studies either be combined or a single one chosen, though they emphasize that choosing a study could lead to unintended bias. Keeping in mind Geyskens et al.'s caution, single studies were chosen on the *a priori* basis of which study provided the most information for subsequent moderator analyses, or failing a difference there, the most participants.

For this review, a few studies included both a traumatized and non-traumatized comparison groups, and traumatized comparison groups were chosen as the comparison group for the meta-analysis because such groups theoretically will better inform the question of whether group differences in intelligence for individuals with and without PTSD is related to PTSD itself—and not just trauma. In addition, some studies included both healthy and psychiatric comparison groups, and this review focused primarily on healthy comparisons in those cases. One prospective study initially included a psychiatric comparison only for pre-trauma analyses and added a healthy comparison at later post-trauma time points. For this and other studies using psychiatric comparison groups only, psychiatric comparisons were included in the review.

The qualitative review addresses studies for which an effect size cannot be computed (e.g., studies using regression analyses or analyses controlling for other variables while not reporting unadjusted data). In addition, whereas the meta-analysis addresses only the effect size for intelligence measured *after* traumatic exposure, the

qualitative review includes prospective studies of pre- and post-exposure measurements. For prospective studies measuring pre- and post-exposure intelligence, the post-trauma cognitive ability effect size was included in the meta-analysis. One study (Koenen, Moffitt, Poulton, Martin, & Caspi, 2007) included two follow-up time points, and only the first one was included in the meta-analysis because it was composed of more participants.

Study sample.

The search strategy and the inclusion/ exclusion criteria yielded 38 independent studies for the overall review, 29 (N = 2305) of which are included in the meta-analysis and the remaining 9 (N = 8723) addressed qualitatively. Descriptions of studies included in the current review are presented in Table 2.

Meta-analysis

To test the main hypothesis that intelligence is lower for individuals with PTSD relative to individuals without PTSD, a fixed-effect meta-analysis was conducted. A fixed-effect model was chosen based on the previous overview of PTSD and intelligence more broadly, which suggests that between studies variability may be the result of study-specific factors (e.g., inclusion of test of memory, veteran vs. civilian sample).

This meta-analysis includes independent effect sizes reflecting differences in verbal intelligence (VIQ), nonverbal intelligence (NIQ), or combined (verbal and nonverbal) full scale intelligence (FSIQ). If studies reported FSIQ alone or reported only VIQ or only NIQ, those statistics were included in the meta-analysis. If FSIQ was reported alongside other factors, whether VIQ or NIQ, only FSIQ was included in the

meta-analysis. If studies reported both VIQ and NIQ with no FSIQ, VIQ and NIQ were combined statistically by averaging the effect sizes (Lipsey & Wilson, 2009).

Additional meta-analyses were conducted for VIQ and NIQ separately. The results of these analyses are not independent of the primary meta-analysis, nor of each other, as data were extracted from overlapping studies. Subsequent moderator analyses are based on the primary meta-analysis alone.

Conceptual moderators. Conceptual moderators comprised two main categories: sample characteristics (i.e., age, sex, race/ ethnicity, level of education, SES, and veteran status) and trauma-related characteristics (e.g., PTSD symptom severity, trauma severity, type of index trauma, and whether index trauma occurred in adulthood). Age, sex, race/ ethnicity, level of education, SES, PTSD symptom severity, and trauma severity were evaluated using continuous data, and veteran status, type of trauma, and whether the trauma occurred in adulthood were evaluated categorically.

Methodological moderators. It is possible that effects sizes could differ across studies due to factors related to study design. Moderator analyses were performed to assess the impact of the exclusion/ non-exclusion of trauma exposure and psychiatric disorders in comparison groups. Moderator analyses were also conducted for the inclusion/ non-inclusion of working memory subtests, and number of separate IQ subtests administered. Number of separate IQ subtests administered was evaluated using continuous data, and all other methodological moderators were evaluated categorically. Because many studies simultaneously excluded head injury, neurological problems, psychosis, severe substance dependence, etc., conclusions about the individual effects of such exclusions cannot be drawn and were not included in the analyses.

Continuous moderator analyses were conducted using weighted least squares regression, weighting effect sizes by the inverse of their variances, and categorical moderator analyses were conducted using an analog to the ANOVA procedure. The analog to the ANOVA groups effect sizes categorically based on an independent variable (e.g., veteran vs. civilian) and, like a standard ANOVA, tests the significance of between group variance. Both analyses are described by Lipsey and Wilson (2001).

Hypotheses

Based on the broader overview of PTSD, intelligence, and the relationship of intelligence to psychopathology more generally, the following hypothesis are presented: 1) Individuals with PTSD will demonstrate a relative deficit in intelligence compared to individuals without PTSD, 2) the magnitude of that group difference will be comparable the magnitude of differences observed for memory deficits in other studies (i.e., $d = .74$ reported in review by Johnsen and Asbjornsen (2008), 3) group differences will precede traumatic exposure, and 4) other factors will impact the magnitude of group differences.

Support for the resource theory will be found in moderator analyses of SES, education, and race/ ethnicity. Previous studies suggest that risk for developing PTSD is greater among non-white individuals (Breslau et al., 1998; Green et al., 1990); however, because this effect was eliminated when controlling for urban residence, race/ ethnicity—if a significant moderator—may be interpreted as an indicator of an effect of social context or SES. In addition, because verbal intelligence is theoretically more sensitive than nonverbal intelligence to environmental factors (Cattell, 1963), finding that individuals with PTSD demonstrate relatively greater deficits verbal intelligence, compared to nonverbal intelligence, may support the resource theory.

Support for the plasticity theory will be found in moderator analyses of trauma severity, whether the index trauma occurred in childhood/ adolescence or adulthood, and whether healthy comparison groups were comprised of traumatized or non-traumatized individuals. In addition, given the potential impact of stress on hippocampal functioning, greater deficits in memory and spatial reasoning, and relatively weaker findings for verbal intelligence, may support the plasticity theory.

Finally, support for the heredity theory will not be found in the meta-analysis, itself, but rather in the qualitative review of prospective studies. Regardless of the pattern of findings, group differences prior to traumatic exposure will offer strong support for this theory.

Other factors (e.g., sex, veteran status, trauma type, use of psychiatric comparisons, and number of subtests used) may not offer as direct support for any single theory but may offer further insight or even new theoretical development.

Results

Quantitative and Qualitative Review of Retrospective Studies

The primary meta-analysis of the difference in post-exposure intelligence between individuals with and without PTSD revealed a significant effect (d = .48; 95% Confidence Interval = .38 - .58; z = 9.38), supporting the hypothesis that participants with PTSD demonstrate a relative deficit (see Figure 1 for a display of individual study effect sizes). This effect is considered small to moderate based on Cohen's (1988) guidelines, in which a d of .50 represents a moderate effect. Orwin's (1983) 'Fail Safe N' was calculated with a criterion value of .20, meaning that the resulting N will reflect the number of studies with an effect size of zero needed to reduce the observed mean effect

size to .20—a small effect. Given the effect size and number of studies included in the review, Orwin's 'Fail Safe *N*' was 40.47, suggesting that an additional 40 studies are necessary to so reduce the mean effect size.

Other studies not included in the meta-analysis offer further support for the overall finding that post-trauma intelligence is relatively impaired for individuals with PTSD. Parslow and Jorm (2007) found in an adult cohort exposed to an Australian bushfire that post-exposure verbal intelligence was significantly lower in those who developed PTSD. In further analyses, they found that verbal intelligence was significantly lower in participants with any re-experiencing symptoms, and they found no such effect for hyperarousal symptoms. In addition, Diamond, Muller, Rondeau, and Rich (2001) found that PTSD re-experiencing symptoms were negatively correlated with verbal intelligence in adult survivors of child maltreatment.

One study not included in the meta-analysis reported non-significant results. Delaney-Black et al. (2002) found that intelligence was not different for children with and without trauma-related distress; however, higher exposure to violence predicted lower intelligence, even after controlling for gender, SES, caretaker verbal intelligence, home environment, and prenatal exposure to alcohol. Saltzman, Weems, and Carrion (2006), one of seven studies included in the meta-analysis to yield an effect size less than .20, found that maltreated children's intelligence was related to PTSD-related impairment, but not symptom level itself. In addition, Twamley, Hami, and Stein (2004) reported a non-significant but small to moderate negative effect for traumatized undergraduate college students with and without PTSD, though sampling college students may have yielded a group with lower levels of functional impairment. Finally, Nixon,

Nishith, and Resick (2004) found that, although verbal intelligence was not related to PTSD symptoms, lower verbal intelligence was related to higher occurrence of childhood, but not adult, trauma.

Overall, based on the results of the primary meta-analysis and the findings of the additional studies, the hypothesis that intelligence is lower in individuals with PTSD relative to those without PTSD is supported by some studies of children. The meta-analysis revealed significant variability between studies ($Q = 55.20$, $p < .001$), suggesting that—still assuming a fixed-effect model—additional factors, such as sample characteristics and methodology, may impact the magnitude of the effect. Conceptual and methodological moderator analyses were conducted to address the variability. See Tables 3 and 4 for the results of the categorical and continuous moderator analyses, respectively.

Conceptual Moderators

Sample characteristics. Sample characteristics hypothesized to modify the magnitude of the mean effect size are age at time of study, sex, race/ ethnicity, education, SES, and veteran status. Sex (percent of sample that was male) and veteran status (veteran sample vs. civilian sample) significantly moderated the magnitude of the mean effect size. No other sample characteristic was found to be a significant moderator.

The moderator analysis of sex revealed a significant impact of the percent of male participants in a sample on the mean effect size for intelligence, such that more men in a sample was related to a greater difference in intelligence between groups; however, a glance at the data revealed a substantial number of studies of all-female or all-male participants. Single-sex samples in this review are likely confounded by type of trauma because all-female samples tend to be based on women with a history of sexual or

intimate partner violence, whereas all-male samples were largely based on veteran samples investigating combat-related PTSD. An additional analysis was performed with these studies removed, and the effect of sex not only remained significant, but also increased.

The analysis of the potential impact of veteran status on the relationship between PTSD and intelligence was significant in that military veteran samples yielded higher effect sizes. Because veteran samples are homogenous for sex and typically age and type of index trauma, it is possible that these variables, and not veteran status itself, influenced these results; however, age did not significantly moderate the magnitude of the mean effect size, but the significant finding for sex may impact the interpretation of these findings. In addition, the effect of combat is difficult to disentangle from being a veteran, but the analysis of trauma type (including civilian exposure to war) may aid such disentanglement.

The analyses for race/ ethnicity (defined as the percentage of participants identifying as Caucasian), education, and SES (defined as the mean Hollingshead, 1975, score) resulted in non-significant findings, though there may still be some impact of SES on group differences in intelligence. The regression model for the analyses of SES yielded an R^2 of .18, indicating that the non-significant moderator still accounted for 18% of the variance in the overall mean effect size for the difference in intelligence between groups. In a sample of maltreated children, De Bellis, Keshavan, Frustaci, et al. (2002) found that both verbal and nonverbal intelligence were significantly lower for children with a PTSD diagnosis relative to a healthy, non-maltreated comparison group, and the difference resulted in an impressive *d* of 1.04; however, upon replication, the overall

difference between groups, though still significant, dropped to a *d* of .30 when groups were matched for SES (De Bellis, Keshavan, Shifflett, et al. (2002). Additional studies attempted to match samples based on SES and still found group differences, suggesting that SES may not play as important a role in this debate. This moderator analysis may reflect that SES does not have an impact on the overall mean effect size, that the analysis itself may be limited due to the limited number of studies included ($k = 7$), or that SES itself is not optimally measured.

Trauma-related characteristics. Trauma-related characteristics hypothesized to modify the magnitude of the mean effect size are PTSD symptom severity, trauma severity, type of trauma, and whether the index trauma occurred in childhood/ adolescence or in adulthood. The analysis of PTSD symptom severity is based studies using the *Clinician-Administered PTSD Scale* (CAPS; Blake et al., 1995), and the analysis of trauma severity is based on studies using the *Combat Experiences Scale* (CES; Keane, Caddell, & Taylor, 1988). Neither PTSD symptom severity, nor trauma severity was a significant moderator. As described earlier, Saltzman et al. (2006) found that intelligence was not related to PTSD symptom level itself, but rather PTSD-related functional impairment. In most studies comparing individuals with and without PTSD, groups are distinguished using DSM-IV diagnostic criteria, which require that symptoms cause significant impairment in social or occupational functioning. Symptom level itself is not sufficient to warrant a diagnosis, and CAPS total scores reflect both frequency and intensity of symptoms, making it a better indicator of PTSD severity than other measures. Nevertheless, no effect was found.

The impact of trauma severity, likewise, was non-significant. Studies not included in the meta-analysis provide mixed support for an impact of trauma severity. Delaney-Black et al. (2002) found that, although there was no relative difference in intelligence between children with and without posttraumatic distress, intelligence was negatively correlated with exposure to more violence. Two prospective studies offer diverging evidence. Kremen et al. (2007) found that high pre-exposure intelligence was associated with a lower risk of developing PTSD even when analyses controlled for combat severity. Thompson and Gottesman (2008), likewise, found that pre-combat intelligence protected against developing lifetime PTSD—but only in response to less severe combat exposure. At more extreme levels of exposure, intelligence was no longer predictive, and the risk of developing PTSD increased regardless of pre-combat intelligence. It is possible that the current analysis, comprised of a limited number of studies ($k = 5$) and assuming a linear relationship, did not adequately characterize the possible impact of trauma severity on the relationship between PTSD and intelligence (an unweighted quadratic estimation did not result in a significant finding either, using these limited data, $R^2 = .33$, $F = .45$, $p = .67$). Unfortunately, although some studies report a linear relationship between trauma severity and intelligence (Delaney-Black et al., 2002; Gurvits et al., 2000; Macklin et al., 1998), they do not explicate the impact of this relationship on the intelligence differences observed in individuals with and without PTSD. Overall, the evidence that trauma severity acts as a moderator is weak or mixed.

Analyses for both the age at the time of the trauma and the type of trauma initially yielded significant results; however, further analyses resulted in no effect of these hypothesized moderators. The analysis for the age at the time that the index trauma was

categorized as childhood/ adolescence, adulthood, and mixed. The 'mixed' category included only two studies with very low effect sizes. Once these studies were removed from the analysis, whether an individual's index trauma occurred in childhood/ adolescence or in adulthood had no effect on the magnitude of group differences for intelligence. Notably, one study found relative deficits in intelligence for women with PTSD as a result of adult rape, but only if the women had also experienced childhood trauma (Nixon et al., 2004). PTSD alone was not enough to explain the difference. Unfortunately, few other studies account for traumatic experiences across the lifespan, so generalizations from this single study are limited.

Similarly, the type of the trauma (i.e., combat, civilian exposure to war, child maltreatment, and mixed/ other) was shown to significantly moderate the magnitude of the mean effect size. Combat and civilian exposure to war, which includes victims of systematic persecution (e.g., the Holocaust) and refugees, resulted in moderate to high effect sizes, followed by a moderate effect for childhood maltreatment. A very small effect was found for the mixed/ other group. This last group was quite heterogeneous, including victims of natural disasters, motor vehicle accidents, stranger assault, adult rape, and partner violence. Once removed, there was no significant group difference, though the direction of the findings may warrant further exploration as group differences in intelligence for veterans and civilians with and without PTSD exposed to war appear comparable and notably—though not significantly—larger compared to child maltreatment. This pattern of findings may impact the interpretation of the finding that veteran samples demonstrate greater group differences in intelligence in that exposure to war, and not simply being a veteran, may produce dramatic and stable cognitive changes

(van Wingen et al., 2012) that may increase risk for developing PTSD and impact performance on tests of intelligence.

Overall, the analyses of conceptual moderators revealed that sex and veteran status significantly moderate the magnitude of the mean effect size of the difference in intelligence between individuals with and without PTSD. Analyses of age, race/ ethnicity, SES, education, PTSD severity, trauma severity, age at time of index trauma, and trauma type yielded non-significant findings. Some non-significant variables, such as type of trauma, may yet be important but not adequately examined by the current analyses, and others, such as SES, may have been based on analyses with too few cases.

Methodological Moderators

Trauma Exposure in Comparison Groups. Whether studies compared the individuals with PTSD to traumatized vs. non-traumatized controls was hypothesized to moderate the magnitude of the effect. The use of non-traumatized controls yielded a moderate to large effect (d = .63), but the effect was substantially reduced for traumatized controls (d = .39), suggesting an effect of trauma itself, separate from PTSD (see Figure 2). This reduction in effect size was particularly notable in samples of children. The effect size differences in intelligence between children with PTSD and non-traumatized controls ranged from .30 to 1.04 (N = 5 studies), but studies of traumatized children with and without PTSD yielded effect size differences between .13 and .27 (N = 4 studies). Trauma exposure in comparison groups appears to reduce the observed group difference in intelligence, suggesting that trauma itself is an important factor in the relationship between intelligence and PTSD, particularly for children.

Psychopathology in Comparison Groups. The exclusion or non-exclusion of psychopathology in comparison groups was hypothesized to be a significant moderator. Studies comparing individuals with PTSD to groups of individuals for whom psychopathology was not explicitly excluded—typically psychiatric controls—yielded significantly lower effect sizes (d = .32) than those comparing individuals with PTSD to individuals with no mental health diagnosis (d = .69). Such a finding supports the idea that intelligence is related to psychopathology more broadly, and not PTSD uniquely.

Operationalization of Intelligence. The way in which intelligence was operationalized may impact observed group differences. The operationalization of intelligence, examined in terms of the number of separate tasks used to measure intelligence, type of intelligence measured, and inclusion of working memory tasks, appeared to have some impact on study results.

The analysis of the number of separate tasks or subtests administered to measure intelligence was significant and suggested that including more subtests results in a larger effect and less variability among results. In the overall review, nine studies were based on single subtests (most often Vocabulary) and resulted in effect sizes ranging from -.32 to 1.38 in magnitude. Operationalizing intelligence as performance on a single task may vastly underestimate or vastly overestimate the actual group differences, though in general the pattern of data suggests that the inclusion of more subtests results in larger group differences.

More pertinent to the relationship between PTSD and IQ than the number of subtests administered is the type of task used to measure intelligence (i.e., a measure of verbal intelligence vs. a measure of nonverbal intelligence). Whether verbal or nonverbal

measures result in greater or lesser group differences may shed light on potential mechanisms. Thus, separate meta-analyses were conducted to estimate the mean effect size for verbal and nonverbal intelligence as a means of addressing the hypothesis that PTSD may be marked by differential disadvantage between these two broad domains of intelligence. See Figure 3 for a comparison of the results of the primary meta-analysis (comprising both verbal and nonverbal intelligence), the verbal meta-analysis, and the nonverbal meta-analysis. Verbal and nonverbal measures produced comparable mean effect sizes, though nonverbal measures resulted in a slightly higher overall effect (d = .58; 95% Confidence Interval = .41 - .75) compared to verbal measures (d = .51; 95% Confidence Interval = .38 - .64). These results suggest that relative deficits in intelligence observed in individuals with PTSD are related to both verbal and nonverbal aspects of intelligence.

Though the previous analyses suggest comparable results for verbal and nonverbal measures, an analysis of more basic cognitive processes could provide for a richer examination of intelligence and PTSD. Unfortunately, analysis at the subtest level is substantially hindered by studies' reports of overall scores only. Only 15 of the 39 studies included in the current review report results in such a way that interpretations can be made at the level of the subtest, and 6 of those studies base their findings on a single subtest—allowing subtest-level interpretation by default. Nevertheless, a qualitative examination of studies whose subtest-level data are presented individually revealed that, although Vocabulary and Block Design subtests are the most commonly-administered, they produce variable results. An examination of studies reporting individual scores for Similarities (Gil, Calev, Greenberg, Kugelmass, & Lerer, 1990; Kaplan et al., 2002;

Saigh, Yasik, Oberfield, Halamandaris, & Bremner, 2006) and Picture Completion (Gil et al., 1990; Kivling-Bodén & Sundbom, 2003; Saigh et al., 2006) revealed that, though administered less often, these subtests appear to produce consistent significant (Similarities) or consistent null (Picture Completion) findings.

Finally, a moderator analysis was conducted to assess whether the inclusion of tasks directly tapping working memory in the operationalization of intelligence would impact the magnitude of group differences for intelligence. As displayed in Figure 4, inclusion of working memory subtests (e.g., Digit Span or Arithmetic on Wechsler tests) significantly moderated the magnitude of the mean effect, resulting in markedly higher effect size differences for studies using such measures (d = .70) compared to those not using such measures (d = .28).

Qualitative Review of Prospective Studies

The results of the quantitative and qualitative review of retrospective studies examining the difference in intelligence for individuals with and without PTSD revealed a significant small to moderate effect, supporting the hypothesis that there are, indeed, group differences; however, these studies do not contribute to the debate of causality. Prospective studies (i.e., studies measuring intelligence prior to trauma exposure) may begin to address this debate. Nine studies measuring pre-exposure intelligence (including three that include post-exposure measurements) were reviewed qualitatively.

Five prospective studies were based on veteran samples, and they provide mixed support for the theory of intelligence as a pre-exposure risk/ protective factor. In a sample of Vietnam veterans, Macklin et al. (1998) found that lower pre-exposure intelligence not only predicted the development of PTSD, but also predicted more exposure to severe

combat and more severe PTSD. When the veterans were assessed again, the difference in intelligence remained, and both groups experienced comparable declines in intelligence over time.

Even stronger support is reported by Kremen et al. (2007) who found in sample of monozygotic (MZ) and dizygotic (DZ) twins recruited from the Vietnam Era Twin Registry (Eisen, Neuman, Goldberg, Rice, & True, 1989) that higher pre-exposure intelligence was associated with a lower risk of developing PTSD, even after controlling for combat exposure, age at military entry, and education. Furthermore, intelligence was similar for combat-exposed and non-exposed MZ twin pairs, regardless of PTSD diagnosis. The same was not true for DZ twin pairs. One of the strongest points of this study is its exclusion of veterans with traumatic experiences prior to being assessed upon entry into the military. Other prospective studies based on veteran samples in the current review do not report excluding (or measuring) prior traumatic experiences. The findings from this study support not only the pre-exposure risk theory, but also a genetic explanation for the relationship between PTSD and intelligence.

Similar to the previous two studies, Thompson and Gottesman (2008) found in a sample of Vietnam veterans that individuals with pre-exposure intelligence in the top 25th percentile were less likely to develop PTSD over the lifespan; however, unlike for Kremen et al. (2007), this finding was no longer significant when analyses controlled for severity of combat exposure. For veterans exposed to less severe combat, higher intelligence protected against developing PTSD, but at more severe levels of combat, intelligence provided no such protection. As the authors suggest, veterans exposed to extreme combat appear to “exhaust both their biological and environmental resources” (p.

581). In addition, combat severity—and not pre-exposure intelligence—predicted current PTSD. This study lends partial support to the theory that intelligence is a pre-exposure protective factor against PTSD, but it asserts that extreme combat may essentially trump such protection.

Not all prospective studies using military samples lend clear support for intelligence as a pre-existing risk or protective factor in the development of PTSD. Gil et al. (1990) found in a sample of outpatient Israeli military veterans that pre-exposure intelligence was not significantly different for veterans with PTSD and veterans with other psychiatric disorders, though the results yielded a small to moderate effect. Like in Macklin et al. (1998), a decline in intelligence was observed between pre- and post-exposure measurements, and the declines were comparable between groups. Group differences in post-exposure intelligence were non-significant but nevertheless noteworthy. Given the effect sizes (d_{pre} = .32, d_{post} = .45), it is possible that lack of significant findings between these groups may be the result of relatively small sample size (12 per group), but an additional psychologically healthy comparison group included for post-exposure analyses performed significantly better on all measures of both verbal and nonverbal intelligence (ass assessed by the *Wechsler Adult Intelligence Scales*, WAIS; Wechsler, 1955), except for the Picture Completion subtest. Although the overall results of the study were not significant, the effect sizes pre- and post-exposure are small to moderate. Furthermore, both veterans with and without PTSD demonstrated lower post-exposure IQ relative to a non-traumatized healthy comparison group, suggesting that trauma and general psychopathology—not PTSD alone—are important factors to consider when discussing group differences in intelligence.

More equivocal findings were reported in another cohort of Israeli veterans. Kaplan et al. (2002) found that participants who later developed PTSD (from both combat- and non-combat-related trauma) performed significantly worse on pre-exposure measures of both verbal and nonverbal intelligence compared to participants without PTSD. Despite the seeming support for pre-exposure intelligence as a risk or protective factor, the authors report that these results were made non-significant when analyses controlled for a measure of motivation to serve in the military. It is possible that this motivation measure may be tapping the participants' effort on tests of intelligence. If one does not want to serve in a compulsory military, one would likely not excel in performance on a test for placement in that compulsory military; however, it is possible (though only speculative) that motivation to serve could be also be framed as more anxiety about serving, which would suggests that analyses controlling for this measure may underestimate the actual difference in intelligence between groups. Though it highlights the importance of measuring additional variables, this study provides unclear support for the current debate.

Prospective studies based on veteran samples offer mixed support for the pre-exposure risk hypothesis. Civilian samples offer more consistent support with one exception. In a Palestinian cohort of children exposed to war-related trauma who were first assessed in 1993 (Time 1), again in 1997 (Time 2), and again in 2000 (Time 3), Qouta, Punamäki, Montgomery, and Sarraj (2007) found that nonverbal intelligence at age 10 to 11(Time 1) was not significantly correlated with symptoms of PTSD seven years later (Time 3). A separate measure of digit span, which was positively correlated with the measure of nonverbal intelligence, at Time 1 did negatively predict depressive

symptoms and positively predict resilient attitudes at Time 3. The analysis for PTSD symptoms relied on number of symptoms only and did not account for PTSD-related impairment, which could have reduced its true relationship to intelligence. Nevertheless, this study offers no support for a pre-exposure risk explanation for the relationship between PTSD and intelligence, particularly for children.

The remaining three prospective studies of civilian samples, on the other hand, offer strong support. Parslow and Jorm (2007) measured pre- and post-exposure verbal intelligence in an Australian cohort who reported exposure to a bushfire that occurred in January 2003. The authors found that lower pre-exposure verbal intelligence was predictive of developing PTSD and was specifically related to having any re-experiencing symptoms—but not hyperarousal symptoms. For both groups, verbal intelligence increased over time due to practice effects, but the increase for participants with PTSD was significantly less than for participants without PTSD. In addition, verbal intelligence measured after exposure to the bush fire was still significantly higher in participants who did not develop PTSD. It is unclear, however, whether participants were exposed to a trauma prior to their initial assessment. The authors report that over 4,105 individuals were randomly selected from compulsory electoral rolls, and 2,404 agreed to be in the study. No exclusions were discussed, suggesting that perhaps some individuals in the study had experienced a previous trauma. Nevertheless, the results of this study would not likely be completely overturned as a result of the misclassification in some cases of post-exposure as pre-exposure intelligence. This study appears to lend support to both sides of the debate in that group differences in verbal intelligence predate trauma exposure and PTSD, and increases over time in verbal intelligence were reduced for

individuals with PTSD, possibly suggesting continued cognitive effects deficits caused by the later development of PTSD.

Offering further support for the pre-exposure risk factor theory, in a Michigan cohort assessed at 6 years of age and again at age 17, Breslau, Lucia, and Alvarado (2006) found that participants with a WISC-R Full Scale IQ greater than 115 (i.e., at least one standard deviation above average) at age 6 were significantly less likely to be exposed to trauma by the age of 17, and they were significantly less likely to develop PTSD in response to trauma. Other risk factors for exposure to trauma included low birth weight, externalizing problems, urban environments, and being male. Being female, as well as having any anxiety disorder, was more predictive of developing PTSD.

Perhaps among the best-designed prospective studies to shed light on the relationship between PTSD and intelligence is Koenen et al.'s (2007) study of a New Zealand cohort assessed at ages 5, 7, 9, 11, 26 and 32. *Stanford-Binet* (Thorndike, Hagen, & Sattler, 1986) and *Wechsler Intelligence Scales for Children—Revised* (WISC-R; Wechsler, 1974) Full Scale scores at age 5 did not predict exposure to trauma by age 26, but lower scores on either measure predicted developing PTSD. By age 32, only the Stanford-Binet remained predictive of PTSD. Several additional variables were measured at each wave of the study. Having a difficult temperament predicted both trauma exposure and PTSD by age 26. In addition, antisocial behavior (based on teacher ratings), hyperactivity, and mothers' internalizing styles all predicted trauma exposure and PTSD by age 26. Several factors predicted PTSD only, including being unpopular (based on teacher ratings), low SES, number of residence changes, and change or loss of a parent. By age 32, in addition to the Stanford Binet, only antisocial behavior and SES remained

predictive of developing PTSD. Unfortunately, the authors do not present subtest-level information for the WISC-R or the Stanford Binet. Regardless, this study is noteworthy for several reasons. First, the authors began assessing children at a very early age (5 years), reducing the likelihood that children included in the study had experienced a trauma prior to cognitive testing. It remains possible but less likely. Second, the authors used two complete batteries to measure intelligence. Third, participants were assessed at 6 different time-points, which may have reduced the chance that relevant information from participants' reports is not forgotten or significantly misremembered between waves. Fourth, and finally, the authors included a number of additional predictors that enrich the current debate. This study lends very strong support to the theory that intelligence is a pre-existing vulnerability to the development of PTSD while at the same time showing that other factors exert influence.

Though the qualitative review of prospective studies yielded mixed results, the evidence strongly favors the theory that relatively lower intelligence is a pre-existing vulnerability to the development of PTSD (and to the exposure to trauma) rather than a deficit observed only after trauma exposure and subsequent symptom development.

Discussion

Summary of Findings from the Current Review

A review of 29 retrospective and 9 prospective studies of group differences in intelligence between individuals with and without PTSD strongly supports both the idea that individuals with PTSD perform more poorly on measures of intelligence relative to healthy comparisons and the idea that these group differences are observed prior to traumatic exposure and symptom onset. A minority of studies reported non-significant

results, and even some of these studies provided evidence that, although intelligence was not found to differ for individuals with and without PTSD, intelligence was related to other relevant variables, such as higher exposure to violence, the occurrence of child trauma prior to an adult trauma, and PTSD-related functional impairment (Delaney-Black et al., 2002; Nixon et al., 2004; Saltzman et al., 2006). Moderator analyses for retrospective studies revealed that the magnitude of the mean effect size was significantly greater for samples of more men and of veterans. Analyses for age, race/ ethnicity, socioeconomic status, trauma severity, and PTSD severity were not significant. The use of non-traumatized comparison groups resulted in larger group differences between individuals with and without PTSD than did the use of traumatized comparison groups, particularly in studies of children, and the use of healthy comparison groups, relative to psychiatric comparison groups, also resulted in greater group differences. Finally, including more subtests and including a working memory task (i.e., Digit Span or Arithmetic) in the measurement of intelligence resulted in greater effect size differences in intelligence, and both verbal and nonverbal intelligence were comparably implicated, though specific subtests (i.e., Vocabulary vs. Similarities, Block Design vs. Picture Completion) appeared to operate differently.

Theoretical Implications

The current review provides support for the theory that group differences in intelligence pre-date trauma exposure and, thus, may be risk factors for the development of PTSD. This finding also supports the theory that intelligence and PTSD may be related as a result of hereditary or other pre-morbid factors and is not simply the result of trauma- or PTSD-induced impairment.

Evidence for the resource theory was limited in that none of the supporting moderators (SES, education, and race/ ethnicity) were found to be significant, and the magnitude of the effect was generally comparable for verbal intelligence and nonverbal intelligence; however, the effect of SES may have been underestimated as a result of a limited sample of studies reporting SES or inadequate measuring of the construct itself. Despite the good overall validity and reliability of the Hollingshead Four-Factor Index (Hollingshead, 1975), Krieger, Williams, and Moss (1997) argued that measures of SES that focus solely on the individual household—and not on the surrounding community or neighborhood—do not account for important social resources.

Based on the plasticity theory, one might expect that earlier exposure to extreme stress or trauma would result in greater negative impact on intellectual development than would later stress. The moderator analysis did not support this assertion, though variance within studies looking at childhood/ adolescent trauma is substantial and significant, suggesting that combining groups across such a large portion of early development may be too simplistic and may ignore the potential for specific time periods of greater sensitivity to stress and cognitive change. It is also possible, quite separately, that the effects of early trauma—if not followed by repeated exposure—may be buffered by cognitive reserve or intelligence, which may diminish with each subsequent exposure. Thus, it is possible that repeated exposure, not simply early exposure, contributes to cognitive changes still consistent with the plasticity theory.

This idea that repeated trauma may play an important role in the relationship between PTSD and intelligence is somewhat supported by the finding, albeit not significant, that wartime trauma for both veterans and civilians yielded notably higher

group differences in intelligence. Such exposure is ostensibly more likely characterized by repeated occurrence. These findings may suggest that central nervous system alterations, such as chronic exposure to high levels of stress hormones (Southwick, Bremner, Krystal, & Charney, 1994) following extended trauma, remain influential. Van Wingen et al. (2012) found that exposure to combat resulted in cognitive changes that persisted for at least 1.5 years, regardless of mental health problems, and in prospective studies that included both pre- and post-exposure measures of intelligence, individuals with and without PTSD showed comparable declines in intelligence over time (Gil et al., 1990; Macklin et al., 1998). Considering measures of intelligence are adjusted for age, declines should not reflect simple aging. Perhaps exposure to trauma has a long-term impact on the brain regardless of whether an individual develops PTSD. In line with this proposition is the finding that the use of traumatized comparison groups, rather than non-traumatized groups, resulted in much lower (but still significant) group differences in intelligence. This finding demonstrates a negative effect of trauma by itself on cognitive functioning. It is possible that relative lower intelligence increases the risk of both traumatic exposure and PTSD, which in turn, could result in further impairment of performance on measures of intelligence.

The finding that the difference in intelligence between groups was strongly impacted by the inclusion of a working memory task and that individuals with PTSD also tended to perform poorly on tasks of spatial and analogical reasoning (discussed in more detail below) may suggest that the functioning of the hippocampus could be an important factor, among many important factors, in cognitive deficits observed with PTSD;

however, whether possible dysfunction occurs as a result of heredity or plasticity—or both—is not clear.

It should be noted that all three of these theories—resources, plasticity, and heredity—may interact in a transactional system of influence (see Sameroff & Fiese, 2000). Nevertheless, it appears that hereditary and plasticity theories may best describe the relationship between PTSD and intelligence. Possible mechanisms of influence are shared genetic contributions to intelligence and risk for PTSD and central nervous system alterations due stress response resulting in lower intelligence and PTSD. A unique possible contribution of the plasticity theory to the theory of the relationship between PTSD and intelligence is the assertion that trauma exerts intergenerational influence (Yehuda, Halligan, & Grossman, 2001), which may call into question the operationalization of *pre-exposure* intellectual ability. For example, there is evidence that prenatal exposure to stress hormones is negatively correlated with child intelligence at age seven (LeWinn et al., 2009). It is possible that maternal trauma history may impact child cognitive functioning, which may, in turn, increase the likelihood that the child will experience trauma and also be less resilient. Much of the debate about the relationship between PTSD and intelligence does not take into account intergenerational effects.

Importantly, two of the strongest findings of this review relate to the operationalization of intelligence—specifically, whether the measurement of intelligence included a working memory task and the number of separate tasks used to measure intelligence. It is clear that the work of Spearman (1927) and Cattell (1963) remains influential in the operationalization of intelligence in research in that intelligence is often measured using one or two tasks—typically a test of vocabulary and spatial reasoning,

ostensibly accounting for both *gf* and *gc*—because, in addition to saving time, the assumption is that *g*, though comprised of these relatively separate factors, can still be approximated with a limited sampling of *s*. In this body of literature, however, use of fewer separate tasks or subtests appears to underestimate group differences in intelligence, perhaps because individuals with PTSD demonstrate not simply a general intellectual deficit, but a specific pattern of deficits.

Studies using healthy comparison groups—rather than psychiatric comparison groups—yielded greater group differences in intelligence for individuals with and without PTSD, suggesting that general psychopathology, not just PTSD, is associated with relative deficits in cognitive functioning; however, particular patterns of deficits may better discriminate between specific forms of psychopathology (e.g., Bloch et al., 2006; Boone, Ananth, Philpott, Kaur, & Djenderedjian, 1991; Koenen et al., 2009; Tiihonen et al., 2005). Individuals with PTSD performed consistently poorer on the Similarities subtests of Wechsler tests but performed equally well for the Picture Completion subtests. Performance on two other subtests, Vocabulary and Block Design, predicted PTSD but with more variability in reported significant findings. It is possible that specific abilities measured by these tests could enrich our theoretical development of causal mechanisms.

The Picture Completion subtest requires a participant to look at a series of pictures of real-life objects and identify the piece that is missing. The subtest is believed to tap cognitive style, particularly field independence, as well as alertness to the environment. Field dependence-independence is typically associated with the ability to disembed information from a complex visual field (e.g., performance on the Embedded

Figures Test; Witkin, 1971); however, Linn and Kyllonen (1981) found that performance on the Picture Completion subtest is part of a factor separate from this conceptualization of field dependence-independence. Picture Completion performance, according to their findings, is associated with the ability to choose effective problem-solving strategies for familiar visual material, when other salient but ineffective strategies are available. This subtest, therefore, does not tap the kind of field independence most commonly described.

In addition, the Picture Completion subtest may not be as demanding as other subtests (Reitan & Wolfson, 1992). One could argue that performances are equal between groups because the task is easy, but another study found that maltreated children (PTSD not assessed) performed significantly worse compared to population norms on four out of five performance subscales, and significantly better on Picture Completion (Frankel, Boetsch, & Harmon, 2000), suggesting perhaps a unique advantage. The authors argue that experiencing abuse may encourage hypervigilance to problems in the environment (e.g., things that are missing or broken).

Performance on another nonverbal subtest, the Block Design subtest, was more variable, but overall, individuals with PTSD performed relatively worse than individuals without PTSD. This subtest requires that participants arrange blocks to match pictures. Like for Picture Completion, this subtest is believed to tap cognitive style, in particular field independence, in addition to spatial reasoning. It also requires additional synthesis and is much more abstract than a Picture Completion task. Linn and Kyllonen (1981) found that performance on this Block Design task requires cognitive restructuring and the ability to create new representations of unfamiliar visual material (e.g., geometric shapes) and is consistent with common conceptualization of field dependence-independence.

Poorer performance the Block Design subtest is suggestive of more field dependence, or difficulty disembedding visual material. Individuals with PTSD appear to be more field dependent than individuals without PTSD and may have increased difficulty on tasks that are more abstract or novel.

The field dependence-independence literature is vast, and many interpretive avenues could be explored. A particularly promising avenue may be found in the results of early studies finding that field dependent individuals were more likely to recall incidental social information for events or tasks (Crutchfield, Woodworth, & Albrecht, 1958; Fitzgibbons & Goldberger, 1971). This finding harkens back to Foa and Kozak's (1993) description of fear structures and trauma memories, which are characterized by the inclusion of a greater number of stimuli, including incidental or even irrelevant stimuli.

In a somewhat similar pattern, individuals with PTSD generally demonstrated relative deficits on measures of vocabulary, but there was some variability, with some studies finding no difference between individuals with and without PTSD (Nixon et al., 2004; Werner et al., 2009); however, in every study that reported performance on the Similarities subtest of the Wechsler tests, individuals with PTSD demonstrated a relative deficit (Geuze et al., 2008; Gil et al., 1990; Saigh et al., 2006; Saltzman et al., 2006). Unlike on a measure of vocabulary where individuals are asked to define a specific word, the Similarities subtest requires that an individual describe how two words are alike. The test-taker must not only know the definition of each term, but also be able to employ analogical reasoning to find the relationship. Individuals with PTSD appear to generally

demonstrate poorer vocabulary and, when measured, consistently demonstrate greater difficulty with analogical reasoning.

Going even beyond the ability to generate relationships among words, Gilberston et al. (2007) found that chronic PTSD was associated with poor performance on allocentric, but not egocentric, spatial tasks. Allocentric spatial processing requires that an individual be able to orient objects based on their relative positions to each other—rather than orienting based on their relative position to the self, using egocentric spatial processing. Allocentric performance was correlated with hippocampal volume, and these findings were shared by non-combat exposed twins. Cattell (1963) considered both *gf* and *gc* to comprise the highest order of the structure of intelligence, relation education, determining relationships among stimuli—whether verbal or visual. It is possible that pre-existing difficulty in discerning relationships among pieces of information may both increase the risk of trauma, and, the authors argue, prevent effective fear extinction. Even a single cue in an otherwise safe context can trigger intense fear.

Again, trauma memories are characterized not only by the inclusion of a larger number of stimuli, but also erroneous or exaggerated associations between stimuli and danger. Factors like field dependence may afford such greater inclusion and thus, as Gilbertson et al. (2007) suggests, impair fear extinction, especially when accompanied by an over-reliance on concrete thinking that may limit one's ability to generate reasonable associations between stimuli, especially regarding beliefs about danger.

Limitations of Current Review

The findings and conclusions of the current review must be carefully considered with the following limitations in mind. First, the use of meta-analytic procedures to

aggregate study findings, though common, remains controversial because, as some argue, it combines apples and oranges. Studies with very different properties—sample characteristics, operationalization of variables, study design—are lumped together and may mischaracterize true effects. Certainly in the current review, apples (e.g., male combat veterans) were lumped with oranges (e.g., female survivors of domestic violence). In addition, meta-analysis is constrained by its specific statistical requirements, which lead to the exclusion of some studies from the quantitative review. Furthermore, while moderator analyses allowed for the separating out effects, they did not control for probable intercorrelation among variables, such as type of trauma and sex.

Another limitation is that the current review only included studies published in peer reviewed journals. Unpublished manuscripts, such as dissertations, were excluded, increasing the possibility that the findings in this review are elevated due to publication bias (i.e., the File Drawer Effect); however, Orwin's (1983) 'Fail Safe N' was calculated and indicated that an additional 40 studies are necessary to reduce the mean effect size to .20.

The lack of significant findings for race/ ethnicity and SES, as well as PTSD severity and trauma severity, may be due in part to the possibility that these variables do not bear on the relationship between intelligence and PTSD and in part to a limited analysis. The weighted least squares regressions included 5 to 8 studies for each analysis, and some researchers suggest that estimates of regression coefficients should be based on a minimum of 5 cases per predictor (Tabachnick & Fidell, 1989). Though the inclusion of at least 5 predictors for each analysis meets or exceeds this recommendation, still many other researchers recommend a minimum of 15 or even 40 (Cohen, 1983) cases per

predictor. The current analyses may have underestimated the effect of race/ ethnicity, PTSD severity, and trauma severity on group differences in intelligence; however, evoking post-hoc power analyses as a means of explaining non-significant findings, and even informal assertions that findings would be significant if analyzed with more cases may imply true statistical relationships in the face of contradictory data. A possible solution to this problem is an urge to researchers to include additional data, such as more detailed demographic characteristics of their samples, when presenting findings.

Limitations of the Broader Research

Current research on this topic is limited in a number of ways. Characterizations of trauma exposure (e.g., age at trauma, severity of trauma, amount of trauma exposure, etc.) and PTSD symptom expression (e.g., current versus lifetime PTSD, symptom severity, cluster-specific analyses, etc.) could enhance our understanding of the relationship between PTSD and intelligence, but many studies do not report such data. How an individual who develops PTSD and then recovers compares to an individual with chronic PTSD is largely unexplored but could help to clarify whether intelligence is a marker of PTSD in general or a measure of long-term failure to recover. In addition, the use of non-traumatized comparison groups may confound the effect of PTSD with the effect of trauma, and studies should be careful when drawing strong conclusions in such circumstances.

In addition, potentially important variables in the study of intelligence and PTSD are often confounded with one other. For example, results based on veteran samples are potentially confounded by sex and type of trauma, among other things. Eren-Koçak, Kiliç, Aydin, and Hizli (2009) suggest that PTSD research should focus more on natural

disasters, which are random, rather other types of trauma that are not random and are often confounded with variables like pre-exposure cognitive abilities, making interpretation of findings difficult. It is important to consider, however, that even random natural disasters have nonrandom negative consequences, often related to socioeconomic status.

Prospective studies allow for stronger interpretation than retrospective studies, but even these improved studies may underestimate the effect of trauma. Many prospective studies, such as those based on military samples, measure intelligence prior to the occurrence of an adult trauma. The assumption appears to be that the adult trauma measured is the first trauma experienced by an individual, though most studies do not report assessing for prior trauma, despite a number of studies reporting veterans with a history of child abuse are at an increased risk of developing combat-related PTSD (Bremner et al., 1993; Engel et al., 1993).

Rosen and Martin (1996) found that males in their military sample (N = 1,060) had a similar rate (15%) of child sexual abuse compared to the 16% reported in a national sample (Finkelhor, Hotaling, Lewis, & Smith, 1990). It is unlikely that large samples of veterans are completely free of men traumatized prior to combat. In fact, in the Vietnam Era Twin Registry almost 14% of veterans reported experiencing more than one type of trauma (i.e., at least one type of trauma, such as rape and personal assault, other than combat) and that experiencing multiple traumas and experiencing trauma at a younger (pre-service) age were each associated with greater risk for developing PTSD following service in the Vietnam War (Koenen et al., 2002). Though prospective studies offer the

strongest support for the theory that intelligence is a pre-exposure risk factor for developing PTSD, these studies are not without their problems.

Another, perhaps smaller area of concern for studies looking at intelligence and PTSD is that group averages for individuals with PTSD included in the current review are well within the average range of intelligence, and averages for individuals without PTSD, especially non-traumatized groups are nearly a full standard deviation above the norm (see Figure 5). It is possible that above average intelligence protects against the exposure to trauma; however, another explanation may arise out of the many different versions of tests administered. Some studies' findings are based on outdated measures, and it is not clear if updated norms are used. Nevertheless, norm changes would likely not account for all of the dramatically-increased scores for intelligence in non-traumatized groups. This body of literature tends to posit that relative deficits in intelligence are associated with increased risk of developing PTSD; however, it is possible that higher relative intelligence—perhaps greater cognitive reserve—is a protective factor both against and following trauma. Such a re-phrasing may appear arbitrary, but it has the potential to refocus the debate.

Finally, the body of literature on the relationship of intelligence to PTSD does not appear to incorporate findings regarding the intergenerational transmission of trauma and PTSD, findings that could enrich theoretical development. Trauma exposure and PTSD in parents have the potential to impact child cognitive development via a number of pathways. For example, Koenen et al. (2003) demonstrated that children's intelligence is negatively impacted by witnessing violence between parents and that this effect is not wholly attributable to heritability. A separate study of children's exposure to domestic

violence showed that, although such exposure is related to memory deficits in children, positive parenting from mothers moderates the negative effect (Jouriles, Brown, McDonald, Rosenfield, Leahy, & Silver, 2008). With the exception of one study that controlled for maternal discipline style when analyzing the relationship of intelligence and PTSD symptoms in children (Qouta et al., 2007), examination of the potential role of intergenerational factors is largely absent in the body of literature reviewed.

Conclusion

The quantitative and qualitative review of both retrospective and prospective studies yielded support for the relationship between PTSD and intelligence. Individuals with PTSD exhibit relatively lower intelligence when compared to individuals without PTSD. Furthermore, prospective studies strongly support the theory that low intelligence is a risk factor for exposure to trauma and to the development of PTSD, rather than simply a result of trauma- or PTSD-induced dysfunction alone. A tendency toward field dependence, relative difficulty with abstract or novel material, and relative difficulty with analogical reasoning may be important mechanisms in the overall relationship, and further studies are needed to elaborate these potential findings.

Lastly, findings from the reviewed studies have the potential to inform theoretical models of intergenerational transmission of trauma-related risk (e.g., negative impact of exposure to domestic violence on children's intelligence; Koenen et al., 2003). It is possible that parenting and other parental factors could impact the relationship between intelligence and PTSD in children, but the existing literature lacks more direct examination of this possible intergenerational effect. The next chapter presents data from a study of intergenerational risk and resilience in a low-income, urban, traumatized

sample. Data from this study allow for examination of trauma, PTSD, and intelligence in a sample of 48 African American mothers and their school-age children.

Chapter III: PTSD and Intelligence in Mothers and their Children

The purpose of this study is to assess whether trauma, PTSD, and intelligence are related in a low-income, urban, African American sample of 48 mothers and 55 children. The first goal of this study is to examine group differences in intelligence for mothers with and without PTSD. Because substance and alcohol use and depression relate to both to PTSD (McFarlane & Papay, 1992) and to cognitive test performance (Hindmarch, Kerr, & Sherwood, 1991; Johnsen, Kanagaratnam, & Asbørnsen, 2008), analyses of group differences in intelligence accounted for these factors. The second goal of this study is to examine the relationship of child PTSD symptoms and child intelligence. As with mothers, analyses accounting for child trauma exposure and child symptoms other than PTSD were conducted.

The third goal of this study is to examine the relationship of maternal factors including maternal trauma exposure, maternal psychopathology, including PTSD, and maternal intelligence to child trauma exposure, child psychopathology, including PTSD, and child intelligence. The fourth goal of this study is twofold—first, to examine the relationship of maternal trauma, psychopathology and intelligence variables with maternal parenting variables (laxness, overreactivity, child abuse potential, and prenatal health); and second, to examine the relationship of the maternal parenting variables on child trauma exposure, child psychopathology, and child intelligence.

Method

Participants

Eligible mothers included adult women who self-identified as African American and were the biological mother and primary caregiver of at least one child between ages 8

and 12 years. In addition, mothers were neither cognitively disabled (i.e., mentally retarded) nor actively psychotic. Eligible children were between the ages of 8 and 12 years and were not diagnosed with autism spectrum disorders, bipolar or psychotic disorder, or cognitive disabilities. If a mother had multiple children eligible for this study, she was allowed to choose which child would participate. Mothers were recruited from the waiting rooms of primary care clinics, obstetric-gynecological clinics, and an outpatient child psychiatry clinic at Grady Memorial Hospital. All mothers provided verbal and written informed consent for their participation in the study, and all children provided study assent with mothers' signed parental permission consent forms. All procedures in the study are currently approved by the institutional review boards of Emory University School of Medicine and Grady Memorial Hospital, Atlanta, GA.

Measures

Demographics. In order to best characterize the sample, the following data were collected: Maternal age, child age, child sex, maternal education (highest grade completed), and household monthly income.

Reynolds Intellectual Assessment Scales (RIAS; Reynolds & Kamphaus, 2003). For this study, intelligence and memory were defined as performance on the RIAS. The RIAS is composed of six subtests (Guess What?, Verbal Reasoning, What's Missing?, Odd-Item-Out, Verbal Memory, and Nonverbal Memory), resulting in an overall Composite Intelligence Index (CIX), as well as three separate indices—the Verbal Intelligence Index (VIX; VIX = Guess What? + Verbal Reasoning), Nonverbal Intelligence Index (NIX; NIX = What's Missing? + Odd-Item-Out), and Composite

Memory Index (CMX; CMX = Verbal Memory + Nonverbal Memory). Only the VIX and NIX contribute to the CIX.

The Guess What? subtest is designed to measure crystallized knowledge, including vocabulary (e.g., "What has many pieces, fits together, and must be solved to make a picture or design?") and general cultural knowledge (e.g., "What has a vaulted ceiling, was commissioned by Pope Sixtux IV, and is famous for its fresco masterpiece?"), similar to Wechsler Vocabulary and Information subtests (Wechsler, 1997, 2008). The Verbal Reasoning subtest is a measure of analytical reasoning abilities or analogical reasoning and requires also vocabulary knowledge (e.g., "Disappointment is to frown, as satisfaction is to _____?"). This subtest may assess abilities comparable to those assessed by a Wechsler (1997, 2008) Similarities subtest. The Odd-Item-Out subtest is a measure of general nonverbal reasoning skills and requires that examinees determine which stimulus does not fit with other stimuli on a page. These latter two subtests—VRZ and OIO—are considered the best measures of general intelligence relative to other subtests included (Reynolds & Kamphaus, 2003). The What's Missing? subtest is another measure of nonverbal reasoning abilities and requires that examinees distinguish between essential and non-essential aspects of a pictured object or scene. This subtest is very similar to a Wechsler (1997, 2008) Picture Completion subtest. The Verbal Memory subtest assesses ability to encode, store, and immediately recall orally-presented short passages of text. Similarly, the Nonverbal Memory subtest requires that an examinee encode, store, and immediately recognize visually-presented material, given the presence of distractors.

The RIAS was the choice measure for the current study for a number of reasons. The very wide normative age range (age 3 years to 94 years) allows administration of the same test to both mothers and children, and the inclusion of memory subtests allows for ease in examining both intelligence and memory. Furthermore, the RIAS is brief relative to Wechsler tests, taking only about 30 minutes to administer and demonstrates significant correlation in several studies with the *Wechsler Intelligence Scale for Children—Fourth Edition* (WISC-IV; Wechsler, 2003), the *Wechsler Adult Intelligence Scale—Fourth Edition* (Wechsler, 2008), and the *Woodcock-Johnson Tests of Cognitive Ability—Third Edition* (Woodcock, McGrew, & Mather, 2001; Edwards & Paulin, 2007; Krach, Loe, Jones, & Farrally, 2009; Smith, McChristian, Smith, & Meaux, 2009). Actual scores for the RIAS, however, are significantly higher—but still within the average range—relative to the other tests (Edwards & Paulin, 2007; Smith et al., 2009).

The RIAS is a reliable measure of intelligence with internal reliability alphas of .84 or higher for all age groups across all subtests (Reynolds & Kamphaus, 2003). In addition, the authors report reliability estimates for indices as being at or above .91, and test-retest reliability of .70. RIAS indices are also reported to be internally consistent across age, sex, and ethnicity. Convergent validity has been established through additional studies, such as Smith et al. (2009) presented above.

For this study, mothers and children whose composite intelligence index or composite memory index fell below 70 were not included in the study. If a mother was excluded on this basis, her child remained in the child-only analyses, but neither mother nor child were included in the mother-child combined analyses. If a child was excluded, the mother was included in mother-only analyses. This exclusion may appear at first to

have the potential to reduce the opportunity for important findings; however, the larger review indicated that individuals with PTSD demonstrated average intelligence. A score below 70 is well below that finding and is likely indicative of larger cognitive deficits unrelated to PTSD. Thus, these mothers and children were removed.

Traumatic Events Inventory (TEI; Gillespie et al., 2009). The TEI was the primary measure of maternal trauma exposure. The TEI is a 13-item self-report history of lifetime exposure to trauma, including combat, serious accident or injury, assaultive violence, child maltreatment, and sexual assault. The TEI also assesses age at first traumatic exposure.

Clinician-Administered PTSD Scale (CAPS; Blake et al., 1995). The CAPS is structured according to the DSM-IV criteria for PTSD. The CAPS measures both frequency and intensity of symptoms, which are both measured on a 0 to 4 scale. Symptoms are coded as present with a frequency greater than or equal to 1 and an intensity greater than or equal to two. A diagnosis of PTSD requires the experience of a criterion A trauma, one re-experiencing symptom, three avoidance symptoms, and two symptoms of hyperarousal lasting longer than one month and causing significant functional impairment. The CAPS assesses current (based on the last month) and lifetime PTSD. For this study, the CAPS was administered to mothers based on two traumatic experiences chosen by the interviewer. If both child and adult traumas were present, the interviewer assessed current and lifetime PTSD on one child trauma and one adult trauma.

The CAPS is considered the gold standard for PTSD assessment (Weather, Keane, & Davidson, 2001). Inter-rater reliability is reported to be .92 to .99 for frequency

and intensity of symptoms. Test-retest reliability ranges from .77 to .96 for the three primary symptom clusters (re-experiencing, avoidance, and hyperarousal), and internal consistency for these clusters ranges from .85 to .87 with a total score internal consistency of .94 (Blake et al., 1995). In addition, convergent validity with the *Structured Clinical Interview for DSM-IV* PTSD module is reported to be .83 (Foa & Tolin, 2000), and sensitivity and specificity are reported to be .74 and .84, respectively (Hovens et al., 1994).

Modified PTSD Symptom Scale (MPSS; Foa & Tolin, 2000; Schwartz, Bradley, Sexton, Sherry, & Ressler, 2005). The PSS is a 17-item self-report scale of current (past two weeks) symptoms of PTSD and includes a measure of both frequency and intensity of symptoms. Like the CAPS, this measure is based on DSM-IV criteria for PTSD. Items are rated on a 0 to 3, with 0 indicating that a symptom is not present at all over the last two weeks and 3 indicating that the symptom has been experienced more than five times in a week. For the purposes of this study, both dichotomous and continuous scores were used. Primary mother-only analyses used a dichotomous PTSD diagnosis based on PSS, indicating presence of at least one intrusive symptom, at least three avoidance/ numbing symptoms, at least two hyperarousal symptoms, and a duration of symptoms lasting at least one month. This method of diagnosis using the PSS has been used in other studies (Jovanovic et al., 2010). Supplemental analyses will use continuous PTSD symptom scores which are based on the sum of frequency scores for the PSS, and this method, too, has been used in other studies (Heim et al., 2009; Ressler et al., 2011).

Beck Depression Inventory-II (BDI; Beck, Steer, & Brown, 1996). The BDI is a 21-item self-report questionnaire measuring current (past two weeks) symptoms of

depression. Items are rated on a 0 to 3 scale, with 0 indicting absence of a symptom and 3 indicating severe presentation of a symptom. The BDI provides a cutoff score for categorical analysis, but for this study only the continuous scores were used.

Drug Abuse Screening Test-10 (DAST-10; Skinner, 1982). The DAST-10 is a 10-item self-report screening measure for substance-abuse related problems occurring in the last year. Items address both consumption and interpersonal and medical consequences and are answered as *yes/ no*. The DAST-10 has performed well across several populations, including the general public and psychiatric outpatients and inpatients (Carey, Carey, & Chandra, 2003; Cocco & Carey, 1998; el-Bassel et al., 1997) demonstrating good validity and reliability (α =.86) (Carey et al., 2003), as well as sensitivity (.84) and specificity (.76) when using a cutoff score of 3 out of the possible 10 (Cocco & Carey, 1998). For the current study, only continuous scores were used.

Alcohol Use Disorders Identification Test (AUDIT; Saunders, Aasland, Babor, & Grant, 1993). The AUDIT is a 10-item self-report screening instrument for problematic alcohol use occurring in the last year. Like the DAST-10, items on the AUDIT assess both consumption and consequences, but responses are coded on a 0 (*never*) to 4 (*daily or almost daily*) scale. The AUDIT is well-validated across multiple samples, ranging from the general public to psychiatric inpatients (Bradley et al., 2003; Maisto, Carey, Carey, Gordon, & Gleason, 2000; Rubin et al., 2006), demonstrating good validity and reliability (α =.83) (Reinert & Allen, 2007), as well as good sensitivity (.90) and specificity (.70) when using a cutoff score of 8 out of the possible 40 (Maisto et al., 2000). For the current study, only continuous scores were used.

Child Abuse Potential Inventory—Second Edition (CAPI; Milner, 1986, 1994). The CAPI is a 160-item self-report screening measure used to assess risk for perpetration of physical child abuse. Seventy-seven items contribute to an overall physical abuse potential score with six subscales—Distress, Rigidity, Unhappiness, Problems with Child and Self, Problems with Family, and Problems from Others—and remaining items contribute to three validity scales (Faking Good, Faking Bad, and Inconsistency). Items are based on attitudes and parenting behavior that have been observed in parents who have been identified as physically abusive (e.g., "A child should never talk back") and are rated as *agree* or *disagree* (Milner, 1994; Milner, Gold, & Wimberley, 1986; Walker & Davies, 2010). Milner et al. (1986) recommends a cutoff score of 215 to indicate high-risk for abuse. The current study used only the primary Abuse Potential score, as well as the Faking Good validity score. Scores were analyzed continuously.

Parenting Scale (PS; Arnold, O'Leary, Wolff, & Acker, 1993). The PS is a 30-item self-report measure that assesses three parenting styles (i.e., laxness, overreactivity, and verbosity) that have been shown to be associated with adverse child outcomes. The PS has demonstrated good internal consistency, test-retest reliability, and convergent validity; however, the verbosity factor has not replicated well in other studies (Collett, Gimpel, Greenson, & Gunderson, 2001), particularly those based on low-income African American samples (Steele, Nesbitt-Daly, Daniel, & Forehand, 2005). Therefore, only the Laxness and Overreactivity subscales were considered. No cutoff score is recommended for these scales, and scores were analyzed continuously.

Violence Exposure Scale for Children—Revised (VEX-R; Fox & Leavitt, 1995). The VEX-R was the primary measure of trauma exposure in children in this study. The

VEX-R is a 22-item self-report interview of exposure to violence. Unlike other measures of child trauma exposure, the items are represented by cartoon depictions of violence (e.g., a picture of an angry man chasing a scared man), and items are rated on a 0 (*never*) to 3 (*lots of times*) scale indicating how often the child reports experiencing or witnessing a trauma. The scale is depicted as four labeled thermometers of increasing "temperature" (frequency of exposure). Media depictions of violence are explicitly excluded. In addition, the VEX-R does not assess sexual trauma. An alpha of .72 to .86 is reported for internal consistency of the VEX-R (Shahinfar, Fox, & Leavitt, 2000), and child-report of violence exposure on the VEX-R is significantly correlated with mother-reported child trauma exposure (Raviv et al., 2001).

UCLA PTSD index for DSM-IV, Child Version (UCLA; Rodriguez, Steinberg, & Pynoos, 1999). The UCLA was the primary measure of PTSD in children for this study. The UCLA is a 20-item self-report interview of current (last month) PTSD symptoms in children. Items are rated on a 0 *(none of the time*) to 4 (*most of the time*) Likert-type scale of how often a symptom is experienced in the last month. For this study an additional set of thermometers corresponding to scale responses was created and added to the UCLA to increase ease of responding for children. According to Decker and Pynoos (2004), the UCLA is internally-consistent with an alpha of .90 and a test-retest reliability.84. Steinberg et al. demonstrated convergent validity of .70 between the UCLA and the PTSD module of the *Schedule for Affective Disorders & Schizophrenia for School-Aged Children, Epidemiologic Version* (Orvaschel & Puig-Antich, 1986) and .82 with the *Clinician-Administered PTSD Scale for Children and Adolescents for DSM-IV* (Nader et al., 1996). In addition, a score of 38 is the suggested cutoff for current PTSD in children,

and this cutoff results in a sensitivity of .93 and specificity of .87. Thus, the UCLA provides both a categorical and dimensional assessment of PTSD in children.

Behavioral Assessment System for Children—Second Edition, Self-Report (BASC-2; Reynolds & Kamphaus, 2004a). The BASC-2 is a self-report measure of personality, emotional health, social functioning, and behavior. For this study, however, only five subtests of the BASC-2 were included—Anxiety, Depression, Attention Problems, Hyperactivity, and Attitude to School. Some items on these subtests are rated as *True/ False*, while most others are rated as *Never*, *Sometimes*, *Often*, or *Almost Always* true. Children 12 years of age in the study took a version of the BASC-2 normed for ages 12 to 21, while other children took the version normed for ages 8 to 11. The scales used in this study are largely the same for children under twelve years old and children twelve years old, with only a couple items added for older children.

Structured Developmental History for the BASC-2 (SDH; Reynolds & Kamphaus, 2004b). The SDH is a structured parent interview assessing a wide range of topics, such as family composition, living situation, and developmental milestones. For the purpose of this study, only the Pregnancy, Birth, and Development portions of the interview were administered, and only items relating to alcohol, tobacco, and substance use, as well as child birth weight, were included for further analyses.

Procedure

Because the current study is imbedded within a much larger study of adults, mothers provided a wide range of data not included in this study. All measures for this study were obtained through verbal interview over the course of approximately three visits. The first visit lasted about 1 to 1.5 hours and included the TEI, PSS, and BDI,

which themselves take about 45 minutes to administer all together. Mothers were compensated $15 for this visit. The second visit lasted approximately 3 hours and included administration of the CAPS. The CAPS itself takes about 1 hour to administer, and mothers were compensated $60 for this visit. On the final visit, mothers and one of their children were interviewed in separate rooms. This visit lasted about 2 to 2.5 hours for children and 1.5 hours for mothers. During this visit, the RIAS was administered separately to mothers and children. The RIAS takes 30 to 45 minutes to administer. For children, the VEX-R, UCLA, and BASC were also administered, taking about an hour. In addition, the developmental history portion of BASC parent-report, the CAPI, and the PS were administered to the mothers and took about 1 hour. Mothers were compensated $80 for this visit, and children received a toy of their choice.

Data-Analytic Plan

The following analyses were conducted to address the goals of the proposed study outlined above. First, demographics (i.e., age, sex, education, and income) were generated for the entire sample. Independent samples T-tests were performed to examine group differences between participants with and without PTSD for demographics, trauma exposure, psychopathology (i.e., depression, alcohol and substance abuse), and intelligence and memory. For children, these analyses were also performed to examine potential differences between boys and girls. Next, Pearson correlations were conducted for mother and child variables. These analyses were used as a means to characterize the sample and to guide further analyses.

To assess whether intelligence and memory do, indeed, differ with PTSD in these women, independent samples T-tests were performed to compare groups. In addition, a

binary logistic regression was conducted to examine whether any identified group difference in intelligence or memory predicted PTSD, controlling for trauma exposure, alcohol and substance use, and other psychopathology. For mothers, a categorical analysis was chosen because most studies of adults included in the literature review characterized PTSD categorically, rather than linearly.

To determine if intelligence is related to PTSD in children, independent samples T-tests and Pearson correlations were conducted. To examine whether any identified group difference or relationship in intelligence predicted PTSD when controlling for other relevant child factors (i.e., child trauma exposure and child psychopathology), a hierarchical multiple regression was performed. In this case, PTSD was examined dimensionally because children are less likely than adults to meet full diagnostic criteria for PTSD, possibly resulting in limited analyses. PTSD diagnosis by age 11-12 years is uncommon with a prevalence of .35% in a large (N = 1,901) community sample (Ford, Goodman, & Meltzer, 2003). Furthermore, PTSD symptom clusters may not fully converge as a true syndrome until later stages of pubertal development (Carrion, Weems, Ray, & Reiss, 2002). Similar to other studies of gender differences in PTSD (Danielson et al., 2009; Hyman et al., 2008), child regression analyses were repeated, separately for boys and girls.

Correlational analyses were conducted to examine the relationships among maternal trauma exposure, PTSD, other psychopathology, intelligence and memory, and self-reported parenting behavior. Additional hierarchical multiple regressions were performed to explore potential factors important to child abuse potential, overreactive, and lax parenting.

In an effort to explore the potential impact of pregnancy health behaviors (i.e., use of substances during pregnancy), independent samples T-tests were conducted comparing women who endorsed the use of alcohol, tobacco, or illicit drugs during pregnancy on trauma exposure and PTSD, as well as on their children's trauma exposure, PTSD, and intelligence and memory.

To examine the impact of maternal factors on the potential relationship of intelligence or memory and PTSD symptoms in children, a regression analysis was conducted, controlling for maternal trauma, PTSD, similar to studies of PTSD and intelligence in children that controlled for maternal discipline style (Qouta et al., 2007) and parental intelligence (Delaney-Black et al., 2002), as well as another study of child stress that controlled for maternal trauma (Jovanovic et al., 2011). Again, child regression analyses were repeated, separately for boys and girls.

Hypotheses

Based on the extant literature, several specific hypotheses were formed. It was hypothesized that: 1a) Women with PTSD would demonstrate relative deficits in intelligence compared to women without PTSD, 1b) these deficits would be most pronounced for the VRZ subtest, which involves analogical reasoning, 1c) performance on the WHM subtest, which is comparable to a Wechsler (1997, 2008) Picture Completion subtest, would not differ between groups or would be higher for individuals with PTSD, and 1d) performance on memory subtests would (i.e., VRM and NVM) be lower for individuals with PTSD, given the robust findings regarding memory deficits with PTSD. It was also hypothesized that 2) other factors like trauma and psychopathology would reduce the predictive power of intelligence or memory such that,

once they were taken into account, group differences in intelligence between women with and without PTSD would decrease or be eliminated.

For children, it was hypothesized that: 3a) Intelligence would be negatively related to PTSD symptom frequency, 3b) this negative relationship would be most pronounced for the VRZ subtest, 3c) performance on the WHM subtest would be unrelated to PTSD or would be modestly positively correlated, and 3d) performance on memory subtests would be lower for children with PTSD. Furthermore, like with mothers, it was also hypothesized that 4) other factors like trauma and psychopathology would reduce the predictive power of intelligence or memory such that, once they were taken into account, a relationship between intelligence or memory and PTSD symptoms would decrease or be eliminated.

It was also hypothesized that: 5a) maternal trauma exposure, PTSD, and intelligence and memory would be positively related to child abuse potential, 5b) an overreactive parenting style, and 5c) poor prenatal health (e.g., use of substances during pregnancy). No specific predictions were made regarding lax parenting style.

Finally, it was hypothesized that: 6) Maternal trauma exposure, psychopathology, and intelligence and memory would be positively related to child trauma exposure, psychopathology, and intelligence, and 7) if accounting for maternal trauma exposure, PTSD, and intelligence and memory, the relationship between child intelligence and PTSD would be substantially reduced or eliminated.

Results

Fifty-eight dyads were recruited. Ten mothers were excluded from the analyses due to substantially incomplete data (N = 2), RIAS CIX or CMX scores below 70 (N = 6),

and high current AUDIT scores (N = 2). The two mothers excluded on the basis of the AUDIT reported outlying scores of 19 and 30, well above the sample mean of 2.17 (2.32) or the clinical cutoff of 8 for likely current alcohol use disorder. The final number of mothers included in the analyses is 48. Some analyses reflect different numbers of participants due to minor instances of missing data, and these instances are noted in tables. Three children were excluded based on the RIAS CIX or CMX scores below 70, leaving 55 children in the analyses, including 29 girls and 26 boys. Forty-five intact dyads remained for further analyses.

Mother Only Analyses

Maternal demographics. Women in this sample reported a mean age of 32.94 (5.87) years. A quarter of the women reported not reaching the 12th grade, and over a quarter more reported reaching 12th grade or graduating high school as the highest level of education. Notably, all but 13 of the 48 women included in the study reported a total household monthly income that fell below the federal poverty guidelines (U.S. Department of Health and Human Services, 2013). This was a conservative estimate that included the number of children living in the household and did not account for any other potential adults in the household. An additional four women reported incomes that fell only $500 or less above the line. See Table 5 for a description of the sample, including household monthly income.

Maternal trauma exposure. On the TEI, all women reported at least one traumatic incident over the lifespan (see Table 6). The most common traumatic experience reported by women in this sample was being attacked without a weapon by an intimate partner (*N*

= 28). Twenty-two women reported experiencing at least one incident of childhood physical, sexual, or emotional abuse.

In correlational analyses, neither education nor income were related to traumatic exposure in women (see Table 7). Women's age, however, was positively correlated with self-reported experiences of child abuse, such that older women were more likely to endorse child abuse.

Maternal PTSD. Because the CAPS was administered during a follow-up visit which occurred on a second day of assessment, only 18 women in the study completed the CAPS. Based on the CAPS, two women met criteria for lifetime PTSD, 8 for current, and 10 did not meet criteria. Given the fewer number of women completing the CAPS, the PSS (completed by 45 women) was chosen to be the primary measure for current PTSD in further analyses. Based on the PSS, 20 women met criteria for current PTSD, demonstrating higher scores across symptom clusters, including more frequent self-reported symptoms of avoidance [t (43) = 5.59, $p < .001$], re-experiencing [t (43) = 3.95, $p < .001$], and hyperarousal [t (43) = 8.96, $p < .001$] (see Table 8).

Women with PTSD more often reported experiencing sudden life-threatening illness [χ^2 (1) = 6.54, $p < .05$], witnessing loved ones murdered [χ^2 (1) = 6.41, $p < .05$], being attacked with a weapon by an intimate partner [χ^2 (1) = 4.85, $p < .05$], and being physically abused as a child [χ^2 (1) = 3.74, $p = .05$]. In addition, these women were marginally more likely to report experiencing sexual contact before age 13 years [χ^2 (1) = 3.08, $p = .08$] and more likely to report forced sexual contact between age 14 and 17 years [χ^2 (1) = 3.97, $p < .05$] and as an adult [χ^2 (1) = 6.15, $p < .05$]. Overall, women with

PTSD reported more separate types of trauma, whether including child abuse [t (45) = 3.71, $p < .001$], or excluding it [t (45) = 3.64, $p < .001$] in the analysis.

Women with and without PTSD did not differ in terms of age, years of education, household monthly income (see Table 5).

Relationship of trauma exposure and PTSD to depression and alcohol and substance use in mothers. Women with PTSD reported higher scores for depression [t (29.47) = 4.03, $p < .001$] (See Table 8). Because DAST-10 scores were not normally distributed (Kolmogorov-Smirnov $Z = 2.44$, $p < .001$), a nonparametric test was used to examine group differences, and women with PTSD reported more substance use (Mann-Whitney $U = 105.50$, $p < .01$). Women with and without PTSD did not differ with respect to self-reported alcohol use as assessed by the AUDIT-10.

Trauma, PTSD, depression, and substance abuse correlated with one another as expected in that they tended to increase with one another, though alcohol abuse was only correlated with depression and marginally with hyperarousal (see Table 7). In addition, women's report of age at first traumatic exposure was significantly negatively correlated with both PTSD and depression such that younger age at first trauma was associated with more frequent symptoms.

Women's age correlated with increasing avoidance symptoms and alcohol use. Income was negatively related to depression and positively related to education. Education was unrelated to trauma or psychopathology.

Maternal intelligence and memory. For women, RIAS index and subtest scores were generally correlated with one another as expected (see Table 9), with stronger correlations observed within indices while still correlating with other indices and

subtests. The WHM subtest, however, was inconsistently correlated, even within NIX, and its distribution was determined to be non-normal (K-S $Z = 1.82$, $p < .01$). In addition, the NVM subtest notably correlated only with the overall memory index and not with any other index or subtest.

Women's age was positively correlated with overall memory performance (CMX). Education was positively correlated with VRZ and VRM subtest performance, as well as with CMX, and household monthly income was positively correlated with VRM subtest performance (see Table 10).

PTSD, intelligence, and memory in mothers. Correlational analyses of the relationships between women's psychopathology and trauma exposure with intelligence and memory are seen in Table 10. In these correlational analyses, NVM is significantly positively associated with frequency of PTSD avoidance symptoms. Unexpectedly, age at first traumatic exposure is negatively associated with NVM such that earlier trauma is related to higher nonverbal memory. Intelligence and memory are not associated here with depression, drug use, or lifetime traumatic exposure, and only marginally with alcohol use. Unexpected positive correlations were observed between VIX and VRZ and women's report of experiencing child abuse.

In categorical analyses, women's RIAS performances across nonverbal subtests were in the average range, regardless of PSS PTSD diagnosis, and performances on verbal subtest were in the low average to below average range (see Table 8). Women with and without PTSD differed marginally on their performance on the VRZ subtest [t (37.71) = 1.95, $p = .06$; $d = .58$] and significantly on the NVM subtest [t (43) = 2.31, $p < .05$; $d = -.69$], such that women with PTSD demonstrated relatively lower verbal

reasoning and relatively higher nonverbal recognition compared to their peers without PTSD. These findings lend partial support to hypotheses 1a-c, though hypothesis 1d was not supported at all. No relative disadvantage for memory was observed, and nonverbal recognition was, in fact, higher in women with PTSD.

To examine whether RIAS subtest performance would predict PTSD diagnosis after controlling for trauma and other psychopathology, a binary logistic regression was performed with lifetime traumatic exposure entered in the first step, followed by depression, alcohol, and substance use in the second step, and verbal reasoning and nonverbal memory entered in the final step. Due to a limited sample size, the number of predictors able to be entered into the regression was necessarily conservative. Theory and additional analyses were used to determine the most appropriate predictors. A separate binary logistic regression was used to determine whether child abuse or non-child abuse trauma would better predict PTSD diagnosis (see Table 11). Because non-child abuse trauma better predicted PTSD in this analysis and because child abuse is nevertheless theoretically important to the primary analysis and also correlated with verbal reasoning performance, lifetime traumatic exposure, including child abuse, was chosen as a single predictor to be included.

Table 12 displays the results of the primary analysis for women. Lifetime traumatic exposure significantly predicted PTSD diagnosis in the first step but was no longer predictive when depression, alcohol, and substance use were included. In the final step of the equation, only lower verbal reasoning (p = .06) and higher nonverbal memory (p = .06) marginally predicted PTSD diagnosis. This does not support the second

hypothesis that accounting for these factors would reduce the ability of intelligence and memory to predict PTSD.

Supplemental analysis. An additional logistic regression was performed, excluding AUDIT-C and DAST-10 scores and including years of education and age at first trauma. Neither variable predicted PTSD diagnosis, but verbal reasoning (b = -.32, p < .05) and nonverbal memory (b = .36, p < .05) significantly predicted PTSD above lifetime traumatic exposure (TEI total score). In this supplemental analysis, depression also significantly predicted PTSD (b = .28, p < .05).

Child Only Analyses

Missing data. In an effort to retain as much child PTSD data as possible, given very low rates of PTSD in community samples of children (Ford et al., 2003), an Estimation-Maximization (EM) algorithm was used to estimate the distribution for two missing items (representing two separate children). With all UCLA items from all children entered, assuming a normal distribution and stipulating 25 iterations, the missing items were determined to be missing completely at random, χ^2 (38) = 56.34, p < .05, and were replaced at an estimated value of 1.11 and 1.65 for the items "I have trouble concentrating or paying attention" and "I try to stay about from people, places, or things that make me remember what happened," respectively.

Child demographics. Children in this sample reported a mean age of 9.85 (1.37) years. See Table 5 for description of relevant maternal demographics.

Child trauma exposure. Based on the VEX-R, all but one child reported either witnessing or experiencing some form of violence. Forty-eight children reported witnessing someone yell at someone else, 46 reported witnessing someone getting

arrested, and 40 reported witnessing someone being beaten up (see Table 13). The most common forms of self-reported experienced violence were being yelled at (N = 45) and being spanked (N = 41).

Boys were more likely to endorse witnessing someone being mugged [χ^2 (1) = 3.96, $p < .05$] or stabbed [χ^2 (1) = 4.81, $p < .05$] (see Table 17). They were also more likely to report experiencing being pushed [χ^2 (1) = 5.13, $p < .02$] and beaten up [χ^2 (1) = 5.25, $p < .05$], and marginally more likely to report being chased [χ^2 (1) = 3.00, $p = .08$], slapped [χ^2 (1) = 3.29, $p = .08$], and threatened with a weapon [χ^2 (1) = 3.54, $p = .06$]. Overall, boys reported experiencing more separate types of violence compared to girls [t (49.70) = 2.19, $p < .05$] (see Table 14).

Child PTSD. Based on the UCLA, 11 children were classified as having PTSD, representing 20.75% of the sample. This finding was higher than expected. Children with and without PTSD did not differ in terms of age, sex, or maternal demographics (see Tables 5 and 15). Children with PTSD demonstrated higher scores across PTSD symptom clusters, including more frequent symptoms of re-experiencing [t (51) = 6.24, $p < .001$], avoidance [t (51) = 6.28, $p < .001$], and hyperarousal [t (51) = 4.40, $p < .001$] (see Table 16).

On the VEX-R, children with PTSD more often endorsed that they had witnessed a person being chased [χ^2 (1) = 4.86, $p < .05$], threatened with a weapon [χ^2 (1) = 5.17, $p < .05$], and shot [χ^2 (1) = 4.08, $p < .05$] and were also marginally more likely to report witnessing someone being slapped [χ^2 (1) = 3.64, $p = .06$] and witnessing someone dealing drugs [χ^2 (1) = 2.78, $p < .10$]. They were also more likely to report experiencing being slapped themselves [χ^2 (1) = 8.07, $p < .01$]. Overall, children with PTSD reported

witnessing [t (51) = 1.92, p = .06] and experiencing [t (51) = 1.77, p = .08] marginally more separate types of trauma compared to children without PTSD (see Table 13).

Relationship of trauma exposure and PTSD to other child mental health problems. Based on categorical analyses, children with PTSD reported more anxiety [t (51) = 2.46, $p < .05$] compared to children without PTSD. They did not differ in terms of depression, school problems, attention problems, or hyperactivity (see Table 16). Additional analyses examining potential gender differences found no group differences between girls and boys for PTSD, anxiety, depression, school problems, attention problems, or hyperactivity (see Table 15).

In correlational analyses presented in Table 17, all subscales of the UCLA (the child self-report of PTSD symptoms) correlated with one another, and symptoms of PTSD were more strongly and consistently associated with anxiety, relative to depression, offering some support to the validity of the UCLA as a measure of child PTSD for this sample.

Overall, child mental health and trauma exposure correlated with one another. PTSD symptom frequency correlated as expected with experiencing and witnessing violence. Symptoms of PTSD were also positively correlated with problems with attention and hyperactivity.

Child intelligence and memory. For children, RIAS index and subtest scores were generally correlated with one another as expected (see Table 18), with stronger correlations observed within indices and weaker correlations with separate indices. Like with mothers, children's WHM subtest was inconsistently correlated, and the NVM

subtest correlated only with the overall memory index and not with any other index or subtest.

In categorical analyses, boys and girls did not differ in terms of intelligence, but girls demonstrated a relative advantage on the memory index [t (52) = 3.74, $p < .01$], including both VRM [t (53) = 2.67, $p < .05$] and NVM [t (53) = 2.02, $p < .05$] (see Table 15).

PTSD, intelligence, and memory in children. Children with and without PTSD did not differ with respect to RIAS performance. Children's RIAS scores across nonverbal subtests were in the average range, regardless of UCLA PTSD diagnosis, and performances on verbal subtests were in the average to low average ranges (see Table 16).

Based on correlational analyses, however, significant negative associations were observed between child PTSD and intelligence, particularly verbal intelligence, (see Table 19), supporting hypotheses 3a and 3c. Although 3b was not supported because child VRZ was not related to child PTSD, both VRZ and VIX were marginally negatively associated with witnessing violence. Furthermore, in separate correlational analyses for boys and girls, the relationship between PTSD and intelligence was particularly pronounced on the GWH subtest for boys and on the VRZ subtest for girls (see Table 20). As in the mother analyses, hypothesis 3d (i.e., memory performance would negatively associated with PTSD symptom frequency in children) was not supported because no relationship between memory and PTSD was observed in children.

Child self-report of anxiety, depression, attitude to school, attention problems, and hyperactivity were related neither to intelligence nor to memory (see Table 20).

To explore hypothesis 4, that the strength of an observed relationship between child intelligence and PTSD symptoms would be noticeably reduced if accounting for child trauma and other psychopathology, a multiple linear regression was performed with child age, trauma, anxiety, depression, attention problems, hyperactivity, and verbal intelligence entered into the model (see Table 21). The model itself significantly predicted PTSD symptom frequency in children, and only child anxiety and verbal intelligence were significant predictors within the model, such that higher anxiety and lower verbal intelligence predicted more frequent symptoms of PTSD. This finding was stronger than expected.

Separate multiple linear regression analyses for boys and girls were conducted, replacing VIX with GWH in the boys only regression and VRZ in the girls only regression (see Table 22). Both models significantly predicted PTSD symptom frequency, though only boys' anxiety was a significant predictor. (These analyses were conducted again with VIX, not GWH or VRZ, included in the models, and the patterns of findings remained).

PTSD, Intelligence, and Parenting

Maternal trauma, psychopathology, and parenting. Supporting part of hypothesis 5a, correlational analyses of mothers' trauma, psychopathology, and CAPI scores revealed significant associations between CAPI scores and all measures of maternal trauma and psychopathology, such that PTSD, depression, alcohol use, substance use, and lifetime trauma exposure, including child abuse and non-child abuse trauma, increased alongside child abuse potential (see table 23). In addition, younger age at first trauma was associated with increased CAPI scores.

Overreactive and lax parenting styles also correlated with aspects of trauma and psychopathology. Overreactive parenting was positively associated with depression, alcohol use, and lifetime trauma exposure, though it was not related to PTSD, offering only partial support for hypothesis 5b. It was also not related to a history of child abuse or demographics. Lax parenting, however, was significantly positively associated with PTSD and negatively associated with education.

Maternal intelligence and parenting. Correlational analyses of maternal intelligence, memory, and parenting (i.e., child abuse potential, overreactivity, and laxness) revealed no significant relationship between intelligence or memory and these parenting variables (see Table 24). These findings do not support part of hypothesis 5a that self-report of parenting behavior and attitudes would be associated with intelligence and memory.

Despite no observed correlations, however, between parenting and intelligence and memory in women, exploratory analyses were conducted to examine potential relationships between VRZ and NVM performances and CAPI, overreactivity, and laxness scores because findings from the current study that suggest that VRZ and NVM may be important to PTSD. In addition, child abuse potential and laxness are positively correlated with PTSD, and overreactivity is positively correlated with trauma exposure.

A multiple linear regression was performed to predict CAPI scores, accounting for the CAPI Faking Good Index and lifetime traumatic exposure in the first step; current PTSD, depression, alcohol, and drug use in the second step; and VRZ and NVM performance in the last step (see Table 25). Each model was significant; however, the addition VRZ and NVM did not explain significantly more variance in CAPI scores, and

only the Faking Good validity index and lifetime traumatic exposure in the first step and depression in the second and third steps were significant predictors.

This analysis was repeated both for overreactive and for lax parenting, removing, of course, the CAPI validity index. For overreactive parenting, only the final model was significant, and only VRZ subtest performance and depression were significant predictors (see Table 26); however, the direction of the relationship between VRZ and overreactivity was in the opposite direction expected, such that higher VRZ scores were associated with a more overreactive parenting style. In the analysis for lax parenting, no model was significant (see Table 27).

Maternal self-report of health factors during pregnancy and birth. Mothers who endorsed any use of cigarettes during pregnancy reported more frequent symptoms of PTSD [t (35) = 2.33, $p < .05$] and more lifetime trauma exposure [t (36) = 2.99, $p < .01$] (see Table 28). Self-reported use of illicit drugs during pregnancy yielded only marginally more lifetime trauma exposure [t (36) = 1.70, $p < .10$]. There was no difference in PTSD symptoms or trauma exposure for mothers who reported any use of alcohol.

In a correlational analysis, mother report of child weight at birth was not significantly associated with maternal PTSD symptoms or maternal trauma exposure, nor was it associated with any aspect of maternal intelligence or memory assessed. Together with the analyses of substance use during pregnancy, these findings offer some support for hypothesis 6c, that maternal trauma exposure, PTSD, and intelligence would be associated with negative health behavior during pregnancy, because PTSD and trauma

exposure were higher in women who reported use of cigarettes during pregnancy, but no other variable was found to be important.

Mother and Child Combined Analyses

Relationships among mother and child trauma exposure, mental health, intelligence, and memory. An examination of the relationships between mother and child trauma exposure and mental health yielded minimal findings (see Table 29). Only mothers' report of experiencing child abuse was significantly positively associated with children's report of experiencing violence. No other findings were significant.

Correlations between mother and child RIAS performances were observed, particularly for verbal subtests (see Table 30). Nonverbal performance less consistently correlated between mothers and children, and child memory performance was almost entirely independent of mother memory performance. Furthermore, only one direct comparison (i.e., a comparing mother and child performance on the same subtest or index) yielded a significant relationship, specifically for VRZ.

No relationships were observed between maternal trauma and mental health and child RIAS performance (see Table 31). Maternal age was significantly negatively correlated with child VRM scores, and maternal education was significantly positively correlated with child VRZ scores.

These results from the analyses presented in Tables 29-31 offer limited support hypothesis 6, that mother and child trauma, mental health, and intelligence and memory would be related.

The relationships between parenting and child trauma exposure, mental health, intelligence, and memory. No significant relationships were observed between parenting

(i.e., child abuse potential, overreactivity, and laxness) and any measured child outcomes, including trauma exposure, mental health, intelligence, and memory (see Table 32).

Mother report of use of substances during pregnancy and child birth weight and child trauma exposure, mental health, intelligence and memory. Children of mothers who reported alcohol, cigarette, or illicit drug use during pregnancy reported equivalent trauma exposure and PTSD symptoms (see Table 28). Unexpectedly, children whose mothers reported alcohol use performed better on the VRM subtest, relative to their peers [t (37) = 2.07, $p < .05$], and children whose mothers reported use of illicit substance earned higher scores for CIX [t (35) = 2.22, $p < .05$], GWH [t (35) = 2.03, $p < .01$], NIX [t (37) = 2.35, $p < .05$], WHM [t (16.73) = 3.94, $p < .05$] (see Table 33). Endorsement of substance use during pregnancy was limited, resulting in very small groups, making interpretations of these findings tenuous, at best. No differences were observed between children whose mothers did or did not report use of cigarettes during pregnancy.

The relationship between mother-reported child birth weight and child outcomes was similarly difficult to interpret. Overall, birth weight was not different between child PTSD groups (see Table 16) and was not associated with child PTSD symptom frequency (see Table 19). It was, however, significantly positively associated with child-reported witnessing violence and significantly negatively associated with VRM subtest performance, particularly for boys (see Table 20). It is possible that these data are impacted by inaccurate recall of child birth weight.

PTSD, intelligence, and memory in children, accounting for relevant maternal factors. To test whether child verbal intelligence would continue to be associated with child PTSD symptoms, even after accounting for potentially important maternal factors, a

hierarchical multiple regression was performed with mother verbal intelligence, trauma, and PTSD symptoms entered in the first step, child age entered in the second step, child violence exposure entered in the third, and child verbal intelligence entered in the final step (see Table 34). No model significantly predicted PTSD in children.

This analysis was conducted again, separately for boys and girls, to examine potential differences in patterns of findings. As in previous analyses, VIX was replaced with GHW for boys and VRZ for girls. For boys, no model significantly predicted PTSD symptoms (see Table 35). For girls, only the third model approached significance, and mother PTSD symptoms and child violence exposure only marginally predicted child PTSD symptoms in that model (see Table 36).

Although these findings may lend support to hypothesis 7, that an observed relationship between child intelligence or memory and PTSD would be reduced when accounting for maternal factors, the maternal factors included in these analyses do not, themselves, account child PTSD as it is presented here.

Discussion

Findings from this study of a low-income, urban, African American sample of 48 mothers and 55 children recruited from hospital clinic waiting areas lend support to some of the hypotheses proposed. As expected, women with PTSD performed relatively poorer on a verbal analogical reasoning task relative to women without PTSD. This result is consistent with the overall literature review, finding that individuals with PTSD reliably perform relatively poorer on measures of analogical reasoning. No difference was observed for a measure of nonverbal reasoning (i.e., WHM subtest) comparable to a Wechsler (1997, 2008) Picture Completion subtest. This null finding was expected, and

no other differences in performance were noted for women with PTSD in this sample. It is possible that these limited findings reflect a less pronounced difference of intelligence in samples well-matched for socioeconomic status. In fact, the magnitude of the group difference in general intelligence was d = .37, just below the lower range of the confidence interval for the weighted mean effect size reported in the literature review.

An unexpected group difference emerged for memory, however, in that individuals with PTSD demonstrated a relative advantage for nonverbal recognition, and this finding was not predicted by the previous literature review. And, contrary to expectations, lower analogical reasoning and higher nonverbal recognition marginally predicted current PTSD diagnosis, even when accounting for lifetime traumatic exposure, current alcohol and substance use, and current depression. It is important, however, to note that, although marginally significant differences were observed, these differences do not represent clinically-significant differences (i.e., regardless of PTSD status, VRZ T-Scores were in the below average range, and NVM T-Scores were in the average range). Nevertheless, even in this highly-traumatized sample, analogical reasoning and nonverbal recognition appear to play potentially important roles in conferring risk, but what those roles might be is difficult to isolate given the broad nature of the constructs.

Analogical reasoning is a process by which an individual attempts to understand a novel situation by drawing on situations already experienced by or familiar to the individual. In using analogy, one must access the mental representation of the base (i.e., the known situation), attempt to align that representation with the target (i.e., the novel situation), and evaluate the fit of that alignment (Gentner & Markman, 1997). In so doing, the individual may draw inferences about the target, including potential

similarities. A traumatic experience may present as a novel situation, and an individual may attempt to understand this new experience by employing analogical reasoning—looking to past experiences, aligning the old and new experiences, and drawing inferences and noting commonalities.

Many alignments, however, will not be adequate without revisions to the mental representations of either the base or the target. One such strategy to improve match is re-representation, which involves changes to the base, target, or both, and the process of re-representation itself may even result in changes to how a base is recollected or a how a target is perceived (Gentner et al., 1997; Kokinov, Vankov, & Bliznashki, 2009). This process appears comparable to the concepts of accommodation and assimilation in cognitive theories of PTSD (Sobel, Resick, & Rabalais, 2009). Assimilation refers to the process of incorporating new information into an existing, unchanged schema. For example, if someone holding the belief that "If I live by the rules, bad things won't happen to me" experiences a traumatic event, rather than changing the belief, that individual may perceive the trauma as evidence of his or her own failings. This change may indicate a re-representation of the target situation. Accommodation, on the other hand, refers to changes to schema itself and may be observed if, for example, the individual now believes that "No matter how I live, bad things will happen to me," and my indicated a re-representation of the base.

Both assimilation and accommodation are necessary cognitive processes in daily life and are only considered problematic if the revisions are grossly inaccurate or overgeneralized. Such problematic revisions are a core feature of PTSD, and they typically result in inaccurate negative beliefs about the self (e.g., "I'm a fool"), other

people (e.g., “All men are dangerous”), and the world around them (e.g., “There is no safe place for me”). It is possible that, in the presence of poor analogical reasoning abilities, an individual’s attempt to align mental representations may result in inappropriate re-representations of the target, base, or both, encouraging the development of maladaptive cognitive schemas, which, in turn, influence behavioral choices (e.g., avoidance) that maintain distress.

Though the finding that women with PTSD perform more poorly on a test of analogical reasoning appears to fit with existing literature and theoretical models of PTSD, the finding that women in this study with PTSD demonstrated a relative advantage for nonverbal recognition is surprising, particularly given the plethora of research suggesting PTSD is associated with memory impairment. One could argue that relative deficits are reported in the literature less consistently for visual memory than for verbal memory, so the current finding is less anomalous than initially presumed; however, no known study reports an advantage for visual memory in individuals with PTSD.

It is possible that nonverbal recognition was not adequately measured in this study. Results from both adults and children showed that performance on the NVM subtest was unrelated to performance on any other subtest, and this finding is consistent with another study reporting poor *g*-loadings for this subtest, including, like in the current study, no correlation even with verbal memory performance (Nelson, Canivez, Lindstrom, & Hatt, 2007) Still, one would not expect that women with PTSD would perform better on this task, particularly when accounting for other factors like trauma, depression, and alcohol and substance use.

It is important, then, to determine what this subtest is measuring. What ability is being tapped, and why would women with PTSD show a relative advantage for it? For each question on this subtest, examinees are asked to carefully observe a geometrical pattern on a page for five seconds and to immediately select that pattern from six patterns presented on the following page. Unlike on the VRM subtest that requires examinees to freely recall verbally-presented passages of text, the NVM subtests does not require the reproduction of the stimulus—only the recognition of it. In other studies specifically measuring visual reproduction (i.e., drawing a figural stimulus from memory), either no group differences were observed or individuals with PTSD showed a relative deficit in performance (Brandes et al., 2002; Gil et al., 1990; Marx et al., 2009). Recognition and reproduction may be associated with separate neural substrates, specifically the perirhinal cortex and the hippocampus, respectively, and the perirhinal cortex is markedly less involved than the hippocampus in the processing of spatial information (Brown & Aggleton, 2001; Bussey, Duck, Muir, & Aggleton, 2000). Furthermore, individuals with severe cognitive impairment, including lesions to the hippocampus, are able to perform forced-choice recognition tasks, but they are unable to engage in retrieval (Brown & Aggleton, 2001). Thus, it is possible that the NVM subtest is more likely to be capturing visual familiarity than spatial information that would otherwise be captured by a visual reproduction task.

This distinction is important if one considers the possibility that the relative advantage for nonverbal recognition observed in women with PTSD may be indicative of a difference in visual cognitive style. Visual cognitive style is often divided into two sub-styles, specifically object visualization and spatial visualization. Individuals are typically

categorized as either verbalizers or visualizers and, if visualizers, categorized as an object or spatial visualizer. Object visualization refers to the ability or tendency to process visual information holistically (i.e., poorer performance on the Embedded Figures Test) and in terms of characteristics like shape and color, whereas spatial visualization refers to the ability or tendency to process visual information more analytically or piece by piece and in terms of the relationships among the different objects within the image (Kozhevnikov, Blazhenkova, & Becker, 2010; Kozhevnikov, Kosslyn, & Shephard, 2005). Furthermore, this distinction is firm in that an individual is very unlikely to demonstrate both high object and high spatial visualization, due to competing visual resources and separate neural pathways, specifically the ventral and dorsal pathways, respectively (Kozhevnikov et al., 2010). Given the demands of the NVM subtest, it is possible that a relatively better performance could be, at least in part, tapping into an object visualization style.

More convincing evidence of this possibility (e.g., higher nonverbal recognition, in addition to lower spatial reasoning) is not available because the RIAS does not provide a straightforward measure of spatial abilities, though one could consider verbal analogical reasoning to be, in part, a result of spatial processing. This is not entirely unreasonable given that the hippocampus is activated during analogical reasoning tasks (Cohen et al., 1999; Eichenbaum, Otto, & Cohen, 1992; Luo et al., 2003; Qiu, Li, Chen, & Zhang, 2008) and given that other aspects of language comprehension have been shown to use visuospatial resources (Fincher-Kiefer & D'Agostino, 2004; Levine & Ortigo, 2006; Richardson, Spivey, Barsalou, & McRae, 2003). If measured directly, would object

visualization present more often in individuals with PTSD? How would such a finding contribute to understanding the relationship between intelligence and PTSD?

Object visualizers are more likely to exhibit a field dependent style in that they may encode visual information more rapidly and holistically and demonstrate difficulty disembedding information from a complex visual field (Kozhevnikov et al., 2005). As previously discussed, field dependent individuals may also be more likely to recall incidental social information for events or tasks (Crutchfield et al., 1958; Fitzgibbons & Goldberger, 1971), and trauma memories are characterized by the inclusion of a greater number of stimuli (Foa & Kozak, 1993). Because spatial visualization is a slower, more analytical process, it is possible that a spatial visualizer encountering a traumatic event may encode fewer stimuli—particularly fewer irrelevant stimuli—into the resulting trauma memory and, thus, be less likely to experience triggering stimuli in future situations. Furthermore, if that individual demonstrates relatively intact verbal analogical reasoning, stimuli that are encountered again may be less likely to be inappropriately associated with beliefs about danger.

An interesting finding in the current study regarding the age at first trauma demonstrated that women who reported being traumatized at earlier ages also tended to report more symptoms of depression and PTSD. This finding appears consistent with previous research of the impact of childhood trauma on adult mental health (Horwitz, Widom, McLaughlin, & White, 2001), but an additional finding that higher nonverbal recognition was also related to earlier trauma has not been previously reported. What this relationship might mean is not clear. One possibility is that women with better nonverbal recognition are simply able to recall earlier experiences, particularly those occurring at

ages characterized by greater reliance on visual over verbal processes. On the other hand, earlier exposure to trauma may result in cognitive changes that facilitate the development of nonverbal recognition abilities. Without available data, whether either of these explanations is plausible is impossible to examine.

Verbal intelligence was a significant predictor of PTSD in children, even when accounting for child age, trauma exposure, anxiety, depression, attention problems, and hyperactivity. Only lower verbal intelligence and higher anxiety predicted more frequent symptoms of PTSD. In subsequent analyses accounting, instead, for mother verbal intelligence, lifetime trauma exposure, and PTSD, as well as child age and exposure to violence, neither verbal intelligence, nor any other variable included in the analysis, predicted child PTSD symptoms. Potential reasons for the mixed findings in children are plenty, including that children were well-matched in terms of SES, that all but one child reported exposure to some form of violence, that child report of PTSD symptoms was notably higher than in other community samples, that all mothers reported trauma exposure, and that mothers, too, reported high frequency of PTSD symptoms. It is possible that this sample is saturated with poverty and stressors to the point that these factors become less predictive, and other individual risk factors (e.g., verbal intelligence and anxiety) become more uniquely predictive.

Interestingly, in exploratory analyses of potential gender differences, boys and girls differed with respect to which specific ability underlying verbal intelligence—vocabulary or analogical reasoning—was related to PSTD symptom frequency. PTSD symptoms in boys appeared to be more strongly negatively related to vocabulary, and symptoms in girls appeared to be more strongly negatively related to analogical

reasoning. Given the results of analyses of mothers, it is possible that relative deficits in analogical reasoning could be more important for women than for men.

How intelligence may impact parenting, and thus potentially the intergenerational transmission of trauma and PTSD, remains unclear. Contrary to findings from a number of studies demonstrating a relationship between relative deficits in cognitive abilities and parenting attitudes and behavior (e.g., Ammerman & Patz, 1996; Deater-Deckard et al., 2010; Kwon, 2007), data from this sample do not support a relationship between parenting and maternal intelligence or memory, nor do these data demonstrate any relationship between parenting and child trauma, mental health, intelligence, or memory. It is possible that the CAPI and the PS are not an adequate measures of abuse potential or parenting style, respectively, in this population, but this explanation is unlikely, given the use of these measures in similar samples of low-income, at-risk families (de Paúl & Domenech, 2000; Medora, Wilson, & Larson, 2001; Steele et al., 2005), as well as the strong correlations in the current study with noted risk factors (i.e., mother mental health and trauma exposure) in the expected direction. Furthermore, although one study of PTSD and depression in children and adolescents found no relationship between maternal discipline style and child or adolescent intelligence, PTSD, or depression (Qouta et al., 2007), still many other studies provide evidence to the contrary (e.g., McCloskey, Figueredo, & Koss, 1995; Repetti, Taylor, & Seeman, 2002).

Exploratory analysis of child abuse potential, overreactivity, and laxness were performed to examine whether the pattern of findings observed for maternal PTSD—specifically that analogical reasoning and nonverbal recognition marginally predicted PTSD, even when accounting for trauma exposure and other psychopathology—would be

useful in understanding parenting behavior. The findings were null for both child abuse potential and laxness; however, verbal reasoning emerged as a significant predictor of overreactive parenting, even when accounting for maternal trauma and psychopathology. Surprisingly, the direct of this finding was the opposite of what was expected. Unlike the relative deficits of analogical reasoning observed in PTSD, higher analogical reasoning predicted maternal overreactivity. What this finding could mean—if anything—is unclear. Further exploration of parenting and child outcomes in this sample is warranted.

The generally weak relationship between mother and child RIAS performance is notable in that there are only marginal one-to-one correlations between mother and child scores for general intelligence and verbal intelligence, and no one-to-one correlations between mother and child scores for nonverbal intelligence, general memory, or for GWH, OIO, WHM, VRM, or NVM subtest scores. A mother's performance on one of these particular subtests was not related to her child's performance on that same subtest. In fact, only mother and child VRZ scores were significantly related. This pattern could suggest both that the RIAS is not an adequate measure of intelligence and memory and could be tapping into different cognitive processes than those for which it is intended (Nelson et al., 2007) and that other factors (e.g., poverty, stress) may reduce heritability (Tucker-Drob, Rhemtulla, Harden, Turkheimer, & Fask, 2011; Turkheimer, Haley, Waldron, D'Onofrio, & Gottesman, 2003).

After all, the large majority of women in this sample reported incomes at or below the poverty line, all 48 reported lifetime traumatic exposure, 22 reported experiencing at least one type of child physical, sexual, or emotional abuse, and all but one of the 55 children reported at least one incident of exposure to violence. Moreover, 20 mothers and

11 children met criteria for PTSD based on self-report measures, well above the rates reported in the literature (Breslau, 2002; Ford et al., 2003). It is important to note that participant eligibility and recruitment included neither a requirement to endorse trauma, nor report of any PTSD symptoms. Furthermore, though efforts were made to recruit participants from an outpatient psychiatry for children, only one child included in this study was so recruited. This sample clearly represents a highly stressed and resource-poor group of families, and although no conclusions about heritability should be drawn from these data, other studies would suggest that performance on measures of intelligence in such a sample may not be as consistently predicted by heritability. Furthermore, performance on measures of nonverbal intelligence should theoretically be less impacted than verbal intelligence by low socioeconomic status (Cattell, 1963), and the findings from this study demonstrate average nonverbal intelligence and low average to below average verbal intelligence, regardless of PTSD status. It is possible that this pattern of findings is the result not only of PTSD, but also of poverty and trauma. Interestingly, children's performances on the VRZ subtest was not different than the mothers' performances overall, and VRZ performance was positively correlated between mothers and children. It is possible that, in addition to the potential genetic contribution to analogical reasoning, mothers may not model the use of analogy for their children, thereby increasing children's risk of inappropriate or inaccurate re-representations of later traumatic events, elevating their risk for psychopathology.

Study Limitations

The findings of the current study are limited by a number of factors, including small sample size, which made analysis of group differences for current versus lifetime

PTSD impossible. Such an analysis may have helped to determine if observed differences in intelligence are reduced or even erased once symptoms have resolved. In addition, analyses of children, particularly those separated by gender, were very limited and may yet produce more substantial findings with a greater number of participants.

The findings were also limited as a result of potential problems with the selected measurement of intelligence and, particularly, memory (e.g., Nelson et al., 2007), which necessitates cautious interpretation of the results of the study. Furthermore, a direct assessment of spatial processing abilities could enhance theory development regarding the relationship between intelligence and PTSD.

Another larger limitation is the use of retrospective reports of traumatic exposure for both mothers and children. Reliance on such reporting substantially limits the ability to make causal attributions to either intelligence or trauma and PTSD. Only a prospective design can start to address whether group differences in intelligence are observed prior to traumatic exposure and symptom outcome.

Unfortunately, though data were gathered regarding mother-report of use of alcohol, cigarettes, or other substances during pregnancy, as well as child birth weight, these data may be impacted by small sample size, underreporting, and inaccurate recall. Some correlations and group differences were observed for these variables that suggest that the intended construct may not be adequately measured (e.g., significant negative correlation between child verbal memory and mother-reported child weight at birth). Pre- and perinatal factors, as well as early developmental milestones, are very important in establishing risk for a variety of intellectual and mental health problems, and, going forward, these constructs should be measured with greater precision.

Study Strengths

Despite these limitations, the current study demonstrates several strengths. Specifically, the groups of women with and without PTSD were well-matched in terms of socioeconomic status and self-report of at least one incident of traumatic exposure (i.e., use of traumatized comparison group). Both of these factors are important because they impact whether conclusions may be drawn regarding intelligence and PTSD, or, rather, if poverty, stress, and trauma are impacting the findings. Even in this low-income, highly-traumatized sample, women with PTSD performed marginally more poorly on a measure of analogical reasoning.

This study also included and specifically analyzed not only general intelligence, but also multiple tests of specific cognitive abilities, including memory, which allowed for greater theoretical enrichment of the relationship between PTSD and intellectual functioning. The use of a single subtest, such a measure of vocabulary, may have yielded no findings on its own, which illustrates the importance of how intelligence is operationalized, particularly in a low-income sample in which aspects of intelligence may not be as strongly interrelated as in other samples characterized by fewer environmental pressures or stresses.

Finally, a major strength of this study is its inclusion of both mothers and children in the design, which allowed for examination of the impact of mother intelligence, trauma, and PTSD on child-report of the same. Other studies of PTSD and intelligence in children have not included parental factors to this extent.

Conclusion

Results from the current study demonstrated group differences in intelligence between women with and without PTSD, particularly a relative advantage for nonverbal recognition and a relative disadvantage for analogical reasoning in those with PTSD. This pattern of findings may suggest facilitated encoding of trauma-related stimuli, in addition to inappropriate associational processing of those stimuli and beliefs about danger.

In children, verbal intelligence predicted PTSD symptom frequency, even accounting for child trauma exposure and psychopathology, but maternal factors (i.e., trauma, PTSD, intelligence, and parenting), were largely unrelated to child outcomes. Although evidence of an intergenerational impact of intelligence on risk for PTSD was not directly observed in this study, other studies should consider the role of parenting on the relationship between children's intelligence and symptoms of PTSD. After all, despite relatively weak correlations between mother and child intelligence and memory, child analogical reasoning was positively correlated with mother analogical reasoning, suggesting that this particular aspect of intelligence may yet be an important marker of risk for these children as they encounter future stresses and trauma.

Measures of analogical reasoning could also represent relatively inexpensive assessments of risk for psychopathology, and, furthermore, analogical reasoning, itself, can be encouraged through training and education (Thompson, Gentner, & Loewenstein, 2000), making it a potential point for intervention.

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Table 1

Symptoms and Diagnostic Criteria for PTSD from the DSM-IV-R

Criterion A—Traumatic Stressor
Exposure to a traumatic event in which both of the following were present: 1. Experiencing, witnessing, or being confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others. 2. Responding with intense fear, helplessness, or horror. In children, this may be expressed instead by disorganized or agitated behavior.
Criterion B—Intrusive Re-experiencing—one or more symptoms
The traumatic event is persistently re-experienced in the following ways: 1. Recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions. In young children, repetitive play may occur in which themes or aspects of the trauma are expressed. 2. Recurrent distressing dreams of the event. In children, there may be frightening dreams without recognizable content. 3. Acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated). In young children, trauma-specific reenactment may occur. 4. Intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

5. Physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.

Criterion C—Avoidance/ Numbing—three or more symptoms

Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by the following:

1. Efforts to avoid thoughts, feelings, or conversations associated with the trauma
2. Efforts to avoid activities, places, or people that arouse recollections of the trauma
3. Inability to recall an important aspect of the trauma
4. Markedly diminished interest or participation in significant activities
5. Feeling of detachment or estrangement from others
6. Restricted range of affect (e.g., unable to have loving feelings)
7. Sense of a foreshortened future (e.g., does not expect to have a career, marriage, children, or a normal lifespan)

Criterion D—Hyperarousal—two or more symptoms

Persistent symptoms of increased arousal (not present before the trauma), as indicated by the following:

1. Difficulty falling or staying asleep
2. Irritability or outbursts of anger
3. Difficulty concentrating
4. Hypervigilance

5. Exaggerated startle response

Criterion E—Duration of the disturbance is more than 1 month.

Criterion F—The disturbance causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.

Specifiers

Acute if duration of symptoms is less than 3 months

Chronic of duration of symptom is 3 months or more

Delayed onset if symptoms begin more than 6 months after stressor

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Table 2

Descriptions and Findings for Studies included in the Review

Study	Sample Description	*N*[a] Age % Male	PTSD Assessment	IQ Measure	IQ *ES*
Bremner et al. (2004)	Adult women with history of childhood sexual abuse with and without PTSD	43 32.8 (7.5) 0	SCID-IV (First et al., 1995)	WAIS-R—AR, VC, PA, and BD (Wechsler, 1981)	.48
De Bellis & Kuchibhatla (2006)	Outpatient children and adolescents with PTSD and healthy community sample	156 12.0 (2.3) 51.3	Modified K-SADS-PL (Kaufman et al., 1997)	WISC-R—VC, DS, BD, and OA (Wechsler, 1974)	.72
De Bellis et al. (2002)	Outpatient children and adolescents with PTSD compared and community sample	104 12.1 (2.3) 58.7	Modified K-SAD-PL	WISC-R— VC, DS, BD, and OA	1.04

De Bellis et al. (2002b)	Outpatient children and adolescents with PTSD and healthy community sample	94 11.6 (2.9) 47.9	Modified K-SAD-PL	WISC-R— VC, DS, BD, and OA	.30
Delaney-Black et al. (2002) †	Children with and without trauma-related distress	299 6.9 52	Levonn Scale (Martinez & Richters, 1993)	WPPSI-R (Wechsler, 1989)	non-sig
Diamond et al. (2001) †	Adult survivors of child maltreatment	50 33.0 (14.1) 36.0	PTSD Symptom Checklist (Southwick et al., 1993)	WAIS-R	sig
Emdad & Sondergard (2006)	Iraqi refugees in Sweden with and without PTSD	50 38.6 (7.3) 100	Clinician-Assessed Interview for PTSD—III (Keane et al., 1996)	Raven's Progressive Matrices (Raven, 1992)	.48

Engelhard et al. (2001)	Vietnam combat veterans with and without PTSD	30 53.0 (2.8) 100	PSS (Foa et al., 1993)	SILS-R (Zachary & Shipley, 1986)	.58
Geuze et al. (2008)	Dutch veterans with and without PTSD	50 34.6 (5.0) 100	SCID-IV and CAPS (Blake et al., 1998; Weathers et al., 2001)	WAIS-III—SI, VC, BD, PA (Wechsler, 1997)	.33
Gilbertson et al. (2007)	MZ twins with and without PTSD; pairs included combat PTSD and unexposed twin	41 52.1 (2.7) 100	CAPS	WAIS-III—VC, AR, PA, and BD	.72
Golier et al. (2002)	Holocaust survivors with and without PTSD and non-exposed Jewish comparison group	97 68.6 (6.1) 37.0	SCID-IV and CAPS	WAIS-R—BD and VC	.75

Gurvits et al. (1996)	Vietnam veterans with and without PTSD	14 46.0 (2.3) 100	CAPS	WAIS-R—IN, DS, AR, PA, BD	.67
Gurvits et al. (2000)	Vietnam veterans with and without PTSD	59 47.3 (2.9) 100	CAPS	WAIS-R—IN, AR, PC, PA, and BD	1.15
Hart et al. (2008)	Former WWI or Korean Conflict POWs with and without PTSD	25 80.1 (2.0) 100	CAPS	North American Adult Reading Test (Blair & Spreen, 1989)	1.38
Johnsen et al. (2008)	Refugees and immigrants from the former Yugoslavia, Chile, and the Middle East with and without PTSD	42 38.1 (9.4) 76.2	MINI (Sheehan et al., 1998) and CAPS-R (Blake et al., 1998)	WAIS-R—PC and SI	.61

Kivling-Boden & Sundbom (2003)	Refugees in Sweden with and without PTSD from the former Yugoslavia	34 38.5 61.8	Harvard Trauma Questionnaire (Mollica & Caspi-Yavin, 1991)	Swedish WAIS-R (Wechsler, 1992)—PC and PA; Figure Classification Test and Block Design Test (Bergman et al., 1985)	.72
Mennen (2004)	Maltreated Latino children referred to child service agencies	31 9.3 (2.1) 35.5	PTSD Inventory (Famularo et al., 1990)	WISC-R—Spanish speaking norms	.27
Nixon et al. (2004)	Outpatient women seeking treatment for PTSD as a result of rape	73 32.3 (9.7) 0	PSS	The Quick Test (Ammons & Ammons, 1962)	-.04

Saigh et al. (2006)	Outpatient youth with and without PTSD not as a result of maltreatment and non-traumatized comparison	115 13.1 (2.5) 84.6	DICA-R (Reich & Welner, 1988)	WISC-III—IN, SI, AR, VC, CM, DS, PC, CD, PA, BD, and OA (Wechsler, 1991)	.73
Saltzman et al. (2006)	Maltreated children recruited from social service departments or mental health clinics	59 10.7 (1.9) 57.6	CAPS-CA (Nader et al., 1996)	WASI—VC, SI, BD, and MR (Wechser, 1999)	.13
Silva et al. (2000)	Outpatient traumatized youth with and without PTSD	59 9.9 (4.1) 66.1	KID-SCID (Hein et al., 1994)	WISC-R	.14
Stein et al. (2002)	Women with and without PTSD as a result of partner violence and non-traumatized comparison group	61 34.5 (9.8) 0	CAPS	WAIS-III—VC	.26

Sutker et al. (1991)	Former WWII or Korean Conflict POWs with and without PTSD; Recruited from VA Hospital	161 63.9 (4.5) 100	DIS (Robins et al., 1981)	WAIS-R	.14
Thomas & De Bellis (2004)	Outpatient maltreated children and adolescents with PTSD and healthy community comparison	182 11.7 (2.5) 48.9	Modified K-SADS-PL	WISC-R—VC, DS, BD, and OA	.67
Twamley et al. (2004)	Undergraduate students with and without PTSD and non-traumatized comparison	230 19.0 (2.0) 27.0	Posttraumatic Stress Diagnostic Scale (Foa, 1995)	American National Adult Reading Test (Grober, Sliwinski, & Korey, 1991)	-.32
Vasterling et al. (1998)	Operation Desert Storm veterans with and without PTSD	43 35.7 (9.2) 74.4	SCID-III-R (Spitzer et al., 1990)	WAIS-R—VC	.96

Vasterling et al. (2002)	Vietnam veterans with and without PTSD	47 50.8 (4.0) 100	SCID-IV	WAIS-R—IN and VC	.66
Werner et al. (2009)	Outpatients in Germany with PTSD and healthy community comparison	24 34.6 (6.7) 33.3	SCID-IV and CAPS	Vocabulary Test (Schmidt & Metzler, 1992)	-.14
Yehuda et al. (1995)	Outpatient combat veterans with PTSD and healthy community comparison	32 44.4 (4.8) 100	CAPS and Mississippi Scale for Combat-Related PTSD (Keane et al., 1988)	WAIS-R—VC and BD	.58
Breslau et al. (2006)†	Michigan cohort born 1983-1985 assessed at age 6 and again at age 17	713 17.0 47.12	DIS for DSM-IV	WISC-R at age 6	.19 (pre)

Gil et al. (1990)	Outpatient Israeli veterans with PTSD or other disorder (additional comparison group not included in meta-analysis)	36 30.2 (9.2) 100	Two clinicians' consensus using DSM-III criteria	Pre: uncited Army IQ Test; Post: WAIS—IN, CM, SI, DS, PC, BD, DSS	.32 (pre) .45 (post)
Kaplan et al. (2002) †	Israeli cohort screened for military eligibility; PTSD diagnosed at later time-point; Includes non-combat trauma	1800 16-17 100	Chart review; diagnosis based on DSM-III-R and DSM-IV criteria	Raven's Progressive Matrices-R, Similarities-R, Arithematic-R, OTIS-R (Lezak, 1995)	.17 (pre)

Koenen et al. (2007) †	Members of New Zealand cohort measured at age 5, 7, 9, 11, 26, and 32; With and without exposure to trauma and PTSD	265 32.0 52.0	DIS for DSM-IV (Robins et al., 1995)	Stanford-Binet (Thorndike et al., 1986) at age 5; WISC-R at ages 7, 9, and 11	.28 (pre)
Kremen et al. (2007) †	MZ and DZ twin pairs from Vietnam Era Twin Registry; pairs included combat PTSD and unexposed twin	2386 41.9 (2.6) 100	DIS for DSM-III-R	AFQT (Bayroff & Anderson, 1963)	.25 (pre)
Macklin et al. (1998)	Vietnam combat veterans with and without PTSD; Recruited from research at VA Hospital	90 49.3 (2.6) 100	CAPS	Pre: AFQT; Post: WAIS-R—DS, VC, AR, PC, PA, and BD—or SILS-R	.83 (pre) .64 (post)

Parslow & Jorm (2007) †	Australian cohort measured in 1999 or 2000 and again in 2003 or 2004; All experienced bushfire in 2003	993 26.7 (1.5) 48.0	Trauma Screening Questionnaire (Brewin et al., 2002)	Spot the Word Test-Version A (Baddeley et al., 1993)	sig (pre) sig (post)
Thompson & Gottesman (2008)†	Vietnam combat veterans with and without PTSD recruited from the Vietnam Experiences Study (CDC, 1988)	2375 37.8 (2.3) 100	Modified DIS-III-A with PTSD Module (CDC, 1989)	AFQT	mixed (pre)
Qouta et al. (2007) †	Members of Palestinian cohort assessed at age 10-11 in 1993, again in 1996 and 2000	65 17.6 (.9) 47.7	Adolescent Version of the Reaction Index (Pynoos, et al., 1987)	The Selah Picture IQ Task (Hefni, 1980) at age 10-11	.10 (pre)

Note: [a]Reflects number, age, and sex of entire sample, with additional comparison groups included; †study not included in meta-analysis (study effect size may still be presented if able to calculate); sig = ES not able to be calculated (e.g., regression, variables "controlled for," etc.), but results were overall significant; non-sig = ES not able to be calculated, but results were overall non-significant; mixed = ES not able to be calculated, but there were mixed results; Measures Abbreviations (in alphabetical order): AFQT = Armed Forces Qualifying Test; CAPS = Clinician Administered PTSD Scale; CAPS-CA = Clinician-Administered PTSD Scale for Children and Adolescents; DICA-R = Diagnostic

Interview for Children and Adolescents-Revised; DIS = Diagnostic Interview Schedule; KID-SCID = Structured Clinical Interview for DSM-IV, Childhood Version; K-SADS-PL = Scheduled for Affective Disorders and Schizophrenia for School-Age Children—Present and Lifetime Version; MINI = Mini-International Neuropsychiatric Interview; OTIS-R = Verbal Intelligence Test Adapted from United States Army Alpha Instructions; PSS = Posttraumatic Symptom Scale; SCID-III-R = Structured Clinical Interview for DSM-III-R; SCID-IV = Structured Clinical Interview for the DSM-IV; SILS-R = Shipley Institute of Living Scales-Revised; WAIS-R = Wechsler Adult Intelligence Scale-Revised; WAIS-III = Wechsler Adult Intelligence Scale-Third Edition; WASI = Wechsler Abbreviated Scales of Intelligence; WISC-R = Wechsler Intelligence Scale for Children-Revised; WISC-III = Wechsler Intelligence Scale for Children-Third Edition; WPPSI-R = Wechsler Preschool and Primary Scale of Intelligence-Revised; IQ Index and Subtest Abbreviations (in alphabetical order): AR = Arithmetic; BD = Block Design; CD = Coding; CM = Comprehension; DS = Digit Span; DSS = Digit Symbol Substitution; FSIQ = Full Scale or General IQ; IN = Information; MR = Matrix Reasoning; NVIQ = Nonverbal or Performance IQ; OA = Object Assembly; PA = Picture Arrangement; PC = Picture Completion; SI = Similarities; VC = Vocabulary; VIQ = Verbal IQ

Table 3

Results of Categorical Moderator Analyses (Analog to the ANOVA)

	Q_b	ES	95% CI	Q_w	*k*
Veteran Status	16.01***				
Civilian		.51	.41 - .61	25.99†	17
Military Veteran		.59	.49 - .69	13.20	12
Age at Time of Trauma	8.10**				
Child/ Adolescent		.48	.38 - .48	33.00***	11
Adult		.44	.34 - .54	14.05	16
Mixed		-.07	-.17 - .03	.04	2
Analysis excluding mixed studies (ES = .51, Q_{Total} = 47.55) yields no group difference					
Type of Trauma	10.63*				
Combat		.63	.53 - .73	13.13	11
Civilian War Trauma		.63	.53 - .73	.47	4
Child Maltreatment		.53	.43 - .63	5.67	6
Mixed/ Other		.28	.18 - .38	25.30***	8
Analysis excluding mixed studies and combining combat/ war exposure (ES = .56, Q_{Total} = 19.52) yields no group difference					
Traumatic Exposure	5.33*				
Excluded in Comparison		.63	.52 - .74	10.24	8
Not Excluded		.39	.28 - .50	39.62**	21

Psychiatric Disorders	13.44**				
Excluded in Comparison		.69	.58 - .80	12.39	10
Not Excluded		.32	.21 - .43	29.37*	19
Working Memory Subtests	16.89***				
Not included/ Unclear		.28	.18 - .38	30.30*	18
Included		.70	.60 - .80	8.07	11

Note: †$p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$; ES = Weight Mean Effect Size, Q_b = between groups variability, Q_w = within groups variability, Q_{Total} = total variability, CI = confidence interval, k = number of studies in each group

Table 4

Results of Continuous Moderator Analyses (Weighted Least Squares Regression)

	R^2	*b*	SE^a	$z_{1.96}$	*k*
Percent Male in Sample	.279	.006	.002	4.153***	29
Percent Male (0 and 100, excluded)	.627	.004	1.177	5.283***	14
Percent White in Sample	.105	-.002	.003	-.919	7
Mean Years of Education	.045	.075	.084	1.476	19
Mean Age at Time of Study	.037	.004	.004	1.509	29
Mean Hollingshead (1975) score	.180	.032	.036	.862	6
Mean CES score	.061	.023	.052	1.509	5
Mean CAPS score	.035	.009	.024	.347	6
Number of IQ Subtests Administered	.161	.067	.034	2.616**	22

Note: **$p < .01$, ***$p < .001$; [a]adjusted based on Hedges and Olkin (1985) recommendations; *b* = unstandardized regression coefficient, *k* = number of studies included in the analysis; CES = Combat Experiences Scale (Keane et al., 1989); CAPS = Clinician-Administered PTSD Scale for the DSM-IV (Blake et al., 1995)

Table 5

Participant Characteristics, including Group Differences for PTSD

Mother Demographics	PTSD (N = 20)	No PTSD (N = 25)	Total (N = 48)
Age—*M* (*SD*)	33.35 (5.53)	32.76 (6.51)	32.94 (5.87)
Highest Grade Completed—*N* (%)			
Less than 12th Grade	5 (25.00)	6 (24.00)	12 (24.50)
12th or High School Graduate	6 (30.00)	8 (32.00)	16 (32.70)
Some College or Technical School	7 (35.00)	8 (32.00)	15 (30.60)
Technical School Graduate	0 (0)	2 (8.00)	2 (4.10)
College Graduate	2 (10.00)	1 (4.00)	3 (6.10)
Household Monthly Income—*N* (%)			
$0 – 249	3 (15.00)	5 (20.00)	9 (18.40)
$250 – 499	2 (10.00)	1 (4.00)	3 (6.10)
$500 – 999	10 (50.00)	7 (28.00)	18 (36.70)
$1000 – 1999	10 (50.00)	6 (24.00)	17 (34.70)
$2000 or more	0 (0)	1 (4.00)	1 (2.00)
Child Demographics	**PTSD (N = 11)**	**No PTSD (N = 42)**	**Total (N = 55)**
Sex—*N* (%) Female	7 (63.64)	22 (52.38)	29 (52.73)
Age—*M* (*SD*)	9.82 (1.25)	9.95 (1.38)	9.85 (1.37)

Note: No demographic differences were observed for mothers or children with or without PTSD.

Table 6

Self-Report of Lifetime Traumatic Events in Mothers (N = 47)

Traumatic Events	*N* (%)
Natural Disaster	16 (34.04)
Serious Accident or Injury	25 (53.19)
Sudden Life-Threatening Illness	15 (31.91)*
Military Combat	0 (0)
Witnessed Close Friend or Family Member Murdered	9 (19.15)*
Attacked with Weapon by partner/spouse	14 (29.79)*
Attacked with Weapon by other than partner/spouse	7 (14.89)
Attacked Without Weapon by partner/spouse	28 (59.57)
Attacked Without Weapon by other than partner/spouse (*N* = 46)	9 (19.57)
Witnessed Violence Between Parents or Caregivers (*N* = 46)	20 (42.55)
Childhood Physical Abuse	13 (25.5)†
Childhood Emotional Abuse	22 (46.81)
Sexual Contact Before Age 13	19 (40.43)†
Forced Sexual Contact between Age 14 and 17 (*N* = 46)	11 (21.6)*
Forced Sexual Contact After Age 17 (TEI; *N* = 45)	12 (25.53)*
TEI Total Types of Trauma—*M* (*SD*)	5.69 (3.34)***
TEI Total Types of Trauma, Excluding Child Abuse—*M* (*SD*)	4.78 (2.67)***
TEI Total Types of Child Abuse (*N* = 44)—*M* (*SD*)	1.09 (1.15) †

Note: †$p < .10$, *$p < .05$, ***$p < .001$, indicating that mothers with PTSD more likely to report noted trauma; TEI = *Traumatic Events Inventory* (Gillespie et al., 2009)

Table 7

Pearson Correlations among Mother Mental Health and Trauma Exposure (N = 47)

	PSS_T	PSS_R	PSS_A	PSS_H	BDI	AUD	DAST	TEI_{Tot}	TEI_{ChAb}	TEI_{NoChAb}	TEI_{age1st}	Age	Edu	Income
PSS_T	-													
PSS_R	.91***	-												
PSS_A	.93***	.81***	-											
PSS_H	.89***	.71***	.72***	-										
BDI	.66***	.52***	.58***	.70***	-									
AUD	.25	.14	.23	.30†	.39*	-								
DAST	.42**	.28†	.35*	.49**	.49**	.22	-							
TEI_{Tot}	.67***	.60***	.55***	.68***	.62***	.16	.60***	-						
TEI_{ChAb}	.55***	.54***	.49***	.51***	.48**	.24	.42**	.65***	-					
TEI_{NoChAb}	.60***	.54***	.47**	.63***	.53***	.14	.56***	.95***	.46**	-				
TEI_{age1st}	-.51***	-.42**	-.49***	-.48***	-.31*	-.03	-.24	-.60***	-.56***	-.52***	-			
Age	.22	.23	.33*	.04	.19	.37*	.11	.10	.32*	.01	-.04	-		

Edu	.01	-.03	.09	-.05	-.11	-.26	.04	-.07	.10	-.17	.09	.22	-	
Income	-.22	-.24	-.20	-.18	-.37*	-.01	-.03	-.24	-.13	-.22	.19	.23	.33*	-

Note: †$p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$; PSS = PTSD Symptom Scale ($_{T}$ = total score, $_{R}$ = re-experiencing score, $_{A}$ = avoidance score, $_{H}$ = hyperarousal score; Foa & Tolin, 2000; Schwartz et al., 2005); BDI = *Beck Depression Inventory-II* (Beck et al., 1996); AUD = *Alcohol Use Disorders Identification* Test (Saunders et al., 1993); DAST = *Drug Abuse Screening Test* (Skinner, 1982); TEI = *Traumatic Events Inventory* ($_{Tot}$ = total types of lifetime trauma exposure, $_{ChAb}$ = total types of child abuse experienced; $_{NoChAb}$ = total types of lifetime trauma exposure, excluding child abuse; $_{age1st}$ = first age of any type of trauma; Gillespie et al., 2009); Edu = years of education

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Table 8

Sample Means for Mother Mental Health, Parenting, and Intelligence and Memory (N = 45)

Mental Health and Parenting	PTSD (N = 20)[a]	No PTSD (N = 25)	Total
PSS Total Score	29.20 (10.69)	8.26 (9.41)***	17.57 (14.44)
PSS Avoidance/ Numbing	11.60 (4.66)	3.64 (4.82)***	7.18 (6.17)
PSS Re-experiencing	6.95 (4.05)	2.40 (3.66)***	4.42 (4.43)
PSS Hyperarousal	10.65 (2.20)	2.20 (2.63)***	5.96 (5.26)
BDI-II (*N* = 20, 22)	27.44 (11.40)	11.40 (8.88)***	19.03 (14.32)
AUDIT-C (*N* = 17, 22)	2.78 (1.62)	1.62 (1.57)	2.17 (2.32)
DAST-10 (*N* = 19, 20)	1.63 (2.11)	.20 (.70)*	.79 (1.42)
Intelligence (RIAS)			
CIX Index Score (*N* = 21, 20)	86.22 (8.00)	89.19 (7.95)	87.98 (8.08)
VIX Index Score (*N* = 18, 22)	83.78 (8.36)	84.27 (11.08)	84.11 (9.83)
GWH T-Score (*N* = 20, 24)	40.40 (5.53)	39.21 (5.48)	39.81 (5.41)
VRZ T-Score (*N* = 18, 23)	34.72 (5.96)	39.39 (9.30)†	37.29 (8.38)
NIX Index Score (*N* = 19, 22)	92.95 (9.33)	95.41 (7.22)	94.62 (8.27)
OIO T-Score	47.90 (8.83)	49.20 (4.97)	48.90 (7.13)
WHM T-Score (*N* = 19, 22)	41.74 (6.75)	42.68 (6.27)	42.42 (6.17)
CMX Index Score	96.60 (8.76)	95.64 (11.70)	95.84 (11.26)
VRM T-Score	43.70 (8.09)	47.08 (8.70)	45.76 (9.74)

NVM T-Score	51.30 (5.41)	46.88 (7.05)*	48.33 (6.91)

Note: [a]Analyses with fewer cases due to missing or incomplete data indicated in first column; †p < .10, *p < .05, ***p < .001; PSS = PTSD Symptom Scale (Foa & Tolin, 2000; Schwartz et al., 2005); BDI = *Beck Depression Inventory-II* (Beck et al., 1996); AUDIT-C = *Alcohol Use Disorders Identification* Test (Saunders et al., 1993); DAST = *Drug Abuse Screening Test* (Skinner, 1982); RIAS = Reynold's Intellectual Assessment Scales (Reynolds & Kamphaus, 2003); CIX = Composite Intelligence Index, VIX = Verbal Intelligence Index, GWH = Guess What? T-score, VRZ = Verbal Reasoning T-score, NIX = Nonverbal Intelligence Index, OIO = Odd-Item-Out T-score, WHM = What's Missing? T-score; CMX = Composite Memory Index, VRM = Verbal Memory T-score, NVM = Nonverbal Memory T-sc

Table 9

Pearson Correlations among RIAS Indices and Subtests for Mothers (N = 47)

	CIX	VIX	GWH	VRZ	NIX	OIO	WHM	CMX	VRM	NVM
CIX	-									
VIX	.90***	-								
GWH	.75***	.74***	-							
VRZ	.72***	.76***	.49**	-						
NIX	.83***	.51**	.55***	.42**	-					
OIO	.78***	.56****	.46**	.55***	.79***	-				
WHM	.50**	.24	.39**	.15	.71***	.16	-			
CMX	.33*	.38*	.37*	.38*	.27†	.21	.19	-		
VRM	.40**	.40**	.31*	.41**	.35*	.23	.29†	.84***	-	
NVM	.01	.14	.22	.12	.03	.07	-.09	.64***	.14	-

Note: †$p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$; RIAS = *Reynolds Intellectual Assessment Scales* (Reynolds & Kamphaus, 2003); CIX = Composite Intelligence Index; VIX = Verbal Intelligence Index; GWH = Guess What? T-Score; VRZ = Verbal Reasoning T-Score; NIX =

Nonverbal Intelligence Index; OIO = Odd-Item-Out T-Score; WHM = What's Missing? T-Score; CMX = Composite Memory Index; VRM = Verbal Memory T-Score; NVM = Nonverbal Memory T-Score

Table 10

Pearson Correlations between Mother Intelligence and Memory and Mental Health and Trauma Exposure (N = 47)

	CIX	VIX	GWH	VRZ	NIX	OIO	WHM	CMX	VRM	NVM
PSS_T	.07	.21	.17	-.02	.01	-.02	.05	.18	.05	.26†
PSS_R	.16	.28†	.19	.09	.09	.04	.12	.13	.08	.14
PSS_A	.04	.21	.16	-.01	-.08	-.10	-.003	.28†	.15	.30*
PSS_H	.002	.11	.13	-.11	.04	.04	.03	.05	-.12	.24
BDI	.13	.03	.02	-.10	.11	.15	-.01	-.17	-.19	-.05
AUD	-.29†	-.25	-.16	-.27	-.22	-.29†	-.02	.04	-.14	.24
DAST	-.19	-.20	-.14	-.28	-.03	-.15	.09	-.12	-.17	.02
TEI_{Tot}	.23	.25	.22	.04	.28	.26	.17	.08	.01	.13
TEI_{ChAb}	.25	.36*	.13	.33*	.10	.18	-.04	.20	.11	.19
TEI_{NoChAb}	.18	.17	.22	-.05	.29†	.27†	.19	-.01	-.06	.07
TEI_{age1st}	-.17	-.25	-.14	-.17	-.11	-.12	-.07	-.21	-.04	-.32*
Age	-.06	.02	-.02	.15	.01	<.001	-.05	.32*	.25†	.22
Edu	.15	.26†	.12	.42**	.06	.08	-.02	.35*	.29*	.21
Income	-.06	.14	-.12	.11	-.06	-.05	.001	.22	.32*	-.03

Note: †$p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$); RIAS = *Reynolds Intellectual Assessment Scales* (Reynolds & Kamphaus, 2003); PSS = PTSD Symptom Scale ($_{Tot}$ = total score, $_R$ = re-experiencing symptom score, $_A$ = avoidance symptom scores, $_H$ = hyperarousal symptom score; Foa & Tolin, 2000; Schwartz et al., 2005); BDI = *Beck Depression Inventory-II* (Beck et al., 1996); AUD = *Alcohol Use Disorders Identification Test* (Saunders et al., 1993); DAST = *Drug Abuse Screening Test* (Skinner, 1982); TEI = *Traumatic Events Inventory* ($_T$ = total types of

lifetime trauma exposure, $_{ChAb}$ = total types of child abuse experienced; $_{NoChAb}$ = total types of lifetime trauma exposure, excluding child abuse; Gillespie et al., 2009); CIX = Composite Intelligence Index; VIX = Verbal Intelligence Index; GWH = Guess What? T-Score; VRZ = Verbal Reasoning T-Score; NIX = Nonverbal Intelligence Index; OIO = Odd-Item-Out T-Score; WHM = What's Missing? T-Score; CMX = Composite Memory Index; VRM = Verbal Memory T-Score; NVM = Nonverbal Memory T-Score

Table 11

Binary Logistic Regression Predicting PTSD from Trauma Exposure

	B	*SE*	*eB*	*eB* 95% CI	*p*
Model—χ^2 (2) = 10.46, R^2 = .28, p = .005					
Total Types of Child Abuse	.13	.32	1.14	1.08 – 2.13	.68
Total Types of Non-child abuse Trauma	.42	.17	1.52	.61 – 2.15	.02

Note: PTSD based on the PTSD Symptom Scale (Foa & Tolin, 2000; Schwartz et al., 2005); TEI = *Traumatic Events Inventory* (Gillespie et al., 2009)

Table 12

Binary Logistic Regression with Verbal Reasoning and Nonverbal Reasoning Predicting PTSD in Mothers above Trauma Exposure, Depression, and Alcohol and Substance Use

	B	*SE*	*eB*	*eB* 95% CI	*p*
Model 1—χ^2 (1) = 8.76, R^2 = .30, p = .003					
Total Types of Trauma, Lifetime (TEI)	.36	.15	1.44	1.06 – 1.95	.02
Model 2—χ^2 (4) =11.42, R^2 = .38, p = .02					
Total Types of Trauma, Lifetime (TEI)	.11	.21	1.11	.73 – 1.69	.61
Depression (BDI-II)	.09	.06	1.09	.98 – 1.21	.12
Alcohol Use (AUDIT-C)	-.04	.23	.96	.61 – 1.51	.86
Drug Use (DAST-10)	.71	.55	1.66	.69 – 5.97	.20
Model 3—χ^2 (6) = 30.22, R^2 = .79, p < .001					
TEI Total Types of Trauma, Lifetime	1.44	.89	4.22	.74 – 24.19	.11
Depression (BDI-II)	.39	.26	1.48	.89 – 2.46	.13
Alcohol Abuse (AUDIT-C)	-.91	.66	.40	.11 – 1.46	.17
Drug Abuse (DAST-10)	-.02	.95	.98	.15 – 6.25	.98
Verbal Reasoning (RIAS)	-.77	.41	.47	.21 – 1.04	.061
Nonverbal Memory (RIAS)	.84	.44	2.32	.98 – 5.50	.057

Note: PTSD based on the PTSD Symptom Scale (Foa & Tolin, 2000; Schwartz et al., 2005); TEI = *Traumatic Events Inventory* (Gillespie et al., 2009); BDI –II = *Beck Depression Inventory-II* (Beck et al., 1996); AUDIT-C = *Alcohol Use Disorders Identification Test* (Saunders et al., 1993); DAST-10 = *Drug Abuse Screening Test* (Skinner, 1982); RIAS = *Reynolds Intellectual Assessment Scales* (Reynolds & Kamphaus, 2003)

Table 13

Self-Report of Lifetime Traumatic Events in Children, including Group Differences for Children with and without PTSD (N = 53)

Traumatic Events—Child Self-Report (VEX-R)	PTSD (*N* = 11)	No PTSD (*N* = 42)	Total
Witnessing Violence—Total Types—*M* (*SD*)	7.54 (3.27)	7.55 (3.27)†	6.16 (2.93)
Someone Yelled At—*N* (%)	10 (90.91)	37 (88.10)	48 (87.27)
Someone Thrown At	8 (72.73)	26 (61.90)	34 (61.82)
Someone Pushed	8 (72.73)	27 (64.29)	35 (63.64)
Someone Chased in Anger	8 (72.73)	15 (35.71)*	24 (43.64)
Someone Slapped	8 (72.73)	17 (40.48)†	26 (47.27)
Someone Beaten Up	9 (81.82)	30 (71.43)	40 (72.73)
Someone Mugged	5 (45.45)	14 (33.33)	20 (36.36)
Someone Directly Threatened with Weapon	3 (27.27)	2 (4.76)*	6 (10.91)
A Child Spanked	7 (63.64)	34 (80.95)	42 (76.36)
Someone Stabbed	2 (18.18)	2 (2.76)	4 (7.27)
Someone Shot	2 (18.18)	1 (2.38)*	3 (5.45)

Someone Arrested	9 (81.82)	36 (85.71)	46 (83.64)
Someone Dealing Drugs	4 (36.36)	6 (14.29)†	11 (20.00)
Experiencing Violence—Total Types—*M* (*SD*)	4.45 (2.54)	3.11 (2.04)†	3.41 (2.29)
Yell At—*N* (%)	9 (81.82)	35 (83.33)	45 (81.82)
Thrown At	6 (54.55)	12 (28.57)	18 (32.73)
Pushed	6 (54.55)	14 (33.33)	21 (38.18)
Chased in Anger	5 (45.45)	11 (26.19)	17 (30.91)
Slapped	6 (54.55)	6 (14.29)**	13 (23.64)
Beaten Up	3 (27.27)	6 (14.29)	10 (18.18)
Mugged	6 (54.55)	13 (30.95)	20 (36.36)
Directly Threatened with Weapon	2 (18.18)	1 (2.38)	3 (5.45)
Spanked	8 (72.73)	32 (76.19)	41 (74.5)
Total Types of Violence Exposure—*M* (*SD*)	12.00 (5.53)	9.00 (4.37)	9.58 (4.94)

Note: †$p < .10$, *$p < .05$, **$p < .01$; VEX-R = *Violence Exposure Scale for Children—Revised* (VEX-R; Fox & Leavitt, 1995)

Table 14

Self-Report of Lifetime Traumatic Events in Children, including Group Differences for Boys and Girls (N = 55)

Traumatic Events—Child Self-Report (VEX-R)	Boys (*N* = 26)	Girls (*N* = 29)
Witnessing Violence—Total Types—*M* (*SD*)	6.69 (3.13)	5.69 (2.70)
Someone Yelled At—*N* (%)	24 (92.31)	24 (82.76)
Someone Thrown At	16 (61.54)	18 (62.07)
Someone Pushed	18 (69.23)	17 (58.62)
Someone Chased in Anger	12 (46.15)	12 (41.38)
Someone Slapped	14 (53.85)	12 (41.38)
Someone Beaten Up	21 (92.31)	19 (65.52)
Someone Mugged	13 (50.00)	7 (24.14)*
Someone Directly Threatened with Weapon	4 (15.38)	2 (6.90)
A Child Spanked	19 (73.08)	23 (79.31)
Someone Stabbed	4 (15.38)	0 (0)*
Someone Shot	2 (7.69)	1 (3.45)
Someone Arrested	22 (84.62)	24 (82.76)
Someone Dealing Drugs	5 (19.23)	6 (20.69)
Experiencing Violence—Total Types—*M* (*SD*)	4.12 (2.47)	2.79 (1.95)*
Yell At—*N* (%)	21 (92.31)	24 (82.76)
Thrown At	10 (38.46)	10 (34.48)
Pushed	14 (53.85)	7 (24.14)*
Chased in Anger	11 (42.31)	6 (20.69)†

Slapped	9 (34.62)	4 (13.79)†
Beaten Up	8 (30.77)	2 (6.90)*
Mugged	10 (38.46)	10 (34.48)
Directly Threatened with Weapon	3 (11.54)	0 (0)†
Spanked	5 (19.23)	9 (31.03)
Total Types of Violence Exposure—*M* (*SD*)	10.81 (5.24)	8.48 (4.45)†

Note: †$p < .10$, *$p < .05$; VEX-R = *Violence Exposure Scale for Children—Revised* (VEX-R; Fox & Leavitt, 1995)

Table 15

Sample Means for Child Mental Health and Intelligence, including Group Differences for Boys and Girls (N = 55)

Child Mental Health—*M* (*SD*)	Boys (*N* = 26)	Girls (*N* = 29)
PTSD Symptom Total, Child-Report (UCLA)	23.50 (14.02)	24.51 (14.85)
Re-experiencing (5 items)	5.29 (4.21)	7.10 (5.73)
Avoidance (8 items)	7.83 (5.38)	7.18 (5.73)
Hyperarousal (5 items)	8.50 (5.06)	8.69 (4.50)
Self-Blame (1 item)	.79 (1.14)	.69 (.93)
Fear of Repeated Trauma (1 item)	1.21 (1.28)	.79 (1.15)
Anxiety, Child-Report (BASC)	50.24 (9.63)	47.76 (8.20)
Depression, Child-Report (BASC)	47.20 (5.21)	46.10 (4.47)
School Problems, Child-Report (BASC)	48.82 (8.05)	46.61 (6.48)
Attention Problems, Child-Report (BASC)	50.00 (9.35)	51.79 (9.87)
Hyperactivity, Child-Report (BASC)	49.56 (10.10)	49.48 (9.35)
Mother-reported Birth Weight (in ounces)	117.85 (21.77)	109.82 (15.02)
Child Intelligence (RIAS)		
Composite Intelligence Index	91.19 (8.85)	93.33 (9.88)
Verbal Intelligence Index	88.96 (8.46)	90.07 (11.84)
Guess What T-Score	41.27 (7.62)	40.48 (9.75)
Verbal Reasoning T-Score	41.27 (6.87)	44.36 (8.61)
Nonverbal Intelligence Index	95.19 (13.74)	99.28 (10.44)
Odd-Item-Out T-Score	50.81 (6.95)	52.52 (7.55)

What's Missing? T-Score	43.88 (12.46)	45.21 (9.48)
Composite Memory Index	91.80 (9.10)	100.17 (7.02)***
Verbal Memory T-Score	39.65 (8.42)	49.60 (8.65)*
Nonverbal Memory T-Score	49.60 (8.65)	53.45 (5.14)*

Note: *$p < .05$, ***$p < .001$ for group differences between girls and boys; UCLA = *UCLA PTSD index for DSM-IV, Child Version* (Rodriguez et al., 1999); BASC-2 = *Behavioral Assessment System for Children—Second Edition, Self-Report* (Reynolds & Kamphaus, 2004); RIAS = *Reynolds Intellectual Assessment Scales* (Reynolds & Kamphaus, 2003)

Table 16

Sample Means for Child Mental Health and Intelligence, including Group Differences for Children with and without PTSD (N = 53)

Child Mental Health—*M* (*SD*)	PTSD (*N* = 11)	No PTSD (*N* = 42)	Total
PTSD Symptom Total, Child-Report (UCLA)	44.09 (4.48)	18.80 (10.97)***	24.05 (14.35)
Re-experiencing (5 items)	12.82 (3.79)	4.58 (3.93)***	6.28 (5.13)
Avoidance (8 items)	14.55 (3.91)	5.63 (4.26)***	7.48 (5.53)
Hyperarousal (5 items)	13.37 (3.29)	7.36 (4.24)***	8.60 (4.72)
Self-Blame (1 item)	1.27 (1.42)	.60 (.86)	.74 (1.02)
Fear of Repeated Trauma (1 item)	2.09 (1.45)	.69 (.98)***	.98 (1.23)
Anxiety T-Score (BASC-2)	54.36 (10.98)	47.74 (7.78)*	48.91 (8.89)
Depression T-Score (BASC-2)	48.36 (46.17)	46.17 (4.64)	46.61 (4.81)
School Problems T-Score (BASC-2)	48.20 (7.32)	46.74 (5.71)	47.69 (7.29)
Attention Problems T-Score (BASC-2)	52.27 (8.39)	50.17 (9.57)	50.96 (9.59)
Hyperactivity T-Score (BASC-2)	49.64 (5.94)	49.05 (10.11)	49.52 (9.60)

Mother-reported Birth Weight (in ounces)	118.00 (21.17)	112.23 (18.36)	113.54 (18.65)
Child Intelligence (RIAS)			
Composite Intelligence Index	88.20 (8.82)	93.39 (9.53)	92.28 (9.36)
Verbal Intelligence Index	85.70 (9.87)	90.39 (10.48)	89.53 (10.23)
Guess What T-Score	38.20 (6.76)	41.61 (9.24)	40.87 (8.70)
Verbal Reasoning T-Score	41.63 (8.99)	43.10 (7.82)	42.87 (7.90)
Nonverbal Intelligence Index	94.55 (13.50)	98.36 (12.06)	97.35 (12.17)
Odd-Item-Out T-Score	51.82 (9.95)	51.57 (6.70)	51.71 (7.26)
What's Missing? T-Score	40.09 (46.21)	43.05 (10.11)	44.58 (10.90)
Composite Memory Index	98.09 (8.15)	96.15 (9.08)	96.30 (9.01)
Verbal Memory T-Score	44.45 (8.43)	42.40 (8.58)	42.71 (8.50)
Nonverbal Memory T-Score	52.09 (4.97)	51.78 (7.76)	51.67 (7.18)

Note: *$p < .05$, ***$p < .001$ for group differences between girls and boys; UCLA = *UCLA PTSD index for DSM-IV, Child Version* (Rodriguez, et al., 1999); BASC-2 = *Behavioral Assessment System for Children—Second Edition, Self-Report* (Reynolds & Kamphaus, 2004); RIAS = *Reynolds Intellectual Assessment Scales* (Reynolds & Kamphaus, 2003)

Table 17

Pearson Correlations among Child Mental Health and Trauma Exposure (N = 55)

	$UCLA_T$	$UCLA_I$	$UCLA_A$	$UCLA_H$	Anx	Dep	SchPrb	AttPrb	Hyp	VEX_{Exp}	VEX_{Wit}	VEX_{Tot}	BirthWeight
$UCLA_T$	-												
$UCLA_I$	.88***	-											
$UCLA_A$	.86***	.62***	-										
$UCLA_H$	.83***	.66***	.57***	-									
Anx	.58***	.49***	.52***	.51***	-								
Dep	.32*	.22	.28*	.34*	.44**	-							
SchPrb	.12	.02	.14	.14	.16	.18	-						
AttPrb	.37**	.31*	.27†	.42**	.41**	.39**	.38*	-					
Hyp	.29*	.26	.19	.39**	.38**	.23†	.44**	.47***	-				
VEX_{Exp}	.28*	.22	.31*	.24	.26†	.16	-.14	.03	.18	-			
VEX_{Wit}	.30*	.30*	.32*	.22	.27*	.11	-.27†	.01	.21	.78***	-		
VEX_{Tot}	.32*	.28*	.33*	.25†	.28*	.14	-.23	.02	.21	.93***	.96***	-	

BirthWeight	.14	.18	.12	.05	.22	.22	-.01	.13	.17	.19	.37*	.31†	-

Note: †$p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$; UCLA = *UCLA PTSD index for DSM-IV, Child Version* ($_{T}$ = Total, $_{I}$ = intrusive, $_{A}$ = avoidance, and $_{H}$ = hyperarousal symptom scores; Rodriguez et al., 1999); Anx = Anxiety, Dep = Depression, SchPrb = School Problems, AttProb = Attention Problems, Hyp = Hyperactivity (*Behavioral Assessment System for Children—Second Edition, Self-Report* Reynolds & Kamphaus, 2004); VEX = *Violence Exposure Scale for Children—Revised* ($_{Exp}$ = total types of trauma experienced, $_{Wit}$ = total types of trauma witnessed, $_{Tot}$ = total types of trauma experienced and witnessed; Fox & Leavitt, 1995); Birth Weight = mother-reported weight of child at birth in ounces

Table 18

Pearson Correlations among RIAS Indices and Subtests for Children (N = 53)

	CIX	VIX	GWH	VRZ	NIX	OIO	WHM	CMX	VRM	NVM
CIX	-									
VIX	.79***	-								
GWH	.70***	.87***	-							
VRZ	.66***	.85***	.47***	-						
NIX	.62***	.14	.18	.06	-					
OIO	.61***	.28*	.29*	.26†	.55***	-				
WHM	.65***	.15	.14	.09	.74***	.17	-			
CMX	.37**	.33*	.34*	.24†	.19	.16	.21	-		
VRM	.33*	.31*	.38**	.18	.18	.11	.19	.73***	-	
NVM	.10	.10	.06	.10	-.002	-.11	.14	.47***	-.16	-

Note: †$p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$; RIAS = *Reynolds Intellectual Assessment Scales* (Reynolds & Kamphaus, 2003); CIX = Composite Intelligence Index; VIX = Verbal Intelligence Index; GWH = Guess What? T-Score; VRZ = Verbal Reasoning T-Score; NIX =

Nonverbal Intelligence Index; OIO = Odd-Item-Out T-Score; WHM = What's Missing? T-Score; CMX = Composite Memory Index; VRM = Verbal Memory T-Score; NVM = Nonverbal Memory T-Score

Table 19

Pearson Correlations between Child Mental Health and Trauma Exposure and Intelligence (N = 55)

	CIX	VIX	GWH	VRZ	NIX	OIO	WHM	CMX	VRM	NVM
$UCLA_T$	-.28*	-.35*	-.28*	-.28*	-.01	.04	-.14	-.08	-.14	.06
$UCLA_I$	-.24†	-.29*	-.21	-.24†	-.04	-.04	-.11	-.05	-.02	.12
$UCLA_A$	-.28*	-.31*	-.28†	-.18	-.03	.08	-.21	-.16	-.23	.03
$UCLA_H$	-.16	-.28*	-.22	-.25	-.06	.09	-.01	-.01	-.09	.03
Anx	-.13	-.12	-.12	-.07	-.03	.10	-.17	-.18	-.09	-.15
Dep	-.12	-.19	-.20	-.14	-.11	.10	-.08	-.12	-.22	.04
SchPrb	.04	.07	.05	.03	-.01	-.02	.03	.07	.10	-.05
AttPrb	-.05	-.20	-.14	-.23	.17	.21	.02	-.04	-.01	-.12
Hyp	-.06	-.05	.01	-.09	.08	.12	-.12	-.16	-.13	-.16
VEX_{Exp}	-.21	-.16	-.14	-.16	-.14	-.20	-.09	-.15	-.20	.002
VEX_{Wit}	-.24†	-.21	-.13	-.25†	-.07	-.17	-.10	-.16	-.23	.03
VEX_{Tot}	-.24†	-.20	-.14	-.22	-.11	-.19	-.10	-.16	-.23†	.02

BirthWeight	-.05	-.02	-.04	-.04	-.03	-.16	.05	-.30†	-.46**	.08

Note: †$p < .10$, *$p < .05$, **$p < .01$; UCLA = *UCLA PTSD index for DSM-IV, Child Version* ($_{T}$ = Total, $_{I}$ = intrusive, $_{A}$ = avoidance, and $_{H}$ = hyperarousal symptom scores; Rodriguez et al., 1999); Anx = Anxiety, Dep = Depression, SchPrb = School Problems, AttProb = Attention Problems, Hyp = Hyperactivity (*Behavioral Assessment System for Children—Second Edition, Self-Report* Reynolds & Kamphaus, 2004); VEX = *Violence Exposure Scale for Children—Revised* ($_{Exp}$ = total types of trauma experienced, $_{Wit}$ = total types of trauma witnessed, $_{Tot}$ = total types of trauma experienced and witnessed; Fox & Leavitt, 1995); Birth Weight = mother-reported weight of child at birth in ounces; RIAS = *Reynolds Intellectual Assessment Scales* (Reynolds & Kamphaus, 2003); CIX = Composite Intelligence Index; VIX = Verbal Intelligence Index; GWH = Guess What? T-Score; VRZ = Verbal Reasoning T-Score; NIX = Nonverbal Intelligence Index; OIO = Odd-Item-Out T-Score; WHM = What's Missing? T-Score; CMX = Composite Memory Index; VRM = Verbal Memory T-Score; NVM = Nonverbal Memory T-Score

Table 20

Pearson Correlations between Child Mental Health and Trauma Exposure and Intelligence, Separately for Boys (Left; N = 26) and Girls (Right; N = 29)

	CIX	VIX	GWH	VRZ	NIX	OIO	WHM	CMX	VRM	NVM	CIX	VIX	GWH	VRZ	NIX	OIO	WHM	CMX	VRM	NVM
$UCLA_T$	-.16	-.36†	-.46*	-.15	.22	.17	.02	-.17	-.39†	.19	-.38*	-.35†	-.16	-.39*	-.28	-.07	-.31	-.06	.03	-.13
$UCLA_I$	-.15	-.28	-.36†	-.13	.14	.05	.01	-.10	-.37†	.30	-.34†	-.31	-.13	-.38*	-.25	-.13	-.23	.02	.10	-.12
$UCLA_A$	-.20	-.36†	-.43*	-.20	.22	.20	-.06	-.24	-.46*	.18	-.33†	-.27	-.18	-.16	-.27	.02	-.37*	-.09	-.02	-.12
$UCLA_H$	.05	-.28	-.39†	-.08	.31	.36†	.18	-.01	-.22	.14	-.35†	-.30	-.09	-.41*	-.27	-.14	-.25	-.10	.02	-.17
Anx	-.09	-.14	-.31	.10	.09	.02	-.02	-.19	-.15	-.08	-.14	-.09	.02	-.17	-.13	.22	-.36†	-.07	.06	-.18
Dep	-.03	-.14	-.24	.04	-.11	.38†	-.14	-.07	-.19	.05	-.20	-.22	-.18	-.27	-.07	-.13	.01	-.14	-.20	.08
SchPrb	-.01	-.002	-.14	.15	-.004	.07	-.03	-.16	-.18	-.01	.09	.13	.24	-.07	.03	-.08	.12	.50*	.50*	-.04
AttPrb	-.003	-.09	-.11	-.05	.15	.16	.003	-.01	.03	-.20	-.11	-.29	-.15	-.41*	.17	.24	.03	-.22	-.12	-.14
Hyp	-.12	-.16	-.17	-.11	.18	.15	-.12	-.28	-.16	-.29	-.002	.03	.16	-.09	-.03	.10	-.11	-.14	-.11	-.06
VEX_{Exp}	.03	-.07	-.01	-.11	.14	.09	.05	-.10	-.18	.05	-.42*	-.23	-.30	-.11	-.46*	-.45*	-.26	.07	-.03	.15
VEX_{Wit}	-.20	-.29	-.12	-.39†	.11	-.03	-.06	-.16	-.23	.02	-.25	-.14	-.16	-.09	-.26	-.27	-.14	.001	-.12	.18

VEX_{Tot}	-.11	-.21	-.07	-.28	.13	.02	-.01	-.14	-.23	.03	-.34	-.19	-.23	-.10	-.36†	-.36†	-.20	.03	-.09	.17
BW	-.12	-.09	-.10	-.06	-.11	-.20	-.04	-.39	-.55*	.02	.10	.07	-.03	.06	.18	-.10	.31	.06	-.22	.37†

Note: †$p < .10$, *$p < .05$; UCLA = *UCLA PTSD index for DSM-IV, Child Version* ($_{T}$ = Total, $_{I}$ = intrusive, $_{A}$ = avoidance, and $_{H}$ = hyperarousal symptom scores; Rodriguez et al., 1999); Anx = Anxiety, Dep = Depression, SchPrb = School Problems, AttProb = Attention Problems, Hyp = Hyperactivity (*Behavioral Assessment System for Children—Second Edition, Self-Report* Reynolds & Kamphaus, 2004); VEX = *Violence Exposure Scale for Children—Revised* ($_{Exp}$ = total types of trauma experienced, $_{Wit}$ = total types of trauma witnessed, $_{Tot}$ = total types of trauma experienced and witnessed; Fox & Leavitt, 1995); BW = mother-reported weight of child at birth in ounces; RIAS = *Reynolds Intellectual Assessment Scales* (Reynolds & Kamphaus, 2003); CIX = Composite Intelligence Index; VIX = Verbal Intelligence Index; GWH = Guess What T-Score; VRZ = Verbal Reasoning T-Score; NIX = Nonverbal Intelligence Index; OIO = Odd-Item-Out T-Score; WHM = What's Missing? T-Score; CMX = Composite Memory Index; VRM = Verbal Memory T-Score; NVM = Nonverbal Memory T-Score

Table 21

Multiple Linear Regression with Anxiety and Verbal Intelligence Predicting Child PTSD Symptom Frequency (UCLA)

	B	*SE*	*t*	*p*	*VIF*
Model—F (7, 43) = 5.25, R^2 = .46, p < .001					
Child Age	2.01	1.60	1.25	.22	1.78
Child Total Types of Trauma (VEX-R)	.12	.40	.29	.77	1.42
Child Anxiety T-Score (BASC-2)	.69	.22	3.15	.003	1.53
Child Depression T-Score (BASC-2)	.35	.43	.82	.42	1.66
Child Attention Problems T-Score (BASC-2)	.30	.22	1.35	.18	1.62
Child Hyperactivity T-Score (BASC-2)	-.09	.22	-.40	.69	1.68
Child Verbal Intelligence Index (RIAS)	-.34	.16	-2.08	.04	1.12

Note: UCLA = *UCLA PTSD index for DSM-IV, Child Version* (Rodriguez et al., 1999); VEX-R = *Violence Exposure Scale for Children—Revised* (Fox & Leavitt, 1995); BASC-2 = *Behavioral Assessment System for Children—Second Edition, Self-Report* (Reynolds & Kamphaus, 2004); RIAS = *Reynolds Intellectual Assessment Scales* (Reynolds & Kamphaus, 2003); VIF = Variance Inflation Factor

Table 22

Multiple Linear Regression Predicting Child PTSD Symptom Frequency (UCLA), Separately for Boys (Top; N = 26) and Girls (Bottom; N = 29)

		B	*SE*	*t*	*p*	*VIF*
Boys	Model—F (7, 16) = 2.88, R^2 = .56, p < .05					
	Child Age	.53	2.69	.20	.85	2.19
	Child Total Types of Trauma (VEX-R)	-.01	.59	-.02	.98	1.48
	Child Anxiety T-Score (BASC-2)	.75	.30	2.52	.02	1.52
	Child Depression T-Score (BASC-2)	.61	.64	.96	.35	2.12
	Child Attention Problems T-Score (BASC-2)	.08	.41	.19	.86	2.29
	Child Hyperactivity T-Score (BASC-2)	-.23	.34	-.68	.51	1.96
	Child Guess What? T-Score (RIAS)	-.47	.33	-1.45	.17	1.21
Girls	Model—F (7, 19) = 3.21, R^2 = .54, p < .02					
	Child Age	-.26	2.16	-.12	.91	1.68
	Child Total Types of Trauma (VEX-R)	.97	.61	1.59	.13	1.35
	Child Anxiety T-Score (BASC-2)	.45	.43	1.05	.31	2.20
	Child Depression T-Score (BASC-2)	-.33	.62	-.53	.61	1.37
	Child Attention Problems T-Score (BASC-2)	.30	.32	.96	.35	1.72
	Child Hyperactivity T-Score (BASC-2)	.26	.35	.73	.47	1.90
	Child Verbal Reasoning T-Score (RIAS)	-.43	.31	-1.38	.18	1.26

Note: UCLA = *UCLA PTSD index for DSM-IV, Child Version* (Rodriguez et al., 1999); VEX-R = *Violence Exposure Scale for Children—Revised* (Fox & Leavitt, 1995); BASC-2 = *Behavioral Assessment System for Children—Second Edition, Self-Report* (Reynolds

& Kamphaus, 2004); RIAS = *Reynolds Intellectual Assessment Scales* (Reynolds & Kamphaus, 2003); VIF = Variance Inflation Factor

Table 23

Pearson Correlations between Mother Trauma Exposure and Mental Health and Parenting (N = 47)

	Abuse Potential (CAPI)	Overreactivity (PS)	Laxness (PS)
PSS Total	.73***	.20	.31*
PSS Reexperiencing	.55***	.08	.32*
PSS Avoidance	.65***	.17	.31*
PSS Hyperarousal	.78***	.29†	.21
BDI Total	.81***	.44**	.20
AUDIT-C Total	.35*	.46**	.19
DAST-10 Total	.53***	.31†	.11
TEI Total Types Trauma	.64***	.31*	.10
TEI Total Types Child Abuse	.45**	.10	.02
TEI Total Types Trauma, no Child Abuse	.58***	.32*	.11
TEI Age at First Trauma	-.43**	-.21	.01
Age	.16	-.04	.23
Education	-.09	-.13	-.29*
Household Monthly Income	-.21	-.13	.01

Note: †$p < .10$, *$p < .05$, **$p < .01$, ***$p < .001$; PSS = PTSD Symptom Scale (Foa & Tolin, 2000; Schwartz et al., 2005); BDI = *Beck Depression Inventory-II* (Beck et al., 1996); AUDIT-C = *Alcohol Use Disorders Identification Test* (Saunders et al., 1993); DAST-10 = *Drug Abuse Screening Test* (Skinner, 1982); TEI = *Traumatic Events*

Inventory (Gillespie et al., 2009); CAPI = *Child Abuse Potential Inventory* (Milner, 1990); PS = Parenting Scale (Arnolds, 1993)

Table 24

Pearson Correlations between Mother RIAS Performance and Parenting (N = 47)

	Abuse Potential (CAPI)	Overreactivity (PS)	Laxness (PS)
Composite Intelligence Index	.08	-.06	-.01
Verbal Intelligence Index	.04	-.05	.02
Guess What T-Score	.02	-.03	-.03
Verbal Reasoning T-Score	.00	.01	-.12
Nonverbal Intelligence Index	.17	-.10	.03
Odd-Item-Out T-Score	.11	-.11	-.02
What's Missing? T-Score	.23	.05	.11
Composite Memory Index	.00	-.23	-.08
Verbal Memory T-Score	.00	-.23	-.21
Nonverbal Memory T-Score	.02	-.09	.16

Note: No significant correlations were observed; RIAS = *Reynolds Intellectual Assessment Scales* (Reynolds & Kamphaus, 2003); CAPI = *Child Abuse Potential Inventory* (Milner, 1990); PS = Parenting Scale (Arnolds, 1993)

Table 25

Hierarchical Linear Regression with Depression Alone Predicting CAPI Child Abuse Potential

	B	*SE*	*t*	*p*	*VIF*
Model 1—F (2, 29) = 15.65, R^2 = .52, p <.001					
CAPI Faking Good Index	-55.28	24.87	-2.22	.03	1.13
Total Types of Trauma, Lifetime (TEI)	13.88	3.41	4.07	<.001	1.13
Model 2— F (6, 25) = 10.62, R^2 = .72, p <.001, $R^2\Delta$ = .20, $p\Delta$ = .01					
CAPI Faking Good Index	-45.02	21.49	-2.09	.05	1.24
Total Types of Trauma, Lifetime (TEI)	.75	4.98	.15	.88	3.55
PTSD Symptom Total (PSS)	1.35	.94	1.45	.16	2.02
Depression Symptom Total (BDI)	3.89	1.15	3.39	.002	2.31
Alcohol Use (AUDIT-C)	-3.99	5.28	-.76	.46	1.13
Substance Use (DAST-10)	2.06	10.86	.19	.85	1.91
Model 3— F (8, 23) = 7.48, R^2 = .72, p <.001, $R^2\Delta$ = .004, $p\Delta$ = .84					
CAPI Faking Good Index	-45.28	22.42	-2.02	.06	1.26
Total Types of Trauma, Lifetime (TEI)	.86	5.64	.15	.88	4.24
PTSD Symptom Total (PSS)	1.09	1.11	.98	.34	2.66
Depression Symptom Total (BDI)	4.24	1.33	3.19	.004	2.90
Alcohol Use (AUDIT-C)	-4.29	5.58	-.77	.45	1.17
Substance Use (DAST-10)	1.56	12.38	.13	.90	2.32
Verbal Reasoning T-Score (RIAS)	.30	1.26	.24	.81	1.28
Nonverbal Recognition T-Score (RIAS)	.91	1.85	.49	.63	1.54

Note: CAPI = *Child Abuse Potential Inventory* (Milner, 1990); TEI = *Traumatic Events Inventory* (Gillespie et al., 2009); PSS = PTSD Symptom Scale (Foa & Tolin, 2000; Schwartz et al., 2005); BDI = *Beck Depression Inventory-II* (Beck et al., 1996); AUDIT-C = *Alcohol Use Disorders Identification Test* (Saunders et al., 1993); DAST-10 = *Drug Abuse Screening Test* (Skinner, 1982); RIAS = *Reynolds Intellectual Assessment Scales* (Reynolds & Kamphaus, 2003); VIF = Variance Inflation Factor

Table 26

Hierarchical Linear Regression with Verbal Reasoning and Depression Predicting PS Overreactive Parenting

	B	*SE*	*t*	*p*	*VIF*
Model 1—F (1, 30) = 1.92, R^2 = .06, p = .18					
Total Types of Trauma, Lifetime (TEI)	1.61	1.16	1.39	.18	1.00
Model 2— F (5, 26) = 2.31, R^2 = .31, p = .07, $R^2\Delta$ = .25, $p\Delta$ = .08					
Total Types of Trauma, Lifetime (TEI)	.21	2.02	.10	.92	3.54
PTSD Symptom Total (PSS)	-.79	.38	-2.08	.05	2.01
Depression Symptom Total (BDI)	1.15	.46	2.48	.02	2.29
Alcohol Use (AUDIT-C)	-.06	2.14	-.03	.98	1.13
Substance Use (DAST-10)	3.54	4.25	.83	.41	1.78
Model 3— F (7, 24) = 2.69, R^2 = .44, p = .03, $R^2\Delta$ = .13, $p\Delta$ = .08					
Total Types of Trauma, Lifetime (TEI)	-1.75	2.06	-.85	.41	4.22
PTSD Symptom Total (PSS)	-.54	.40	-1.34	.19	2.63
Depression Symptom Total (BDI)	1.21	.49	2.48	.02	2.88
Alcohol Use (AUDIT-C)	.74	2.04	.36	.72	1.17
Substance Use (DAST-10)	7.64	4.35	1.76	.09	2.13
Verbal Reasoning T-Score (RIAS)	1.01	.46	2.20	.04	1.27
Nonverbal Recognition T-Score (RIAS)	-.82	.68	-1.21	.24	1.53

Note: PS = Parenting Scale (Arnolds, 1993); TEI = *Traumatic Events Inventory* (Gillespie et al., 2009); PSS = PTSD Symptom Scale (Foa & Tolin, 2000; Schwartz et al., 2005); BDI = *Beck Depression Inventory-II* (Beck et al., 1996); AUDIT-C = *Alcohol Use*

Disorders Identification Test (Saunders et al., 1993); DAST-10 = *Drug Abuse Screening Test* (Skinner, 1982); RIAS = *Reynolds Intellectual Assessment Scales* (Reynolds & Kamphaus, 2003); VIF = Variance Inflation Factor

Table 27

Hierarchical Linear Regression with PTSD Alone Predicting PS Lax Parenting

	B	*SE*	*t*	*p*	*VIF*
Model 1—F (1, 30) = .61, R^2 = .02, p = .44					
Total Types of Trauma, Lifetime (TEI)	1.43	1.83	.78	.44	1.00
Model 2— F (5, 26) = 1.94, R^2 = .27, p = .12, $R^2\Delta$ = .25, $p\Delta$ = .09					
Total Types of Trauma, Lifetime (TEI)	-3.88	3.19	-1.22	.23	3.54
PTSD Symptom Total (PSS)	1.29	.60	2.16	.04	2.01
Depression Symptom Total (BDI)	.55	.73	.76	.46	2.29
Alcohol Use (AUDIT-C)	3.68	3.38	1.09	.29	1.13
Substance Use (DAST-10)	2.11	6.71	.31	.76	1.78
Model 3— F (7, 24) = 1.54, R^2 = .31, p = .20, $R^2\Delta$ = .04, $p\Delta$ = .52					
Total Types of Trauma, Lifetime (TEI)	-5.41	3.52	-1.53	.14	4.22
PTSD Symptom Total (PSS)	1.41	.69	2.03	.05	2.63
Depression Symptom Total (BDI)	.71	.83	.85	.40	2.88
Alcohol Use (AUDIT-C)	4.22	3.49	1.21	.24	1.17
Substance Use (DAST-10)	5.24	7.43	.71	.49	2.13
Verbal Reasoning T-Score (RIAS)	.90	.78	1.15	.26	1.27
Nonverbal Recognition T-Score (RIAS)	-.37	1.15	-.32	.75	1.53

Note: PS = Parenting Scale (Arnolds, 1993); TEI = *Traumatic Events Inventory* (Gillespie et al., 2009); PSS = PTSD Symptom Scale (Foa & Tolin, 2000; Schwartz et al., 2005); BDI = *Beck Depression Inventory-II* (Beck et al., 1996); AUDIT-C = *Alcohol Use Disorders Identification Test* (Saunders et al., 1993); DAST-10 = *Drug Abuse Screening*

Test (Skinner, 1982); RIAS = *Reynolds Intellectual Assessment Scales* (Reynolds & Kamphaus, 2003); VIF = Variance Inflation Factor

Table 28

Mother and Child PTSD Symptoms and Trauma Exposure Compared across Mother-reported Use of Substances during Pregnancy

		Maternal PTSD Symptoms (PSS)	Maternal Lifetime Trauma (TEI)	Child PTSD Symptoms (UCLA)	Child Lifetime Trauma (VEX-R)
Alcohol	No (*N* = 35)	18.16 (14.68)	5.63 (3.57)	22.11 (14.77)	9.18 (5.25)
	Yes (*N* = 3)	23.00 (17.77)	7.67 (3.21)	31.67 (20.98)	11.00 (4.58)
Cigarettes	No (*N* = 33)	16.45 (13.96)*	5.19 (3.32)**	22.77 (15.41)	9.48 (5.26)
	Yes (*N* = 5)	32.00 (13.45)	9.80 (2.17)	24.00 (15.77)	8.00 (4.69)
Illicit drugs	No (*N* = 32)	17.09 (14.93)	5.38 (3.45)†	24.32 (15.01)	9.65 (5.47)
	Yes (*N* = 6)	26.17 (11.94)	8.00 (3.52)	15.83 (16.61)	3.88 (1.58)

Note: †$p < .10$, *$p < .05$, **$p < .01$; PSS = PTSD Symptom Scale (Foa & Tolin, 2000; Schwartz et al., 2005); TEI = number of different types of trauma experienced, *Traumatic Events Inventory* (Gillespie et al., 2009); UCLA = *UCLA PTSD index for DSM-IV, Child Version* (Rodriguez, et al., 1999); VEX-R = number of different types of violence exposed to, *Violence Exposure Scale for Children—Revised* (Fox & Leavitt, 1995)

Table 29

Pearson Correlations between Mother (N = 45) and Child (N = 45) Mental Health and Trauma Exposure

	PSS_T	PSS_I	PSS_A	PSS_H	BDI	AUD	DAST	TEI_T	TEI_{ChAb}	TEI_{NoChAb}
$UCLA_T$	.19	.22	.18	.13	.07	.02	-.06	-.04	.02	-.01
$UCLA_I$	.21	.23	.19	.15	.06	-.04	-.11	.02	.02	.08
$UCLA_A$	.13	.19	.11	.05	.13	-.002	-.03	-.11	.02	-.12
$UCLA_H$	.19	.19	.16	.16	.06	.04	-.03	.01	-.03	.04
Anx	.13	.15	.19	.04	.08	.07	.08	-.09	-.06	-.05
Dep	-.02	-.03	-.03	.01	-.03	.06	.08	-.16	-.06	-.16
SchPrb	-.02	-.03	.002	-.04	-.14	.12	.18	-.05	.06	-.08
AttPrb	-.11	-.10	-.16	-.02	.08	-.06	.01	-.19	-.07	-.16
Hyp	-.01	.01	-.05	.01	-.06	-.08	-.18	-.14	-.14	-.13
VEX_{Exp}	.01	.03	.04	-.03	.02	-.10	-.12	.04	.31*	-.04
VEX_{Wit}	.06	.06	.13	-.04	.06	-.04	-.18	-.06	.13	-.09
VEX_{Tot}	.04	.05	.09	-.03	.05	-.07	-.16	-.02	.23	-.07

Note: *$p < .05$; UCLA = *UCLA PTSD index for DSM-IV, Child Version* ($_T$ = Total, $_I$ = intrusive, $_A$ = avoidance, and $_H$ = hyperarousal symptom scores; Rodriguez, Steinberg, & Pynoos, 1999); Anx = Anxiety, Dep = Depression, SchPrb = School Problems, AttProb = Attention Problems, Hyp = Hyperactivity (*Behavioral Assessment System for Children—Second Edition, Self-Report* Reynolds & Kamphaus, 2004); VEX = *Violence Exposure Scale for Children—Revised* ($_{Exp}$ = total types of trauma experienced, $_{Wit}$ = total types of trauma witnessed, $_{Tot}$ = total types of trauma experienced and witnessed; Fox & Leavitt, 1995); PSS = PTSD Symptom Scale ($_T$ = total score, $_I$ = intrusive symptom score, $_A$ = avoidance symptom scores, $_H$ = hyperarousal symptom score; Foa & Tolin, 2000; Schwartz et al., 2005); BDI = *Beck Depression Inventory-II* (Beck et al., 1996); EDS = *Emotional Dysregulation Scale* (Bradley et al., 2011); AUD = *Alcohol Use Disorders Identification Test* (Saunders et al., 1993); DAST = *Drug Abuse Screening Test* (Skinner, 1982); TEI = *Traumatic Events Inventory* ($_T$ = total types of lifetime trauma exposure, $_{ChAb}$ = total types of child abuse experienced; $_{NoChAb}$ = total types of lifetime trauma exposure, excluding child abuse; Gillespie et al., 2009)

Table 30

Pearson Correlations between Mother (N = 45) and Child (N = 45) RIAS Performance

	CIX_m	VIX_m	GWH_m	VRZ_m	NIX_m	OIO_m	WHM_m	CMX_m	VRM_m	NVM_m
CIX_c	.28†	.38*	.41**	.44**	.08	.15	.05	.36*	.31*	.24
VIX_c	.12	.28†	.31*	.27†	-.10	-.03	-.03	.23	.15	.21
GWH_c	-.02	.19	.12	.11	-.26	-.17	-.15	.15	.07	.17
VRZ_c	.21	.27†	.34*	.36*	.12	.11	.17	.27†	.21	.18
NIX_c	.35*	.34*	.36**	.45**	.25	.32*	.11	.38*	.36*	.19
OIO_c	.06	.09	.05	.24†	<.001	-.03	.04	.32**	.31*	.15
WHM_c	.40*	.37*	.44**	.40**	.32*	.43**	.12	.26†	.25	.12
CMX_c	.01	.12	.14	.17	.07	.04	.16	.20	.10	.22
VRM_c	.07	.17	.15	.07	-.06	.07	-.10	.16	.003	.27†
NVM_c	-.08	-.05	-.05	.11	-.01	-.03	.08	.05	.15	-.11

Note: †$p < .10$, *$p < .05$, **$p < .01$; RIAS = *Reynolds Intellectual Assessment Scales* (Reynolds & Kamphaus, 2003); $_c$ = child score; $_m$ = mother score; CIX = Composite Intelligence Index; VIX = Verbal Intelligence Index; GWH = Guess What T-Score; VRZ = Verbal Reasoning T-Score;

NIX = Nonverbal Intelligence Index; OIO = Odd-Item-Out T-Score; WHM = What's Missing? T-Score; CMX = Composite Memory Index; VRM = Verbal Memory T-Score; NVM = Nonverbal Memory T-Score

Table 31

Pearson Correlations between Mother (N = 45) Mental Health, Child Abuse Potential, and Trauma Exposure and Child (N = 45) Intelligence and Memory

	PSS_T	PSS_R	PSS_A	PSS_H	BDI	AUD	DAST	TEI_{Tot}	TEI_{ChAb}	TEI_{NoChAb}	TEI_{age1st}	Age	Edu	Income
CIX	.12	.05	.14	.13	.02	-.23	-.06	.17	.02	.15	-.17	.07	.19	.03
VIX	.07	-.02	.13	.05	.00	-.11	-.01	.17	-.05	.16	-.11	.15	.22	.09
GWH	.17	.03	.24	.17	.10	.00	-.03	.20	-.02	.18	-.12	.04	.10	.03
VRZ	-.12	-.12	-.07	-.15	-.19	-.28	-.02	.03	-.15	.03	-.03	.21	.36*	.18
NIX	.16	.11	.12	.19	.04	-.27	-.08	.14	.09	.11	-.15	.00	.14	-.02
OIO	.20	.14	.16	.24	.14	-.11	-.02	-.02	.06	-.09	.09	.03	.30†	.12
WHM	.08	.07	.06	.10	-.02	-.30†	-.08	.20	.09	.21	-.25†	-.01	-.01	-.11
CMX	-.10	-.21	-.08	.01	-.24	-.18	.08	.04	-.13	.08	-.20	-.13	.20	.23
VRM	.00	-.15	-.02	.14	-.04	-.26	.11	.11	-.05	.08	-.16	-.32*	.27	.12
NVM	-.05	-.04	-.01	-.08	-.23	.10	.04	.00	-.03	.07	-.18	.19	-.04	.17

Note: †$p < .10$, *$p < .05$; PSS = PTSD Symptom Scale ($_T$ = total score, $_R$ = re-experiencing score, $_A$ = avoidance score, $_H$ = hyperarousal score; Foa & Tolin, 2000; Schwartz et al., 2005); BDI = *Beck Depression Inventory-II* (Beck et al., 1996); AUD = *Alcohol Use Disorders Identification* Test

(Saunders et al., 1993); DAST = *Drug Abuse Screening Test* (Skinner, 1982); CAPI = Child Abuse Potential Inventory (Milner, 1990); TEI = *Traumatic Events Inventory* ($_{Tot}$ = total types of lifetime trauma exposure, $_{ChAb}$ = total types of child abuse experienced; $_{NoChAb}$ = total types of lifetime trauma exposure, excluding child abuse; $_{age1st}$ = first age of any type of trauma; Gillespie et al., 2009); Edu = years of education

Table 32

Pearson Correlations between Maternal Parenting Variables and Child Trauma, Mental Health, and Intelligence and Memory

	Abuse Potential (CAPI)	Overreactivity (PS)	Laxness (PS)
Child Trauma and Mental Health			
PTSD Total (UCLA)	.12	.02	.02
PTSD Re-experiencing (UCLA)	.09	.06	.07
PTSD Avoidance (UCLA)	.18	.02	.05
PTSD Hyperarousal (UCLA)	.06	-.04	-.03
Anxiety T-Score (BASC-2)	.01	-.13	.20
Depression T-Score (BASC-2)	-.02	.01	.19
School Problems T-Score (BASC-2)	-.01	-.06	.20
Attention Problems T-Score (BASC-2)	.05	.04	.11
Hyperactivity T-Score (BASC-2)	.02	.09	.14
Total Types Violence Experienced (VEX-R)	-.12	-.17	.04
Total Types Violence Witnessed (VEX-R)	.06	.16	.21
Total Types Violence (VEX-R)	-.04	.09	.19
Child Intelligence and Memory			
Composite Intelligence Index (RIAS)	.13	.15	.08
Verbal Intelligence Index (RIAS)	.14	.15	-.10
Guess What T-Score (RIAS)	.26†	.20	-.04

Verbal Reasoning T-Score (RIAS)	-.03	.06	-.15
Nonverbal Intelligence Index (RIAS)	.07	.07	.24
Odd-Item-Out T-Score (RIAS)	.18	.05	.06
What's Missing? T-Score (RIAS)	-.04	.05	.27†
Composite Memory Index (RIAS)	.04	.12	-.20
Verbal Memory T-Score (RIAS)	<.001	.09	-.22
Nonverbal Memory T-Score (RIAS)	.10	.09	-.02

Note: †$p < .10$; CAPI = *Child Abuse Potential Inventory* (Milner, 1990); PS = Parenting Scale (Arnolds, 1993); UCLA = *UCLA PTSD index for DSM-IV, Child Version* (Rodriguez, Steinberg, & Pynoos, 1999); BASC-2 = *Behavioral Assessment System for Children—Second Edition, Self-Report* (Reynolds & Kamphaus, 2004); VEX-R = *Violence Exposure Scale for Children—Revised* (Fox & Leavitt, 1995); RIAS = *Reynolds Intellectual Assessment Scales* (Reynolds & Kamphaus, 2003)

Table 33

Child RIAS Performance Compared Across Mother-reported Use of Substances during Pregnancy (N = 38)

		Alcohol (*N* = 3)	Cigarettes (*N* = 5)	Illicit Drugs (*N* = 6)
CIX	No	92.31 (9.99)	91.61 (9.73)	90.90 (9.68)*
	Yes	94.67 (9.61)	99.50 (8.85)	100.33 (6.77)
VIX	No	89.97 (10.36)	89.55 (10.20)	89.14 (10.32)
	Yes	91.67 (13.20)	94.50 (12.58)	94.83 (10.36)
GWH	No	40.59 (9.11)	39.94 (8.45)†	39.69 (8.77)*
	Yes	45.00 (11.00)	49.00 (12.03)	47.17 (9.33)
VRZ	No	43.79 (7.77)	43.81 (7.86)	43.50 (8.04)
	Yes	41.33 (8.39)	41.75 (7.23)	44.00 (6.48)
NIX	No	97.88 (11.65)	97.06 (11.64)	96.26 (11.52)*
	Yes	101.00 (13.45)	107.00 (7.70)	107.83 (6.49)
OIO	No	51.38 (7.77)	51.12 (7.65)	50.90 (7.86)
	Yes	52.33 (1.53)	54.25 (5.56)	54.33 (4.18)
WHM	No	44.44 (10.75)	43.70 (11.08)	42.90 (10.88)*
	Yes	47.00 (15.13)	52.50 (5.32)	53.67 (5.61)
CMX	No	95.71 (8.65)	96.18 (9.02)	95.35 (8.90)
	Yes	100.00 (9.85)	95.00 (5.89)	99.67 (6.98)
VRM	No	42.91 (7.53)*	43.33 (8.14)	42.77 (8.19)
	Yes	52.00 (9.64)	46.25 (6.70)	48.17 (5.00)
NVM	No	50.94 (7.80)	51.06 (7.87)	50.65 (8.00)

Yes	47.33 (1.53)	47.25 (1.89)	50.67 (5.05)

Note: †$p < .10$, *$p < .05$; RIAS = *Reynolds Intellectual Assessment Scales* (Reynolds & Kamphaus, 2003); CIX = Composite Intelligence Index; VIX = Verbal Intelligence Index; GWH = Guess What T-Score; VRZ = Verbal Reasoning T-Score; NIX = Nonverbal Intelligence Index; OIO = Odd-Item-Out T-Score; WHM = What's Missing? T-Score; CMX = Composite Memory Index; VRM = Verbal Memory T-Score; NVM = Nonverbal Memory T-Score

Table 34

Hierarchical Linear Regression Predicting PTSD Symptoms in Children

	B	*SE*	*t*	*p*	*VIF*
Model 1—F (3, 32) = .94, R^2 = .08, p = .43					
Mother Verbal Intelligence (VIX)	-.15	.25	-.58	.56	1.09
Mother Total Types of Trauma, Lifetime (TEI)	-.92	.94	-.97	.34	1.67
Mother PTSD Symptom Total (PSS)	.34	.21	1.60	.12	1.61
Model 2—F (4, 31) = .88, R^2 = .11, p = .49, $R^2\Delta$ = .02, $p\Delta$ = .41					
Mother Verbal Intelligence (VIX)	-.17	.25	-.69	.50	1.11
Mother Total Types of Trauma, Lifetime (TEI)	-.90	.95	-.95	.35	1.68
Mother PTSD Symptom Total (PSS)	.33	.21	1.56	.13	1.61
Child Age	1.66	1.98	.84	.41	1.02
Model 3— F (5, 50) = 1.35, R^2 = .18, p = .27, $R^2\Delta$ = .08, $p\Delta$ = .09					
Mother Verbal Intelligence (VIX)	-.06	.25	-.22	.83	1.20
Mother Total Types of Trauma, Lifetime (TEI)	-.94	.92	-1.02	.31	1.68
Mother PTSD Symptom Total (PSS)	.30	.21	1.44	.16	1.63
Child Age	-.24	2.20	-.11	.92	1.35
Child Total Types of Trauma (VEX-R)	.98	.56	1.74	.09	1.38
Model 4— F (6, 29) = 1.53, R^2 = .24, p = .21, $R^2\Delta$ = .08, $p\Delta$ = .16					
Mother Verbal Intelligence (VIX)	.01	.25	.04	.97	1.24
Mother Total Types of Trauma, Lifetime (TEI)	-.77	.91	-.85	.40	1.70
Mother PTSD Symptom Total (PSS)	.28	.20	1.36	.18	1.64

Child Age	.00	2.17	.00	1.00	1.36
Child Total Types of Trauma (VEX-R)	.75	.57	1.31	.20	1.49
Child Verbal Intelligence (VIX)	-.34	.23	-1.46	.15	1.19

Note: VIX = Verbal Intelligence Index, Reynolds Intellectual Assessment Scales (Reynolds & Kamphau, 2003); TEI = *Traumatic Events Inventory* (Gillespie et al., 2009); BDI –II = *Beck Depression Inventory-II* (Beck et al., 1996); VEX-R = *Violence Exposure Scale for Children—Revised* (Fox & Leavitt, 1995); VIF = Variance Inflation Factor

Table 35

Hierarchical Linear Regression Predicting PTSD Symptoms in Boys

	B	*SE*	*t*	*p*	*VIF*
Model 1—F (3, 13) = .54, R^2 = .11, p = .66					
Mother Verbal Intelligence (VIX)	-.21	.45	-.46	.65	1.11
Mother Total Types of Trauma, Lifetime (TEI)	-.04	1.21	-.04	.97	1.53
Mother PTSD Symptom Total (PSS)	.30	.29	1.04	.32	1.61
Model 2—F (4, 12) = .81, R^2 = .21, p = .54, $R^2\Delta$ = .10, $p\Delta$ = .24					
Mother Verbal Intelligence (VIX)	-.19	.44	-.43	.68	1.11
Mother Total Types of Trauma, Lifetime (TEI)	-.15	1.18	-.13	.90	1.54
Mother PTSD Symptom Total (PSS)	.36	.28	1.26	.23	1.66
Child Age	-3.62	2.90	-1.25	.24	1.04
Model 3—F (5, 11) = .60, R^2 = .22, p = .70, $R^2\Delta$ = .002, $p\Delta$ = .87					
Mother Verbal Intelligence (VIX)	-.16	.49	-.32	.76	1.28
Mother Total Types of Trauma, Lifetime (TEI)	-.16	1.24	-.13	.90	1.54
Mother PTSD Symptom Total (PSS)	.34	.31	1.12	.29	1.78
Child Age	-3.79	3.17	-1.19	.26	1.14
Child Total Types of Trauma (VEX-R)	.15	.88	.17	.87	1.36
Model 4—F (6, 10) = .99, R^2 = .37, p = .48, $R^2\Delta$ = .37, $p\Delta$ = .15					
Mother Verbal Intelligence (VIX)	-.14	.46	-.31	.76	1.28
Mother Total Types of Trauma, Lifetime (TEI)	-.16	1.16	-.14	.89	1.54
Mother PTSD Symptom Total (PSS)	.23	.30	.78	.45	1.89
Child Age	-4.13	2.98	-1.38	.20	1.15

Child Total Types of Trauma (VEX-R)	.01	.83	.02	.99	1.37
Child Guess What? Score (GWH)	-.74	.47	-1.58	.14	1.17

Note: GWH = Guess What?, Reynolds Intellectual Assessment Scales (Reynolds & Kamphau, 2003); TEI = *Traumatic Events Inventory* (Gillespie et al., 2009); BDI –II = *Beck Depression Inventory-II* (Beck et al., 1996); VEX-R = *Violence Exposure Scale for Children—Revised* (Fox & Leavitt, 1995); VIF = Variance Inflation Factor

Table 36

Hierarchical Linear Regression Predicting PTSD Symptoms in Girls

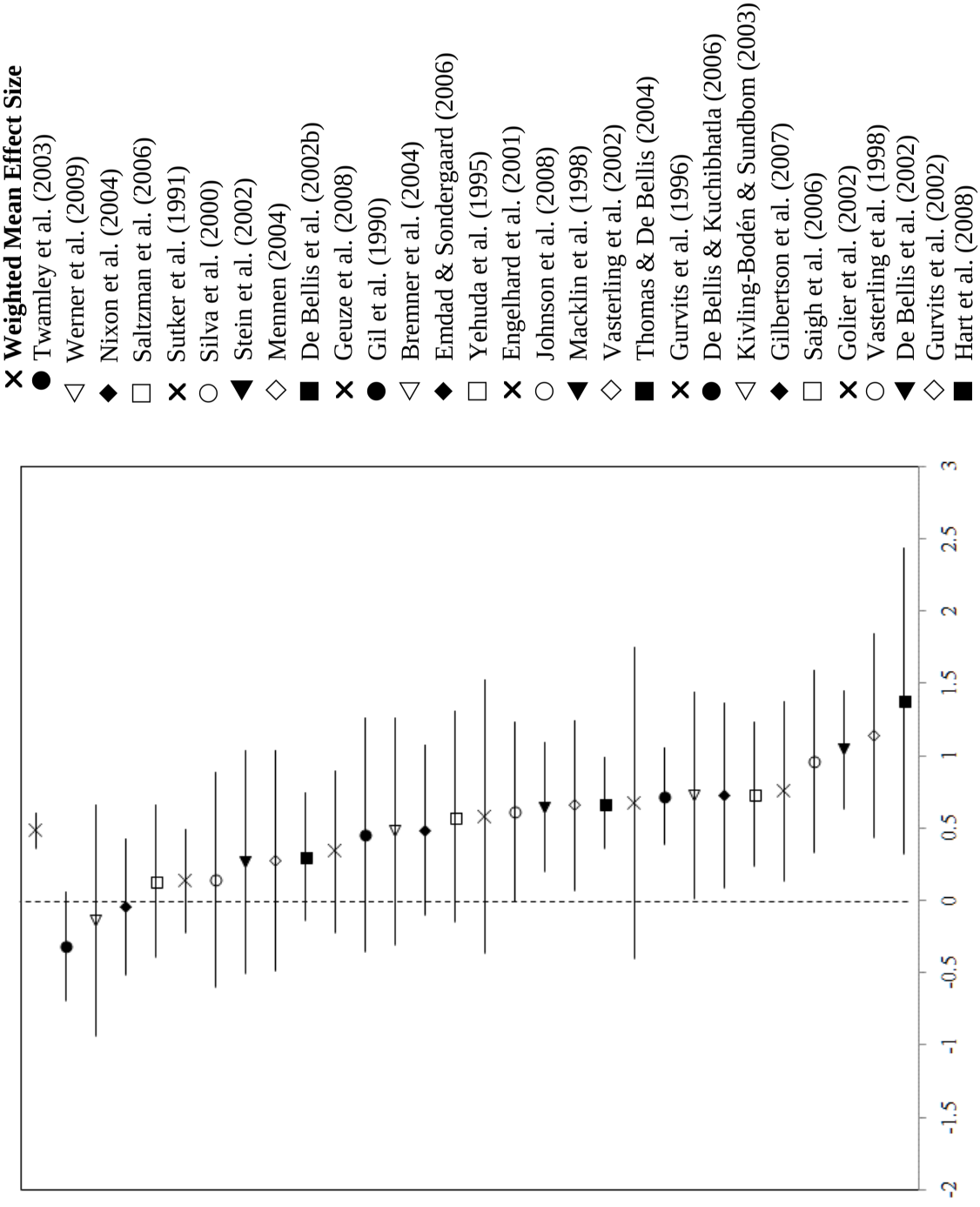
	B	*SE*	*t*	*p*	*VIF*
Model 1—F (3, 16) = 1.58, R^2 = .23, p = .23					
Mother Verbal Intelligence (VIX)	.14	.37	.37	.72	1.45
Mother Total Types of Trauma, Lifetime (TEI)	-3.83	1.92	-1.99	.06	2.13
Mother PTSD Symptom Total (PSS)	.48	.38	1.28	.22	1.60
Model 2—F (4, 15) = 1.99, R^2 = .35, p = .15, $R^2\Delta$ = .12, $p\Delta$ = .12					
Mother Verbal Intelligence (VIX)	.05	.36	.13	.90	1.48
Mother Total Types of Trauma, Lifetime (TEI)	-3.86	1.83	-2.11	.05	2.13
Mother PTSD Symptom Total (PSS)	.54	.36	1.51	.15	1.62
Child Age	4.18	2.53	1.65	.12	1.05
Model 3—F (5, 14) = 2.57, R^2 = .48, p = .08, $R^2\Delta$ = .13, $p\Delta$ = .08					
Mother Verbal Intelligence (VIX)	.19	.34	.55	.59	1.55
Mother Total Types of Trauma, Lifetime (TEI)	-2.52	1.83	-1.37	.19	2.51
Mother PTSD Symptom Total (PSS)	.65	.34	1.93	.07	1.67
Child Age	.30	3.12	.10	.93	1.86
Child Total Types of Trauma (VEX-R)	2.05	1.09	1.88	.08	2.57
Model 4—F (6, 13) = 2.05, R^2 = .49, p = .13, $R^2\Delta$ = .008, $p\Delta$ = .67					
Mother Verbal Intelligence (VIX)	.20	.35	.56	.58	1.56
Mother Total Types of Trauma, Lifetime (TEI)	-2.34	1.93	-1.21	.25	2.63
Mother PTSD Symptom Total (PSS)	.64	.35	1.83	.09	1.68
Child Age	.40	3.22	.12	.90	1.87

Child Total Types of Trauma (VEX-R)	1.98	1.14	1.74	.11	2.63
Child Verbal Reasoning Score (VRZ)	-.18	.41	-.44	.67	1.25

Note: VRZ = Verbal Reasoning, Reynolds Intellectual Assessment Scales (Reynolds & Kamphau, 2003); TEI = *Traumatic Events Inventory* (Gillespie et al., 2009); BDI –II = *Beck Depression Inventory-II* (Beck et al., 1996); VEX-R = *Violence Exposure Scale for Children—Revised* (Fox & Leavitt, 1995)

Figure Caption

Figure 1. Error-bar chart displaying the effect sizes and 95% confidence intervals for studies included in the meta-analysis (dashed line indicates effect size of 0)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Werner et al. (2009)
Nixon et al. (2004)
Saltzman et al. (2006)
Sutker et al. (1991)
Silva et al. (2000)
Stein et al. (2002)
Mennen (2004)
De Bellis et al. (2002b)
Geuze et al. (2008)
Gil et al. (1990)
Bremner et al. (2004)
Emdad & Sondergaard (2006)
Yehuda et al. (1995)
Engelhard et al. (2001)
Johnson et al. (2008)
Macklin et al. (1998)
Vasterling et al. (2002)
Thomas & De Bellis (2004)
Gurvits et al. (1996)
De Bellis & Kuchibhatla (2006)
Kivling-Bodén & Sundbom (2003)
Gilbertson et al. (2007)
Saigh et al. (2006)
Golier et al. (2002)
Vasterling et al. (1998)
De Bellis et al. (2002)
Gurvits et al. (2002)
Hart et al. (2008)
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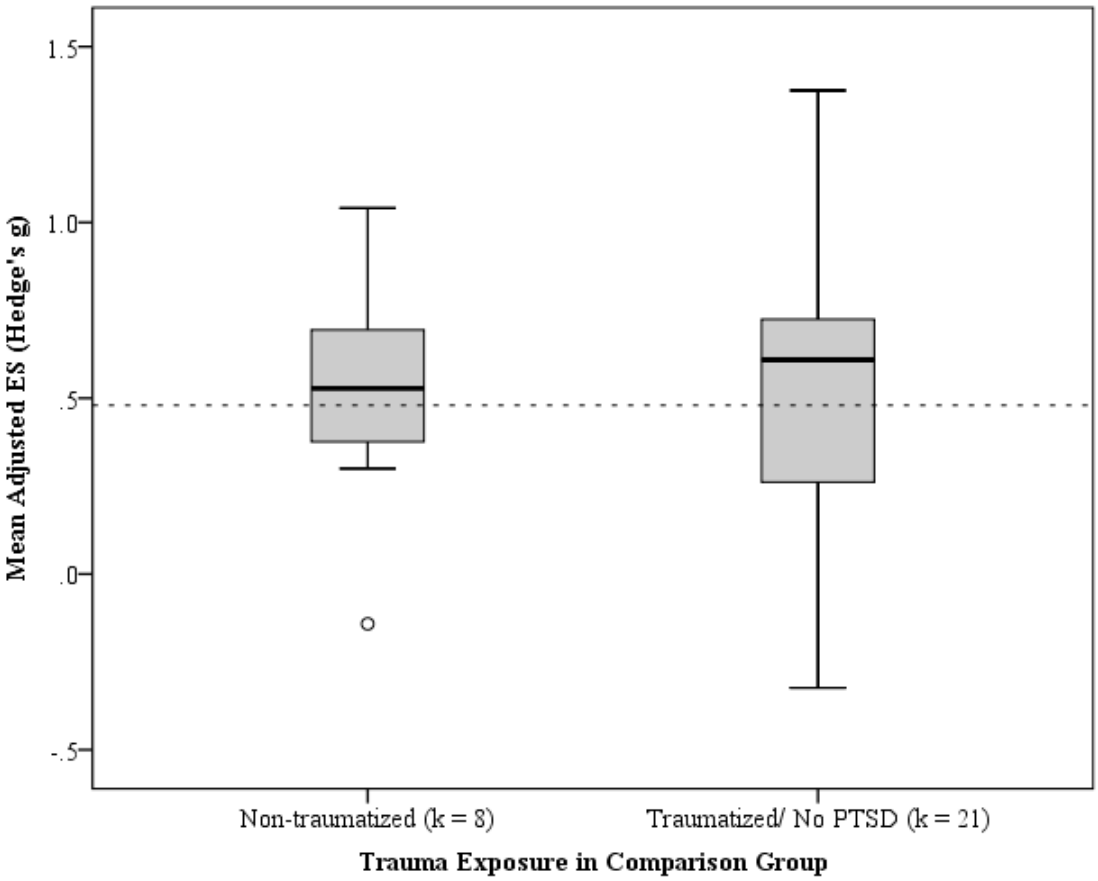
-1
-0.5
0
0.5
1
1.5
2
2.5
3

Figure Caption

Figure 2. Error-bar chart displaying weighted mean effect sizes for studies including traumatized comparison groups versus studies using non-traumatized comparison groups (dashed line indicates weighted mean effect size from primary meta-analysis)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.5
.0
-.5
Mean Adjusted ES (Hedge's g)
Non-traumatized (k = 8)
Traumatized/ No PTSD (k = 21)
Trauma Exposure in Comparison Group

Figure Caption

Figure 3. Error-bar chart displaying weighted mean effect sizes for verbal intelligence, nonverbal intelligence, and combined intelligence (dashed line indicates weighted mean effect size from primary meta-analysis)

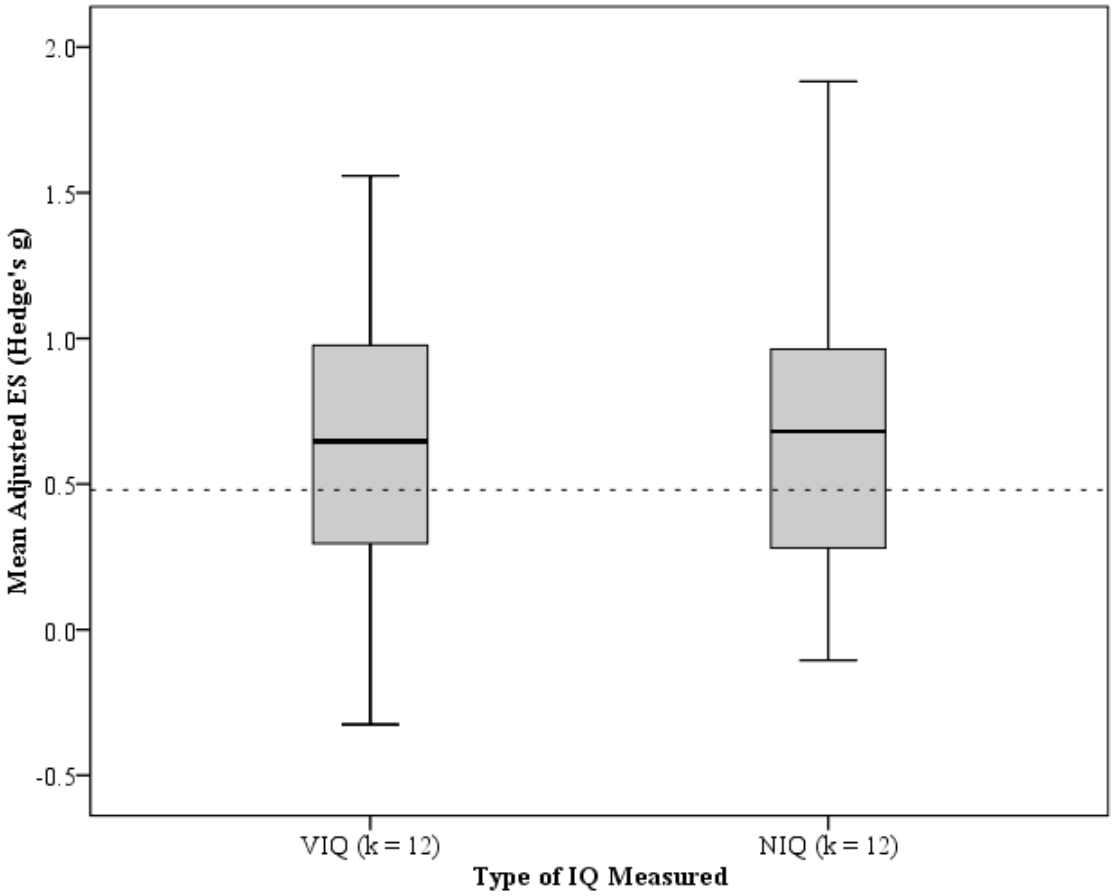

2.0
1.5
1.0
0.5
0.0
-0.5
Mean Adjusted ES (Hedge's g)
VIQ (k = 12)
NIQ (k = 12)
Type of IQ Measured

Figure Caption

Figure 4. Error-bar chart displaying weighted mean effect sizes for studies including measures of working memory in the assessment of intelligence versus studies not including measure of working memory (dashed line indicates weighted mean effect size from primary meta-analysis)

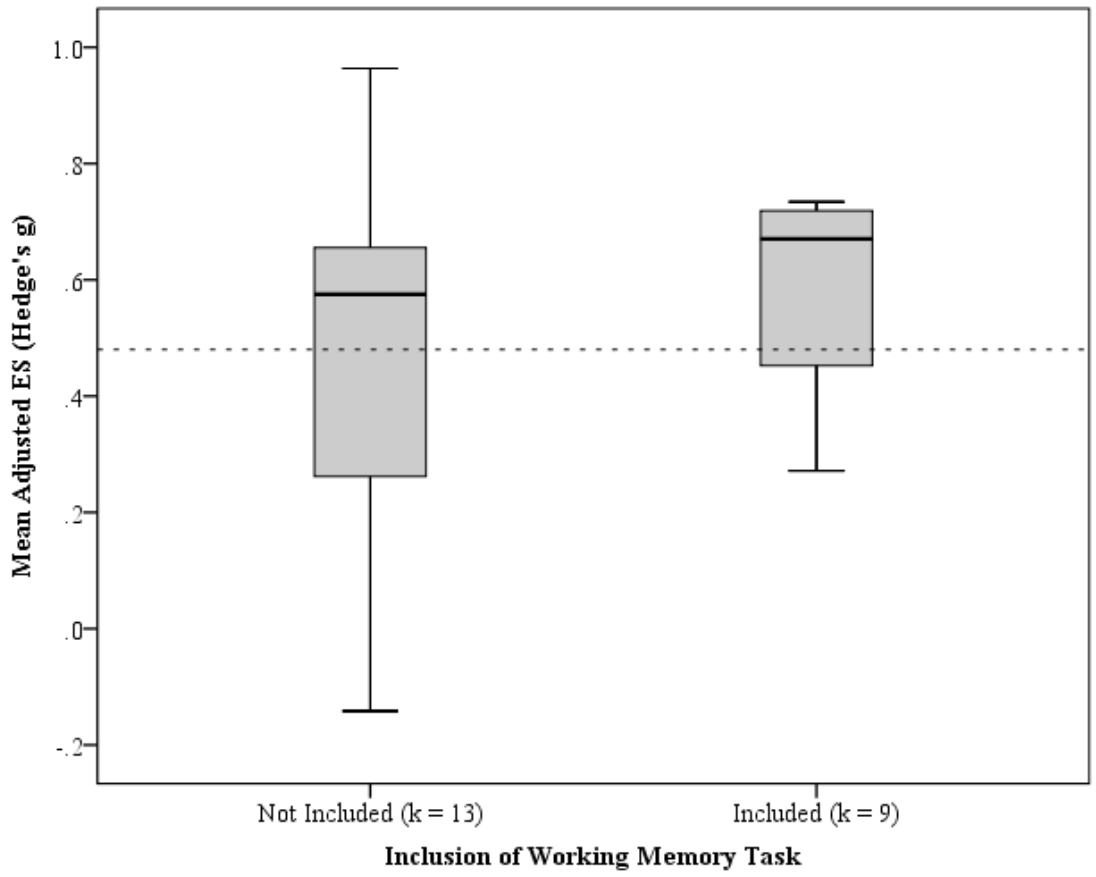
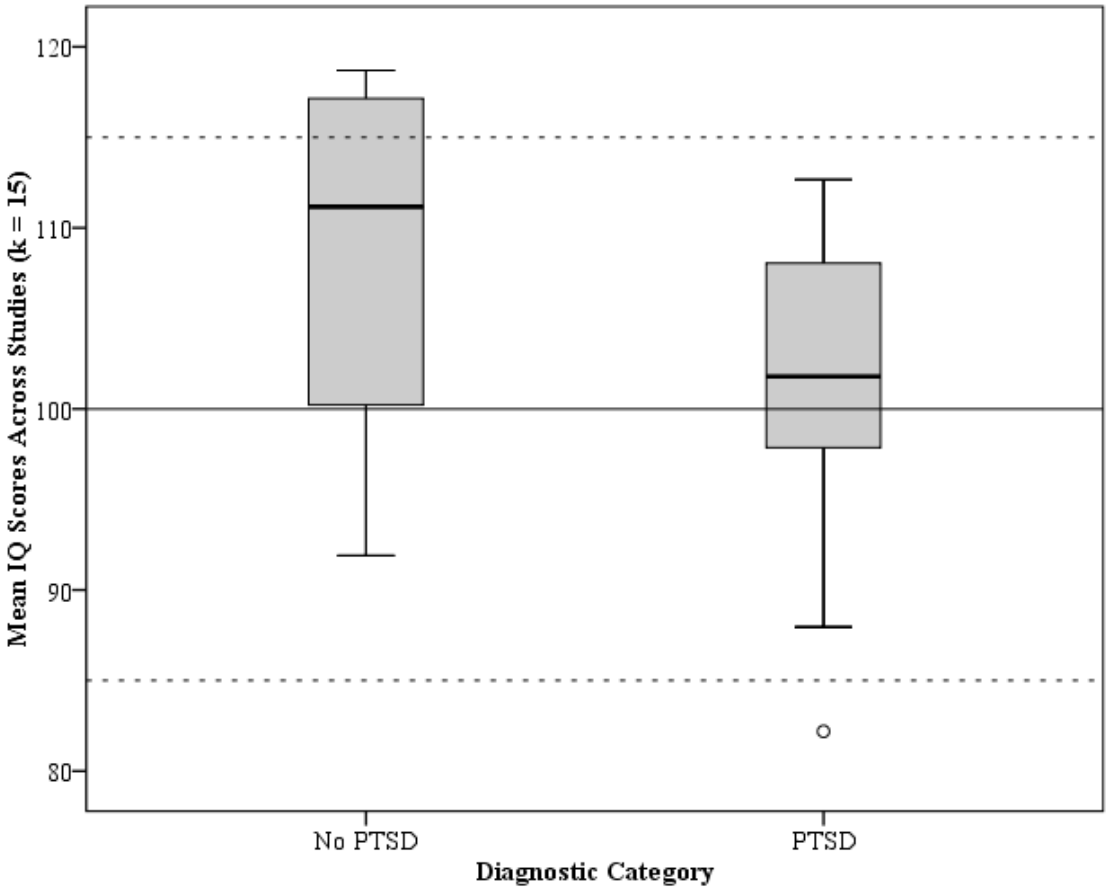

1.0
.8
.6
.4
.2
.0
-.2
Mean Adjusted ES (Hedge's g)
Not Included (k = 13)
Included (k = 9)
Inclusion of Working Memory Task

Figure Caption

Figure 5. Error-bar chart displaying mean intelligence test performance for individuals with and without PTSD across studies included in the meta-analysis (solid line indicates population mean of 100 and dashed line indicates population standard deviation of 15)


120
110
100
90
80
Mean IQ Scores Across Studies (k = 15)
No PTSD
PTSD
Diagnostic Category