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Memory functioning in children exposed to domestic violence trauma

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MEMORY FUNCTIONING IN CHILDREN EXPOSED TO DOMESTIC VIOLENCE TRAUMA

By

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ABSTRACT

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MEMORY FUNCTIONING IN CHILDREN EXPOSED
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2001/02
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This thesis describes the memory functioning in children exposed to domestic violence trauma. Research findings suggest that adult participants with a clinical diagnosis of Post-Traumatic Stress Disorder (PTSD) as defined by the Diagnostic Statistical Manual-IV (American Psychiatric Association, 1994) suffer from impairments in memory associated with reductions in hippocampal volume. However, a question arises as to whether children who have been exposed to domestic violence trauma will differ from controls in their memory functioning. A question also arises as to the relationship between the children's parent-reported behaviors and their memory functioning. The Children's Memory Scale (CMS) (Cohen, 1997) was administered to 22 children residing at a shelter for battered women in Delran, New Jersey. The children's mothers completed the Child Behavioral Checklist (CBC) (Achenbach, 1991).

The data were analyzed comparing the children's CMS (Cohen, 1997) scores to those of the CMS standardization sample, and comparing the children's CMS (Cohen) results to their CBC (Achenbach, 1991) results. Findings indicate that the children differed significantly from controls only in their CMS (Cohen) delayed verbal recall scores. No significant correlations were revealed for the CMS (Cohen) and CBC (Achenbach) indices. Implications for future research are discussed.

MINI-ABSTRACT

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This study describes the memory performance in children exposed to domestic violence trauma, as well their parent-reported behaviors. On the Children's Memory Scale (CMS) (Cohen, 1997), participants (N=22) scored significantly lower than the standardization sample only in delayed verbal recall. No correlations were found between CMS (Cohen) and Child Behavioral Checklist (Achenbach, 1991) data.

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INTRODUCTION

Stress can be characterized in terms of behavior and physiology. With respect to behavior, stress can be defined as any stimulus that is a threat to homeostasis (Dorio, Viau, & Meaney, 1993). In other words, stress is an environmental factor that could alter the normal functioning of an organism, including the way the organism perceives the environment and the self.

With respect to physiology, stress is characterized by the activation of autonomic processes related to the stress response. The main stress response involves the hypothalamic-pituitary-adrenal (HPA) axis. In the HPA axis, the hypothalamus releases corticosterone releasing factor, or CRF, to the anterior pituitary. The anterior pituitary then manufactures and releases adrenocorticotrophic hormone (ACTH) in response. From the circulatory system, ACTH reaches the adrenal cortex, where either corticosterone or cortisol (depending on the system) is released (Stansbury & Gunnar, 1994). The HPA axis is under the regulation of a complex neural system which increases cortisol production due to hypothalamic stimulation under conditions of stress. Once cortisol production is increased, the brain's inhibitory feedback systems, such as the hippocampus and pituitary, reduce the future production of CRF and ACTH (Sapolsky, Krey, & McEwen, 1986).

While stress is an environmental factor that can interfere with the normal functioning of an organism including its world view, trauma involves stress of a degree and severity that it can overwhelm the organism's coping mechanisms

(Hubbard et al., 1995). In addition, a salient characteristic of a traumatic experience is the interpretation an individual assigns to them (Cicchetti & Toth, 1997). In contrast to chronic stress, trauma can be associated with a single incident; the severity of traumatic events is such that a single incident can have profound effects on an individual's well-being and development (Hubbard, Realmuto, Northwood, & Masten, 1995).

It is pertinent to explain that while I use the term "trauma" throughout this paper, there is no agreement upon the operational definition of "trauma" in the literature. The DSM-IV definition of trauma focuses on a perceived threat of death or bodily injury, or the witnessing of death or bodily injury (American Psychiatric Association, 1994), but this definition is agreed upon loosely at best, and is not referred to in much of the scientific research. In addition, many of the conditions examined in the research are rather tame (e.g. a visit to the dentist's office or a sprained ankle) compared to the extreme conditions children are frequently subjected to in our society (e.g. witnessing a drive by shooting, being battered by a relative). The DSM-IV definition emphasizes threat to one's physical rather than emotional status. Many theorists hypothesize that emotional betrayal may be the salient feature of traumatic exposure (Freyd, 1996; Shay, 1996).

Associated with this idea is the issue of the cause of trauma. While natural disasters may cause tremendous stress, they are perceived much differently than incidents created by human beings of their own volition, such as rape, torture, or mutilation. Natural disasters do not incline the victim to engage

in self-blame, shame, guilt or secrecy to the same degree as human crimes. Within the range of human crimes, even the relationship between victim and perpetrator plays a critical role in how an event is perceived. Viewing community violence perpetrated by strangers may be quite different than viewing domestic violence at home (Cicchetti, Toth, & Lynch, 1997). This issue remains sticky, making research comparisons a difficult matter.

Terr (1991) proposes thinking of trauma as two typologies. Type I trauma consists of a single shocking incident. While it may be a protracted incident, such as a kidnapping in which a child is held hostage for a period of time, the incident, once over, is not repeated. In contrast, Type II trauma is reoccurring or chronic in nature, such as numerous instances of sexual or physical abuse over a duration of weeks or years. Terr posits that Type I trauma produces vivid, meticulous memory, whereas Type II trauma produces sketchy or non-existent memories of the traumatic incidents. Terr theorizes that in order to handle repeated trauma, children will rely on dissociation, or find other ways to distance themselves from the experience. Terr theorizes that dissociation leads to weakened encoding, resulting in poorer memory of the traumatic experiences. The scope of this thesis entails the effects of Type II trauma on the general memory functioning of children, rather than the children's memories for traumatic events themselves.

While there is still some debate, cognitive psychologists tend to acknowledge two main types of memory: explicit (declarative) memory and implicit (nondeclarative) memory. Explicit memory is memory of which we are consciously aware as an adult. It can be retrieved as an image or thought, exists

in some temporal time frame, and can be discussed or referred to verbally. However, in an infant (nonverbal human), the requirement of conscious awareness is not applicable. Explicit memory enables humans to recall objects, events, places, and things associated with them. Explicit memory can occur in one iteration/trial and sometimes can involve the "self." For instance, the person may see him/herself according to a scene, such as what he/she was doing on September 11, 2001 when the World Trade Center collapsed. (Nelson & Carver, 1998).

In contrast, implicit memory is viewed as a combination of various other subcategories of memory. As a whole, it can be viewed as nondeclarative memory. Implicit memory is thought to be unconscious and thought to require numerous trials. In addition, it may not relate to the "self" in any way. An example of implicit memory is priming. This could involve presenting stimuli to subjects during a trial phase that are presented much more briefly during a testing phase. The subject's quicker or more reliable response to the items seen during priming as compared to similar but new material is taken as evidence that priming has occurred. Another example of implicit memory is procedural learning. In a Serial Reaction Time (SRT) task, subjects might be asked to respond to a pattern of images flashing across a computer screen. After several trials, the subject might not have a conscious knowledge of the image pattern. However, the subject might still respond with greater and greater speed, suggesting that the pattern has been learned at the unconscious level. Other examples of implicit

memory might include conditioning, skilled motor learning, and artificial grammar learning (Nelson & Carver, 1998).

Based on decades of research, it seems that explicit memory is disproportionately reliant upon structures of the medial temporal lobe, such as the hippocampus, rhinal cortex, and parahippocampal gyrus. In contrast, the structures associated with implicit memory depend upon the subtype of memory under consideration. Perceptual priming, for example, is likely to be reliant upon areas of the visual cortex. Likewise, auditory priming is likely to rely upon the auditory cortex. Conditioning is likely to depend upon certain brain stem nuclei and the cerebellum, whereas procedural learning is likely to be related to the basal ganglia. (Hiltz, 1995; Nelson & Carver, 1998; Schacter & Tulving, 1994).

Learning and memory require alterations in the nervous system, most likely via synapse formation. Researchers Greenough et al. suggest two mechanisms by which synapses are formed, leading to changes in the nervous system as a result of environmental stimuli (Black, Jones, Nelson, & Greenough, 1998; Greenough & Black, 1992). The first, "experience-expectant" synaptogenesis, is a process of synaptic formation requiring some minimal level of stimuli exposure. For example, the acquisition of binocular depth perception requires normal visual input in order to achieve ocular dominance (Crair, Gillespie, & Stryker, 1998). If both eyes are not aligned to converge effectively on a distant target, eyesight fails to develop properly. If no steps are taken to create proper alignment by the time the amount of synapses reaches the adult

quantity (around the end of the preschool period), stereoscopic vision is permanently sacrificed.

In contrast, synaptogenesis that is "experience-dependent" maximizes the individual's adaptation to the environment. An example would be specific learning that occurs in response to unique features of the environment. Experience-dependent synaptogenesis is unique to the individual, whereas experience-expectant synaptogenesis is thought to apply to all members of a species (Greenough & Black, 1992).

Researchers Greenough and Black (1992) propose that "expectation" is characterized by an unpatterned, temporary overproduction of synapses in a relatively wide area during a sensitive period. The period of sensitivity depends upon the system in question. For instance, visual experience must occur early on for the visual system to develop as normal. Beyond the period of sensitivity, synapses that did not form connections (or formed abnormal ones) are lost. Expected experiences create patterns of neuronal activity that will be preserved, rather than retracted. The model assumes that synaptic contacts are transient and require confirmation via environmental stimuli to become targeted for preservation. Without such confirmation, they are retracted in keeping with a developmental schedule or as better established synapses compete for preservation. Research on humans (Huttenlocher, 1994) and monkeys (Rakic, Bourgeois, Eckenhoff, Zecevic, & Goldman-Rakic, 1986), suggests that synapses are widely overproduced early on, so that unnecessary connections can be subsequently eliminated. The overproduction seems to aid the nervous system's

preparation for the impact of a wide range of environmental stimuli by producing a quantity such that stimuli-related neural activity can choose a functionally appropriate pattern for further development (Greenough & Black).

These two mechanisms whereby synapses are formed, experience-expectant and experience-dependent synaptogenesis, involve windows of both opportunity and vulnerability. Experiences occurring appropriately, at the right time of development, aid the individual in such ways as learning. However, "inappropriate" experiences, such as abuse or trauma, can occur at a time when synapses are requiring confirmation. The results can be significant, as in damage to the hippocampus and HPA axis with consequent memory and emotional impairments (Greenough & Black, 1992).

Exposure to trauma can lead to the development of posttraumatic stress disorder (PTSD). PTSD was officially described as a psychiatric disorder in 1980 and included in the third edition of the Diagnostic and Statistical Manual (DSM-III) (American Psychiatric Association, 1980) to detail the psychological consequences of exposure to extremely traumatic events. Before 1980, psychiatric syndromes resulting from trauma exposure were thought to be transient and were named according to the precipitating event (e.g. the concentration camp syndrome, the rape trauma syndrome, combat neurosis). As similarities between these conditions were recognized, and the impact of trauma on mental health became apparent, a broad diagnosis was necessary to describe the like chronic symptoms of the disorder and distinguish it from adjustment disorders. (Golier & Yehuda, 1998).

Memory-related impairments are prominent among the PTSD diagnostic criteria. The DSM-IV (American Psychiatric Association, 1994) criteria for PTSD are broken down into "A," "B," "C," & "D" subcomponents. First, the diagnosis requires that the person has been exposed to a shocking event involving both of the following:

1. The person has experienced, viewed, or been confronted with an incident involving actual or threatened death or bodily harm, or a threat to the physical well-being of oneself or another's.
2. The person responded to this shocking event with feelings of extreme fear, helplessness, or horror.

The "A" criterion above includes both an objective component (the incident) and a subjective component (the person's interpretation of the incident and affective reaction).

Symptoms are broken down into those related to re-experiencing the incident(s) (B criteria), persistent avoidance of associated stimuli (C criteria), and elevations in arousal (D criteria). The B criteria are as follows (American Psychiatric Association, 1994):

- Recurrent, intrusive, distressing recollections of the trauma
- Recurrent, distressing dreams of the event – very true-to-life nightmares
- Sense of reliving the experience (flashbacks)
- Intense psychological distress at exposure to trauma reminders (internal or external)
- Psychological reactivity on exposure to trauma reminders

The diagnosis of PTSD requires the presence of at least one of these five B criteria symptoms.

The diagnosis of PTSD requires at least one of the first two and one of the remaining five C criteria symptoms, as follows (American Psychiatric Association, 1994):

- Efforts to avoid trauma-related thoughts or feelings
- Efforts to avoid trauma-related activities, situations, or people
- Psychogenic amnesia
- Diminished interest in activities
- Detachment from others
- Restricted range of affect or numbing
- Foreshortened future

These overlap a great deal with symptoms for major depression.

Two of the following D criteria (elevations in arousal) symptoms are required for a PTSD diagnosis (American Psychiatric Association, 1994):

- Sleep disturbance
- Irritability or difficulty regulating anger
- Difficulty concentrating
- Hypervigilance
- Exaggerated startle response

All of the symptoms must last more than a month and cause clinically significant impairment in functioning or distress. Some of the above symptoms overlap with those of generalized anxiety disorder (as well as depression), and the DSM-IV

categorizes PTSD as an anxiety disorder. In keeping with all anxiety disorders is the conflict between attending to threatening stimuli while attempting to avoid the elaboration of such stimuli, both cognitively and behaviorally. A hallmark of PTSD is a biphasic reliving and denial of an incident, with alternative intrusive and numbing effects. When avoidance efforts fail to reduce stress, numbing occurs in its stead.

One of the primary distinctions between a traumatic event of life-threatening proportions and another type of serious stressor is that removing the "stressor" often relieves or eliminates the negative effects of the stressor. In fact, physicians who believe stress is contributing to a patient's worsening condition will often prescribe a stress reduction or removal regimen. In contrast, PTSD describes the effects of trauma exposure that can continue decades after an incident has occurred, or can even span a lifetime. In PTSD, the patient has often created a distance between him/herself and the trauma, and yet the memory for a traumatic incident persists, with associated elevations in arousal as if the incident were recently taking place. For this reason, PTSD can be thought of as the body's failure to recover completely from the biological consequences of its stress response, since the events it is "re-experiencing" are not occurring at present in "real time" (Yehuda, 2000).

PTSD is but one of the psychiatric responses to trauma. For instance, following the eruption of Mt. St. Helens, increased incidents of new-onset depression and generalized anxiety disorder were reported in the community in addition to PTSD (Shore, Tatum, & Vollmer, 1986). Given the variety of

phenomenological responses to like events, it is not surprising that there is heterogeneity in psychiatric responses to traumatic events.

While only recently acknowledged, PTSD is one of the most prevalent psychiatric disorders, afflicting 8% of the United States population over the lifespan (Kessler, Sonnega, Bromet & Nelson, 1995). Exposure to trauma is even more frequent, with 50% of women and 60% of men having experienced a significant traumatic incident at least once (and often more than once) in their lifetimes. While PTSD and trauma and more widespread than initially assumed, PTSD only occurs in a fraction of the population exposed to trauma (Golier &Yehuda, 1998).

The assessment of incidents of a particular disease or condition in a specific population at a given time is referred to as prevalence (Last, 1983). Recently, the prevalence of PTSD has been examined in the general population via nationally representative samples (Resnick, Kilpatrick, & Dansky et al., 1993; Kessler et al., 1995; Breslau, Kessler, & Chilcoat, 1998) in addition to samples from specific geographical areas (Norris, 1992; Helzer, Robins, & McEvoy, 1987; & Davidson, Hughes, & Blazer, 1991). Studies have also addressed the conditional risk for the development of PTSD – the prevalence of PTSD among those exposed to trauma (Norris; Resnick et al; Kessler et al; Breslau et al., 1998; Breslau, Davis, & Peterson, 1997).

Recent studies of the adult population in general reveal lifetime prevalence of PTSD rates ranging from 1.0% (Helzer et al., 1987) to 9.2% (Breslau, Davis, Andreski, & Peterson, 1991). Studies limited to representative samples of women

reveal ranges from 12.3% (Resnick et al., 1993) to 13.8% (Breslau et al., 1997). These findings are consistent with other population surveys revealing that women seem more likely to develop PTSD (Helzer et al., 1987; Davidson et al., 1991), even though men report greater exposure to traumatic incidents (Norris, 1992; Kessler et al., 1995; Breslau et al, 1995; Breslau, 1998). Breslau, Kessler et al. (1998) reported gender as a significant risk factor in a study controlling for other sociodemographic factors, with women twice as likely as men to develop the disorder.

Norris (1992) examined the occurrence and impact of types of traumata in Southeastern United States. Norris found that 7.3% of participants with trauma exposure had experienced PTSD within the past year. The lowest rate of PTSD resulted from combat (2%) and the highest resulted from sexual assault (14%). PTSD resulted from exposure to violence, death, or accidents (7% - 11%) more frequently than from other environmental hazards (5% - 8%). Resnick et al. (1993) found a significantly higher rate of PTSD resulting from crime (25.8%) versus non-crime (9.4%) traumata. In addition, Resnick et al. (1993) found that women with histories of physical assault had the highest lifetime rates (38.5%) and present rates (17.8%) of PTSD. Women with histories of rape had PTSD lifetime and present rates of 32% and 12.4%, respectively. Similarly, Kessler et al. (1995) found that the prevalence rates for PTSD varied according to trauma-type. Rape was the most likely trauma-type to lead to PTSD in both men and women. Other trauma types likely to lead to PTSD were combat exposure, childhood abuse (physical, sexual, neglect), and threat by a weapon. Davidson et

al. (1991) found that participants with PTSD most frequently reported exposure to threat, seeing another hurt/killed, physical assault, accident, and combat. Breslau et al. (1991) reported similar findings, with the exception of rape which had a high risk rate of 80%. Interestingly, Breslau et al. (1991) found that respondents with PTSD were most likely to cite the immediate unexpected death of a loved one, experienced by 60% of participants, as the precipitating event for the onset of PTSD, with a PTSD risk of 14.3%.

In addition to gender and trauma-type risk factors for the development of PTSD, there are personality characteristics associated with the disorder. Helzer et al. (1987) found that PTSD is related to an array of other psychiatric disorders (e.g. obsessive-compulsive disorder, manic-depressive disorder, and dysthymia). Similarly, Davidson et al. (1991) reported a high prevalence of other disorders in respondents, including somatization disorder, schizophrenia/schizophreniform disorder, social phobia, and panic disorder. In addition, Helzer et al. found PTSD predictable via an assessment of behavioral problems emergent prior to age 15, such as stealing, vandalism, truancy and lying, with the rate of PTSD positively correlated to the number of behavioral problems exhibited. Researchers Breslau et al. (1991) found that neuroticism, as well as preexisting anxiety and depression, increased the likelihood of developing PTSD after exposure to trauma. Bromet, Sonnega, & Kessler (1998) found that a history of affective and/or anxiety disorders was predictive of PTSD onset after exposure to trauma for both males and females.

Family psychiatric history is another risk factor for the development of PTSD after exposure to trauma. Davidson et al. (1991) found that patients with PTSD were 2.8 times more likely to report psychiatric illness in their family members. Breslau et al. (1991) found that a family history of antisocial behavior increased the risk of PTSD (odds ratio [OR] = 2.05), as did a family history of anxiety disorder (odds ratio [OR] = 2.9). Bromet et al. (1998) reported similar findings in that parental mental disorder, in both men and women, was a risk factor for the development of PTSD (odds ratio [OR] = 1.9).

Research findings are inconsistent regarding ethnic risks for PTSD. Davidson et al. (1991) reported that nonwhites were at a higher risk for PTSD, although the results did not achieve statistical significance. The 1996 Detroit Area Survey of Trauma (Breslau et al., 1998) revealed nonsignificant differences once adjustments were made to control for other variables. Once inner city residence was removed from the model, the nonwhites versus whites adjusted odds ratio for PTSD became 1.8.

Several studies have examined the impact of trauma exposure and PTSD on participants' health and use of medical and psychiatric services and subsequent cost. The research completed to date has focused mainly on adult men and women who are survivors of combat and sexual assault, reducing its generalizability. Boscarino (1997) examined the medical histories of Vietnam veterans 20 years post-combat (n = 1399), controlling for selection bias, socioeconomic status, hypochondriasis, and behavioral risk factors. Those who developed PTSD had significantly greater lifetime prevalence of circulatory,

digestive, musculoskeletal, nervous system, respiratory, and nonsexually transmitted infectious diseases. Wolfe, Schnurr, & Brown (1994) reported that Veterans with PTSD had a higher risk of dermatologic (OR = 3.88, $p < .001$), pain (OR = 3.32, $p < .001$), gastrointestinal (OR = 3.23, $p < .01$), opththalmologic (OR = 3.09, $p < .01$), endocrinologic (OR = 3.09, $p < .01$), gynecologic (OR = 2.38, $p < .01$), and cardiovascular problems (OR = 2.02, $p < .05$) than non-PTSD veterans.

Waigandt, Wallace, & Phelps (1990) examined the long-term physical health complaints of 51 rape survivors in comparison to 51 age-matched controls. Results reveal statistically significant increases in the number of symptoms of rape versus nonrape participants ($t = 5.51$, $p < .01$), in the number of negative health behaviors reported ($t = 5.05$, $p < .01$), and in female reproductive physiological symptoms reported ($t = 6.21$, $p < .01$). Similarly, Golding (1994) found that women who were sexually assaulted reported experiencing poorer health, greater functional limitations, greater medically explained and unexplained somatic symptoms, and more chronic diseases. Golding found significant odds ratios for burning sensation in sexual organs (OR = 3.23, paralysis (OR = 3.10), pain with urination (OR = 2.76), and diarrhea (OR = 2.74). Sexual assault survivors had a significantly greater prevalence of diabetes (OR = 2.35, [$p < .01$]) and physical disability (OR = 1.96 [$p < .05$]). Koss, Koss, & Woodruff (1990) examined the effect of criminal victimization on women's health and medical requirements from an HMO sample (n = 413; mean age = 36.4 years, range, 19-69

years). Severely victimized women reported greater distress and poorer health ($p < .01$).

Felitti, Anda, & Nordemberg (1998) examined the relationship between exposure to trauma during childhood and engagement in behaviors in adulthood that are considered high-risk (leading causes of death). Childhood traumas such as psychological, physical and sexual abuse; violence directed at mother, mental illness, criminal activity, and drug abuse among family members were those considered. Researchers found a statistically significant relationship between the number of types of traumas to which participants were exposed and each of the health risk behaviors examined: smoking, severe obesity, physical inactivity, depressed mood, and attempts at suicide ($p < .001$). In addition, researchers reported a dose-response relationship ($P < .05$) between the number of trauma exposures and the diagnosis of the following: ischemic heart disease, cancer, chronic bronchitis, hepatitis, skeletal fractures, and poor self-reports of health.

At this time, no official information exists on the costs of traumatic experience and PTSD. However, Golding, Stein, & Siegel (1988) estimated the relationship between sexual assault and reliance upon mental health and medical services, using data from the Los Angeles ECA study. Researchers found that respondents were twice as likely to have utilized mental health care within the previous 6 months if they were survivors of sexual assault than if not (17.8% vs. 9.0%, $p < .01$). Sexual assault survivors were also significantly more likely to report a physical health visit within the previous 6 months than non-assault respondents (60% vs. 44%, $p < .01$). Similarly, Waigandt et al. (1990) found that

rape survivors (51) reported a significantly greater number of visits to physicians than non-assault controls ($p < .03$).

Greenberg, Sisitsky, & Kessler (1999) calculated the societal cost related to anxiety disorders utilizing NCS data. Using multivariate regression, adjusting for demographics and psychiatric comorbidity, they estimated both direct costs (psychiatric services costs such as counseling and hospitilization, nonpsychiatric medical costs such as emergency room or primary care visits, and prescriptions) and indirect costs (absenteeism and reductions in work-product due to anxiety). Researchers estimated a $42.3 billion dollar loss in 1990 alone, equating to $1542 per sufferer. Researchers estimated that nonpsychiatric medical treatment accounted for 54% of the total, whereas psychiatric treatment accounted for only 31%, suggesting that misdiagnosis of anxiety disorders contributes to the societal cost. Researchers noted that participants with PTSD and panic disorder had the greatest rates of service use.

The prevention of trauma-related hippocampal atrophy is of the utmost importance. In our society, violence is a frequent occurrence (Cicchetti & Toth, 1995; Lynch & Cicchetti, 1998; McCauley et al., 1997). This prevalence of violence poses as a societal health hazard (Barnett, Manly, & Cicchetti, 1993). Glucocorticoid mediated toxicity to the hippocampus could lead to learning and memory difficulties affecting a child's educational growth trajectory. Compounding this problem, PTSD symptoms such as hyperarousal and difficulty concentrating, trusing and socializing with others can prohibit a child's social development (Cicchetti & Toth, 1995). Prevention of trauma is the best way to

avoid the aforementioned developmental problems (Toth & Cicchetti, 1993). Societal awareness of the prevalence and seriousness of the problem is crucial, since early intervention is critical to the prevention of developmental problems (Cicchetti, Toth, & Hennessy, 1993). However, the development of appropriate treatment strategies for those who have been exposed to trauma requires a better understanding of the effects of trauma, including any impact on the memory functioning of the victims. Some research has examined the impact of trauma on memory functioning in adults. However, there is a paucity of research on the impact of trauma, such as domestic violence trauma, on children's memory functioning. For this reason, it is critical to examine the memory functioning in children who have been exposed to domestic violence trauma. Research is needed to see if the patterns reported by researchers for adult populations exposed to trauma can be seen in children as well.

2

LITERATURE REVIEW

Over ten years ago, researchers found that the increased levels of glucocorticoids associated with the stress response damage the hippocampus, an area of the brain involved in memory as well as learning (McEwen et al., 1992; Sapolsky, 1996). Researchers found that monkeys exposed to extreme stress had damage to their CA3 subfield of the hippocampus (Uno, Tarara, Else, Suleman, and Sapolsky, 1989). Glucocorticoids implanted directly into the hippocampus of monkeys produced a similar effect.

Researchers found that glucocorticoids cause reductions in the branching of dendrites (Watanabe, Gould, & McEwen, 1992; Wooley, Gould, & McEwen, 1990) and neuronal loss (Uno et al., 1990), specific to the hippocampal area (Packan & Sapolsky, 1990). Glucocorticoids disrupt cellular metabolism, increasing the vulnerability of hippocampal neurons to insults such as endogenously released excitatory amino acids (Armanini, Hutchins, Stein, & Sapolsky, 1990; Sapolsky et al., 1986; Sapolsky & Pulsinelli, 1985, Virgin et al., 1991). In addition, they enhance the accumulation of extracellular glutamate (Stein-Behrens, Lin, & Sapolsky, 1994). Hippocampal atrophy as a consequence of stress is correlated with deficits in memory performance (Luine, Villages, Martinex, & McEwen, 1994) with the magnitude of deficits in the learning of new maze routes correlated with the number of CA3 hippocampal cells damaged (Arbel, Kadar, Silberman, & Levy, 1994).

Research on humans with epilepsy undergoing temporal lobectomy revealed that the left hippocampus plays a significant role in verbal memory. Verbal memory deficits are more severe when hippocampal lesions are bilateral rather than unilateral, revealing that both hippocampi are involved in verbal memory (Bremner & Narayan, 1998). Researchers have found that Vietnam veterans with combat-related PTSD matched to healthy controls for age, race, years of alcohol abuse, years of education, handedness and SES (socioeconomic status) had significantly greater deficits in verbal memory based on their performance on the Wechsler Memory Scale (WMS) – Logical Component for both immediate and delayed recall (Russell, 1978).

Similar verbal memory deficits have been reported for PTSD subjects using the Selective Reminding Test – Verbal Component (Hannay & Levin, 1985), as well as other measures of verbal declarative memory function (Uddo, Vasterling, Brailey, & Sutker, 1993; Yehuda et al., 1995a). Research on survivors of childhood abuse reveals similar verbal declarative memory function deficits (Bremner, Randall et al., 1995).

Researchers testing the hypothesis that traumatic stress results in hippocampal damage found an 8% decrease in MRI-based measurement of right hippocampal volume in subjects with Vietnam combat-related PTSD as compared to matched controls. These decreases in right hippocampal volume were associated with short-term memory deficits on the WMS-Logical, percent retention subcomponent ($r = 0.64$; $p < 0.05$). In this study, no volume differences

were reported for the bilateral left temporal lobe (minus hippocampus), caudate, or amygdala (Bremner et al., 1995a).

Researchers have compared hippocampal volume in patients with Vietnam combat-related PTSD, Vietnam veterans without PTSD and healthy, non-veterans. Gurvitz et al. (1996) found a statistically significant bilateral decrease in hippocampal volume (26%) in subjects with PTSD. Gurvitz et al. also examined left and right hippocampal volume separately and found statistically significant decreases in left and right hippocampal volumes for subjects with PTSD, after adjustments were made for years of alcohol abuse via analysis of covariance. No differences were reported for ventricular, amygdala, or whole brain volume between groups. Level of combat exposure as reported on the Combat Exposure Scale had a significant correlation with hippocampal volume and visual delayed recall errors (Gurvitz et al.). Studies of survivors of childhood abuse report similar findings (Bremner et al., 1997b; Stein, Koverola, Hanna, Torchia, & McClarty, 1997).

A function of the hippocampus is to inhibit the release of CRF from the hypothalamus and chronically stressed animals have elevated levels of CRF (Feldman & Conforti, 1980; Herman, Schafer, & Young, 1989). Vietnam veterans with PTSD have elevated levels of CRF in their cerebrospinal fluid based on lumbar puncture as compared to age and sex matched controls (Bremner et al., 1997a).

Lowered cortisol levels are associated with chronic PTSD (Mason, Giller, Kosten, Ostroff, & Podd, 1986; Yehuda, Southwick, Nussbaum, Giller, & Mason,

1991). Researchers found that women with low cortisol levels 17-151 days after sexual assault have higher exposures to previous trauma and increased risk for the onset of PTSD (Resnick, Yehuda, Pitman, & Foy, 1995). Within 50 hours of sexual assault trauma, however, low cortisol levels were not found to correlate with ensuing development of PTSD (Yehuda, Resnick, Schmeidler, Yang, & Pitman, 1998).

These findings pose a question as to how elevated cortisol can be the cause of hippocampal atrophy in PTSD. Perhaps increases in cortisol at the time of trauma do persisting damage to hippocampal neurons, reducing hippocampal volume as revealed with MRI (Bremner, Krystal, Southwick, & Charney, 1995; Bremner, Krystal, Charney, & Southwick, 1996; Bremner, Vermetten, Southwick, Krystal, & Charney, 1998). If so, decreases in cortisol would characterize the chronic stages of the disorder due to alterations in cortisol regulation. Research findings above suggest that acute trauma is associated with CRF/HPA system hyperactivity, while chronic PTSD might encourage long-term dysregulation producing an altered HPA/cortisol system. Another possibility is that the sensitivity of the glucocorticoid receptors in the hippocampus to cortisol could be the crucial variable in one's susceptibility to hippocampal atrophy from stress. Patients with high levels of cortisol from Cushing's Disease have been found to have hippocampal atrophy, as well as deficits in cognitive memory (Starkman, Gebarski, Berent, & Schteingart, 1992). Another interpretation, however, is that genetically prescribed small hippocampal volume could be a risk factor for the onset of PTSD.

One might wonder how a hormonal system directed toward survival could become toxic to the brain in particular circumstances. It is possible that in an acute-stress situation, releasing large quantities of glucocorticoids might be more important to the survival of the organism than hippocampus and memory function preservation. From a Darwinian perspective, loss of memory function might not become problematic until later in life, long after the passing on of one's genes during reproduction has been accomplished. In this interpretation, long-term memory function is sacrificed for the sake of immediate survival and the reproduction of the species.

Researchers have examined the effects of stress at different stages of development. Research with animals supports the notion that early stress can impact an organism throughout its life cycle. Prenatal exposure to light and noise (Fride, Dan, Feldon, Halevy, & Weinstock, 1986), early deprivation of maternal support (Levine, Weiner, & Coe, 1993; Stanton, Gutierrez, & Levine, 1988) and early exposure to excessive manipulation (Levine, 1962) caused heightened glucocorticoid response to future stressors. Early exposure to maternal deprivation stress is associated with heightened adreno cortical responsivity as exemplified by greater glucocorticoid responsivity to ACTH challenge (Stanton, Gutierrez, & Levine, 1988). Research with male rats reveals that stressors in the postnatal period can modify the CRF mRNA, median eminence CRF breakdown, and the release of CRF (Plotsky & Meaney, 1993), as well as the release of ACTH (Ladd, Owens, & Nemeroff, 1996). In the maternal deprivation condition, male rats had fewer glucocorticoid receptors in the hippocampus, hypothalamus,

and frontal cortex during assessments of dexamethasone binding. Microdialysis revealed that the male rats also had heightened norepinephrine levels in the hypothalamus paraventricular nucleus (PVN). In varying the maternal foraging requirements of nonhuman primates, researchers found that behavioral disruptions were exhibited for years in that the subjects were more subordinate and meek, as well as less social. Monkeys raised in this condition also had higher corticotropin releasing factor (CRF) levels in the cerebral spinal fluid in adulthood (Coplan et al., 1996). Hence, it seems early stressors can permanently affect the HPA axis.

Positive life experiences during early critical periods in an organism's development might serve as mediators for the damaging effects of stress. Researchers found that with rat pups, daily handling within the first month of birth followed by return of the rat pups to their mothers increased the Type II glucocorticoid receptor binding throughout the life cycle of the rats. The rats developed heightened feedback responses to glucocorticoids and reduced glucocorticoid-associated hippocampal atrophy as adults (Meaney, Aitken, van Berkel, Bhatnagar, & Sapolsky, 1988; Meaney, Aitken, Sharma, & Sarrieau, 1989). The mother's continued licking of her handled pups upon their return seems to assist the pups in building a type of tolerance to the stressful handling condition (Liu et al., 1997). Perhaps in the early postnatal stage, pertinent neural systems are programmed at that time to handle stress in a particular manner.

Since animals display glucocorticoid-mediated hippocampal and memory function damage from exposure to stress, researchers have pondered whether humans display the same effects. Studies of adults with PTSD who were exposed

to childhood physical and/or sexual abuse revealed that the subjects displayed deficits in short-term memory on the Wechsler Memory Scale (WMS) – Logical Component for verbal memory (Wechsler, 1997), deficits in recall, both immediate and delayed, and deficits on their performance scores on the Verbal Selective Reminding Test (Hannay & Levin, 1985), as compared to controls matched on age, sex, race, years of education, and years of alcohol abuse ($p<0.01$). Short-term memory deficits were significantly correlated with the composite severity score of the Early Trauma Inventory (Bremner, Vermetten, & Mazure, 1998) ($r = - 0.48$; $p < .05$). No differences were found in IQ (WAIS-R) (Wechsler, 1981) or visual memory (WMS-Figural Component) (Wechsler) in abuse survivors versus controls (Bremner, Randall et al., 1995).

Researchers found that male and female adults with histories of child abuse (physical or sexual) resulting in long-term PTSD had a statistically significant left hippocampal reduction of volume of 12% ($p < 0.05$) as compared to controls matched for age, sex, handedness, race, years of education, and years of alcohol abuse. A reduction of right hippocampal volume of 3-8% was found as well, but this reduction was not statistically significant. When researchers entered age, education, and alcohol abuse into multivariate analyses with stepwise linear regression, they found that PTSD was significantly related to decreases in hippocampal volume. The same relationship, however, was not found for the temporal lobe, caudate, or amygdala (Bremner et al., 1997b).

Researchers compared left hippocampal volume of 21 sexually abused women to that of 21 non-abused controls. They found a 5% reduction of volume

for the abuse-history condition, with hippocampal atrophy correlated to degree of dissociation in the abused adults, most of whom had PTSD (Stein et al., 1997).

A number of studies suggest a relationship between stress and depression (reviewed in Mazure, 1994). Hypercortisolemia has been reported to occur with episodes of depression. Researchers have also reported MRI-based hippocampal atrophy in subjects recovering from depression (Sheline, Wang, Gado, Csernansky, and Vannier, 1996). Researchers Bremner and Narayan (1998) controlled for differences in entire brain volume in subjects treated for depression as compared to controls.

Stress seems to impact memory and the hippocampus differently, according to a subject's stage of development. Subjects with PTSD due to later-life trauma, such as Vietnam Veterans, tend to have greater right hippocampal atrophy. Subjects who have PTSD due to early-life trauma tend to have more atrophy to the left hippocampus. For subjects with early-life trauma, there is a correlation between deficits in memory function and degree of abuse not found for subjects who experienced later-life trauma. (Bremner, Randall et al., 1995). Some researchers reported a relationship between the arithmetic subscale of the IQ Test and abuse measures (Lewis, Shanok, Pincus, & Glaser, 1979). It has been suggested that a genetically influenced low IQ could pose as a risk factor for the onset of PTSD (McNally & Shin, 1995). However, it seems likely that the stress of trauma at a young age could have a greater impact on the developing brain.

These findings have critical implications for public healthy policy, given the amount of violence to which we are exposed. Research findings suggest that

16% of women report that they experienced childhood sexual abuse occurring before age 18 (McCauley at al., 1997). Another study found that 9% of Detroit inner city youngsters suffered from PTSD after witnessing shootings, child abuse, and/or domestic violence (Breslau et al., 1991). It follows that the number of youngsters in our society suffering from PTSD could be in the hundreds of thousands. The abuse and ill-treatment of our youngsters might affect their academic performance, with long-term repercussions for our society (Cichetti et al., 1993). Researchers Saigh, Mroweh, and Bremner (1997) found significantly poorer academic performance in Lebanon youths with civil war-exposure PTSD (n = 12) according to their performance on the Metropolitan Academic Achievement Test (Prescott, Balow, Hogan, & Farr, 1986) in relation to both nonstressed (15) and stressed-without-PTSD (n = 16) Lebanese youths' performance on the same measure. Deficits were reported for the subtests of vocabulary, reading, spelling, language, mathematics and science. If our youngsters suffer from memory dysfunction related to the hippocampal toxicity associated with stress, the implications for public health policy are profound. Difficulties in learning and school performance are likely, with life-long consequences.

Changes in memory in clients who are survivors of child abuse are a current controversy. Dissociation and memory fragmentation are common with this population. Abuse survivors often remember only a portion of an incident or certain characteristics of an incident. For example, an abuse survivor might remember a shivering sensation if he/she had been locked in a shed during winter.

Later, the survivor might connect the shivering sensation with the location of the shed, and might then remember the details of an incident. Such examples of gradual memory recovery have been in great dispute for almost a decade (Bremner & Marmar, 1998).

Some claim that incidences of graduate or "delayed" recall result from therapists who "plant" the ideas in their clients' minds through improper reinforcement or careless use of certain techniques such as hypnosis. Some therapists have allegedly been so insistent that their clients were abused that these clients began to have difficulty distinguishing the suggestions from reality. Mental health professionals differ greatly in their degree of skill, training, standards and professional experience. While in the past, some therapists might have been too eager to dredge up clients' recollections of traumatic experiences, the current trend is in the opposite direction. Many therapists are careful not to suggest to a client that he/she might have been abused. For those clients with a history of abuse who need to work through their feelings, this trend can be problematic as well.

Research on the hippocampus is pertinent to the debate on the "delayed recall" of child abuse. A key function of the hippocampus is the integration of various aspects of memory for the sake of recall. Research suggests that at the time of recall, the hippocampus locates an incident according to time, place and context. It is possible that hippocampal atrophy associated with trauma can fragment or alter memories for the incidents (Bremner et al., 1996). For the example of the abuse survivor who was locked in a shed in winter, the client

might remember the tingly sensation of the cold, but might not have a visual memory of the shed or an affective memory of terror or anger. It is possible that therapy facilitates the linking of these aspects of the experience to form a complete memory for the incident. In this scenario, memory fragmentation and delayed recall would occur only in the presence of PTSD psychopathology as a consequence of hippocampal atrophy and reduced function. Clients exposed to traumatic events who do not develop PTSD would not experience such fragmentation of memory or delayed recall, since there is no reduction in hippocampal volume or function.

There might be an interaction between stress and aging serving to further hippocampal atrophy and decreased memory function. Researchers Sapolsky, Krey, and McEwen (1985) presented a "glucocorticoid cascade" model for memory dysfunction due to the aging process. In their research on rodents, they found progressively higher glucocorticoid peripheral levels with increases in age. These higher levels were associated with greater hippocampal atrophy and memory dysfunction, as well as further increases in glucocorticoid peripheral levels resulting from lowered inhibition of glucocorticoid release in the hippocampus. In addition, the rats exhibited a delayed return to baseline of glucocorticoid levels after stress exposure, perhaps related to a down regulation of hippocampal glucocorticoid receptors with age (Sapolsky, Krey, & McEwen, 1983a, 1983b). There was a loss of receptor plasticity with age as well (Eldridge, Brodish, & Kute, 1989; Eldridge, Fleenor, & Kerr, 1989).

There is a paucity of research on humans related to glucocorticoids, memory, the hippocampus and aging. The studies that exist report mixed findings (Stein-Behrens & Sapolsky, 1992; Urban, 1992). However, there is some support for elevations in cortisol levels in humans with age (Armanini et al., 1993; Lupien et al., 1994; Swaab et al., 1994; Van Cauter, Leproult, & Kupfer, 1996). There is also some support for decreased feedback sensitivity to cortisol in humans with age (O'Brien, Schweitzer, Ames, Tuckwell, & Mastwyk, 1994). The elderly had fewer glucocorticoid receptors as measured by leukocytes (Armanini et al., 1992, 1993) and changes in cortisol responsiveness in times of stress (Gotthardt et al., 1995; Raskins et al., 1995; Seeman et al., 1995). Higher cortisol levels (Lupien et al.) and reduced feedback-sensitivity/inhibition to cortisol (O'Brien et al.) were associated with increased memory dysfunction. Acute glucocorticoid administration produced greater memory dysfunction in younger versus older participants, suggesting that the elderly have less glucocorticoid binding in the hippocampus (Newcomer, Craft, Hershey, Askins, & Bardgett, 1994). While some studies found decreased hippocampal volume in the elderly as measured by MRI (Convit et al., 1995), others did not (Sullivan, Marsh, Mathalon, Lim & Pfefferbaum, 1995). Researchers have found structural changes in the hippocampi of participants with age-related memory impairment (Soininen et al., 1994; Parnetti et al., 1996) and these structural changes were correlated to the impairment (Soininen et al.)

Researchers Seeman et al. (1995) examined the connection between stress and cortisol in elderly participants and found the elderly exhibit elevations in

ACTH and cortisol with stress exposure. Those participants with low self-esteem had 6 times the elevation levels of those with high self-esteem when exposed to a driving stressor.

Little research has addressed the relationship between age and PTSD symptomotology. However, a study by Kato, Asukai, Miyake, Minakawa, and Nishiyama (1996) examined the level of short-term PTSD symptoms in Hanshin-Awaji earthquake evacuees. Participants under age 60 (n = 50) and over age 60 (n = 73) were interviewed at 3 weeks and at 8 weeks post-evacuation. At the three-week interval, all participants from each age group suffered from hypersensitivity, irritability, sleep disturbances, and depression. At 8 weeks, the elderly group reported significant reductions in 8 of 10 symptoms, whereas the younger group reported no reductions in symptoms.

Similarly, researchers examined 179 participants after the 1988 earthquake at the 1.5-year interval (Goenjian et al., 1994). No significant differences were found between younger and older adults on the PTSD reaction index. However, symptomotology profiles for the older adults differed significantly from those of the younger adults in that they had higher arousal and fewer intrusive symptoms.

There is a paucity of biological research between age and PTSD. However, researchers Yehuda et al. (1995b) reported significantly lowered cortisol levels in elderly Holocaust survivors as measured by 24-hour urine samples, while Lemieux and Coe (1995) reported significantly raised cortisol levels in younger individuals with abuse-related PTSD. Perhaps PTSD in

younger participants is associated with hypercortisolemia, whereas in older participants there is hypocortisolemia, perhaps related to HPA axis dysregulation.

It is possible that stress and aging interact to create hippocampal susceptibility to glucocorticoid-mediated toxicity. Researchers Levy, Dachir, Arbel, & Kadar (1994) administered slow-release corticosterone to produce simulated stress in young (3-month-old) and middle- aged (12-month-old) rats. The middle-aged rats exhibited significantly greater cognitive deficits. An examination of the HPA axis is pertinent to research with the age and stress relationship as it its focus. In animal research, chronic stress is linked to glucocorticoid-receptor down regulation, as well as impaired return to baseline glucocorticoid levels after stress reintroduction (Ladd, Owens, & Nemeroff, 1996) and aging is associated with the same (Sapolsky et al., 1983a, 1983b). However, in clinical research with PTSD participants, an examination of peripheral lymphcytes reveals an increase in glucocorticoid receptors. Dexamethasone might be the key factor in this discrepancy, for researchers Newcomer et al. (1994) have found dexamethasone-related memory dysfunction in normal young participants, but not in normal elderly participants with a loss of hippocampal glucocorticoid receptors due to the ordinary aging process. More research in needed on the relationship between memory dysfunction and dexamethasone.

Research on animals suggests that various agents might be useful for treating or preventing glucocorticoid mediated toxicity of the hippocampus and memory dysfunction. Watanabe, Gould, Cameron, Daniels, and McEwen (1992) found phenytoin (Dilantin) to reverse atrophy of the hippocampus due to stress,

perhaps by excitatory amino acid-related neurotoxicity modulation. The agents tianeptine and dihydroepiandosterone (DHEA) have been found to do the same (Watanabe, Gould, Daniels, Cameron, & McEwan, 1992). Researchers found hippocampal neurons unique among brain-based neurons in their ability to self-regenerate (Gould, Tanapat, McEwen, Flugge, & Fuchs, 1998). It is not known whether human hippocampal atrophy is reversible, but researchers have found that cognitive therapy reverses memory impairments in Lebanese youths with PTSD (Saigh, 1988). Research on psychotherapy in treating child abuse survivors reveals that it is productive in mediating the effects of abuse, perhaps through its effects on the brain (Cicchetti & Toth, 1995; Toth & Cicchetti, 1993).

3

PARTICIPANTS, PROCEDURE, MATERIALS AND METHOD, DESIGN, MEASURES, DATA ANALYSIS

PARTICIPANTS

The participants were children (n=22) and their mothers (n=22) receiving shelter services and/or counseling at a New Jersey battered women's shelter. The mothers signed informed consent documents allowing their children, ages 3 to 11, to participate in a therapeutic program called the PALS project, as well as PALS related research.

PROCEDURE

Upon entering the study, the mothers completed the Child Behavior Checklist (CBCL) (Achenbach, 1991), as well as various other standardized assessments for use in the PALS project and research. The first 22 consenting children whose mothers also gave written and verbal consent to the researcher and who fell into the appropriate age range for the Children's Memory Scale (CMS) (Cohen, 1997), an age range of 5 to 16, were given the CMS. CMS scores were examined in conjunction with scores for those same children (n=22), as reported by their mothers, on the Child Behavioral Checklist (CBCL). I, Deanna Cosgrove, was the sole administrator of the CMS. However, there were several graduate students, including this researcher, trained by Dr. Linda Jeffrey during group training sessions to administer the CBCL, who were involved in the administration of the CBCL.

MATERIALS AND METHOD

The researcher practiced administration of the CMS to friends' children in the appropriate age range for the CMS (ages 5 to 16). A stopwatch was used to time participants' completion of the subtests of the CMS. The CMS was administered by paper and pencil in one location, at a quiet table in a sectioned off portion of the shelter. Mothers waited in an adjacent area out of sight and sound from their children while the CMS was administered. A calendar was used to schedule appointments for the CMS and each child was tested separately. The researcher was unable to test each child at the same time of day, due to scheduling difficulties. However, each child was tested while the room was well lit during times of the day the mothers felt were appropriate for their children. Each child was tested as quickly, upon admission to the PALS Project, as an appointment for testing and actual administration could take place.

DESIGN

This is a descriptive study of the memory functioning and behavioral correlates of children residing at a shelter for battered women in New Jersey.

MEASURES

CHILDREN'S MEMORY SCALE (CMS)

The Children's Memory Scale (CMS) (Cohen, 1997) is a comprehensive assessment instrument created to evaluate learning and memory functioning in persons aged 5 to 16. The CMS requires individual administration and can be included in psychoeducational, psychological, and neuropsychological evaluations, among others. The CMS was designed to assist school psychologists,

child neuropsychologists, and clinical psychologists as a standardized instrument for the evaluation of learning and memory processes in persons within the aforementioned age range.

The CMS (Cohen, 1997) assesses functioning within the domains of verbal learning (auditory), visual/non-verbal learning, and attention/concentration. Each domain is evaluated via two core subtests, as well as one supplemental subtest. The core subtests can be administered in a total of 30-35 minutes, while the supplemental battery requires an additional 10-15 minutes for administration. Each subtest within the verbal and non-verbal domains contains both an immediate and a delayed memory component. The timeframe between the immediate and delayed memory components of the subtests is around 30 minutes. A summary of each subtest is provided in Tables 1 and 2 in Appendix A of this thesis. The General Memory Index is an indicator of global memory functioning and incorporates the immediate and delayed memory indices from the verbal and visual domains.

The CMS (Cohen, 1997) allows clinicians to examine the following constructs:

- Immediate recall
- Delayed recall
- Verbal versus visual recall
- Encoding versus retrieval success
- Free recall as opposed to recognition
- Literal or nearly literal versus thematic recollection

- Attention/concentration
- Memory in conjunction with ability
- Correlations of memory with academic achievement, language ability and executive processes

CMS STANDARDIZATION

The CMS (Cohen, 1997) was standardized on 1,000 children (500 males and 500 females), ages 5 through 16. Age groups examined were 5, 6, 7, 8, 9, 10, 11, 12, 13-14, and 15-16. Each age group was comprised of 100 participants with equal numbers of males and females. Each age group consisted of the same proportion of Whites, African Americans, Hispanics, and other race or ethnic groups as reported for U.S. children aged 5- 16 by the U.S. Bureau of the Census (1995). The U.S. was divided into four regions – the Northeast, North Central, South, and West, and children were included in the sample in accordance with the proportion living in each region. In addition, the sample was stratified in accordance with parental education levels (8^{th} grade or less, 9^{th} through 11^{th}, high school or equivalency, 1-3 years of college or technical training, 4 or more years of college).

CMS EXCLUSION CRITERIA

CMS (Cohen, 1997) exclusion criteria are as follows:

- Below grade level
- Repeated a grade
- Referred for special education or remedial services
- Diagnosed with a neurological disorder

- Sustained an injury placing the individual at risk for memory impairment

CMS RELIABILITY AND VALIDITY

The split-half method was used to analyze the internal consistency of the CMS (Cohen, 1997). Tables 3 and 4 in Appendix A display the reliability coefficients for core and supplemental subtests. Tables 5, 6, and 7 in Appendix A present the test-retest stability coefficients for the CMS index scores, including means and standard deviations for both testing sessions, and uncorrected and corrected stability coefficients. The data reveal that the CMS performs with adequate stability across time for the various age-bands. Tables 8, 9, 10, 11, 12, and 13 in Appendix A depict the decision consistency stability coefficients for the classification cut points (impaired, borderline, low average., average., high average, etc.) as relatively stable. Inter-rater reliability is very high for the CMS; the intra-class correlations are presented in Tables 14 and 15 in Appendix A. The standard errors of measurement for the core subtests, indexes and supplemental subtest scores are depicted in Tables 16, 17, 18 and 19 in Appendix A.

The manual of the CMS (Cohen, 1997) claims that the CMS has undergone various reviews and revisions by experts to ensure adequate content validity. Tables 20 and 21 in Appendix A depict the Pearson correlation coefficients for CMS subtests for the age bands, revealing a moderate to high correlation between immediate and delayed recall for each subtest. Subtests within domains correlate in the low to moderate range, with the exception of Faces and Dot Locations. Correlations between subtests across domains are usually lower than between

subtests of the same domain. The General Memory Index correlates moderately to highly with other memory indexes.

CMS CORRELATIONS WITH DAS

DAS (Elliot, 1990) is an instrument involving individual administration designed to assess general intellectual ability for persons aged 2-18. The CMS General Memory and Learning indices (Cohen, 1997) correlate highly with the spatial abilities scale of the DAS, as depicted in Table 22 in Appendix A.

CMS CORRELATIONS WITH OLSAT

The OLSAT (Otis, 1989) is a group-administered instrument for the assessment of academic ability. The Attention-Concentration Index of the CMS (Cohen, 1997) correlates highly with all OLSAT ability measures, as depicted in Table 23 in Appendix A. As shown, the General Memory Index of the CMS correlates moderately to highly with OLSAT scores.

CMS CORRELATIONS WITH WIAT

The WIAT (The Psychological Corporation, 1992) is a standardized instrument of academic achievement, individually administered, focusing of the areas of reading, writing, math, and language. Table 24 in Appendix A depicts the Pearson correlation coefficients and mean performance data on the WIAT and CMS indices (Cohen, 1997), revealing a moderate positive correlation between many CMS measures and those for the WIAT. While visual memory measures correlated in the low range, verbal memory measures displayed the highest positive correlation to academic achievement. The Attention/Concentration Index correlated highly with academic achievement.

CMS CORRELATIONS WITH WCST

The WCST (Grant & Berg, 1993) is a neuropsychological instrument for the assessment children and adults in terms of problem solving, cognitive flexibility and the ability to use feedback to change problem-solving behavior. Table 25 in Appendix A depicts Pearson correlation coefficients and mean performance data on the two measures, WCST and CMS (Cohen, 1997). The data reveal low to moderate positive correlations between the CMS indices and the WCST scores. The General Memory and verbal memory indices correlate in the moderate range with WCST data. The Attention-Concentration Index of the CMS is highly correlated with problem solving. Visual memory indices of the CMS are weakly correlated with the WCST data.

CMS CORRELATIONS WITH CCT

The CCT (Boll, 1993) is a neuropsychological instrument for the assessment of children's and adolescent's abstract reasoning, memory, cognitive flexibility, and the ability to utilize feedback. It contains a direct assessment of memory. Table 26 in Appendix A depicts the Pearson correlation coefficients and mean performance for the CMS (Cohen, 1997) and CCT scores, revealing non-significant correlations between the CMS and Level 1 CCT performance, but moderate, positive correlations between General Memory, Verbal Memory and Learning indices.

CMS CORRELATIONS WITH CELF-3

Language processes have been classified into two related, but functionally distinct domains: those involved in understanding or receiving language, and

those involved in production or expression (Semel, Wiig, et al., 1995), and the CELF-3 was designed to measure these two domains (Semel, Wiig, et al., 1995). Table 27 in Appendix A depicts the Pearson correlation coefficients and mean performance data on the two measures, CELF-3 and CMS (Cohen, 1997), revealing low to moderate correlations. Most of the correlations are nonsignificant, with the exception of the CMS Attention/Concentration Index and all CELF-3 scores. General Memory and Verbal Immediate indices of the CMS correlate with CELF-3 receptive and total language scores, but do not correlate with those for expressive language. The CMS Learning Index correlates with receptive CELF-3 scores, but does not correlate with those for expressive or total language.

CMS CORRELATIONS WITH WMS-III

The WMS-III (Wechsler, 1997) is a classic adult memory scale with additional norms for adolescents aged 16-17. Table 28 in Appendix A depicts Pearson correlation coefficients and mean performance data for CMS (Cohen, 1997) and WMS-III indices, revealing high positive correlations between the two instruments. As shown, the highest correlations are between measures within the same domain. The data reveal a strong convergent validity between the two measures.

CMS CORRELATIONS WITH WRAML

The WRAML (Sheslow & Adams, 1990) is an instrument, individually administered, for the assessment of memory and learning in children and adolescents. Table 29 in Appendix A depicts Pearson correlation coefficients and

mean performance data for the CMS (Cohen, 1997) and WRAML, revealing low to high positive correlations between the measures. The highest correlations were found between measures of the same domain, with the exception of a high correlation between the CMS Attention/Concentration Index and the Verbal Memory Index of the WRAML, as well as a low correlation between the CMS Visual Immediate and the WRAML Visual Memory indices.

CMS CORRELATIONS WITH CVLT-C

The CVLT-C (Delis, Kramer, et al., 1994) is an instrument for the assessment of verbal memory and learning in children and adolescents. Table 30 in Appendix A depicts Pearson correlation coefficients and mean performance data for the CMS (Cohen, 1997) and CVLT-C, revealing low to moderate positive correlations between the CMS Attention/Concentration, Verbal Immediate, Verbal Delayed, Delayed Recognition, and General Memory indices and those of the CVLT-C. Table 31 in Appendix A depicts correlations, means, and standard deviations for certain of the CVLT components and the CMS Word Lists scores, revealing strong convergent validity. The smallest correlations are between the CVLT-C intrusions measure and the Long Delay and Delayed Recognition scores for the Word Lists subtest of the CMS.

The CMS (Cohen, 1997) can be administered by a trained technician, but must be administered by someone with the appropriate graduate or professional training. The examiner should be familiar with the APA (1985) guidelines for educational and psychological testing.

CHILD BEHAVIORAL CHECKLIST (CBCL)

The Child Behavior Checklist (CBCL) (Achenbach, 1991) is designed to produce standardized descriptions of children's behavior, including competencies and problems, rather than diagnostic information. It is designed to produce one component of an empirical, multiaxial assessment.

It is designed for completion by a parent or guardian with at least a fifth-grade reading ability. Otherwise, the CBCL (Achenbach, 1991) can be administered by interview. No special qualifications are necessary. The person completing the checklist should be aware that its purpose is to obtain a view of the child's behavior as the parental/guardian sees it. The person completing the checklist should be informed that there could be some items that don't apply, since the CBCL was designed for a wide array of children. Confidentiality should be discussed with the parent/guardian and any questions regarding the CBCL should be answered.

The CBCL (Achenbach, 1991) can be completed in as little as 10 minutes, but usually takes about 16 minutes for completion.

The CBCL (Achenbach, 1991) can be scored by hand following instructions in Appendix A of the manual, but administration and scoring of the CBCL require training in standardized assessment methodology and procedures, as well as experience and training in working with parents and children.

The CBCL (Achenbach, 1991) consists of 20 competence items designed to obtain parent/guardian views of the child's participation in activities, athletics, games, hobbies, work-related tasks, social and school functioning, and ability to

work alone, as well as with others. Specific problem areas are addressed in 118 problem item questions, as well as 2 open-ended problem questions, and are scored on a 3-level scale.

The CBCL (Achenbach, 1991) was standardized on a subset of a non-handicapped national sample of 7 to 18-year olds assessed Spring, 1989. Participants were selected as representative of the 48 contiguous states in accordance with SES, ethnicity, region, and residence data. Data for the 4-6 year olds were gathered identifying households in the survey with children (N=398) in that age range. When a household had more than one child of that age range, the child with the next-occurring birthday was selected. Children receiving mental health services or attending remedial classes within the preceding 12-month period were excluded.

CBCL RELIABILITY

The CBCL (Achenbach, 1991) manual claims that scale and item scores of the CBCL discriminate quite well between referred and non-referred participants. The manual states that the test-retest reliability is high, while the standard error of measurement is small. The manual claims an overall intra-class correlation coefficient (ICC) via one-way analyses of variance of .927 for the 20 competence items and .959 for the 118 specific problem questions (both $p<.001$). These correlation data reveal very high inter-interviewer reliability in scores for each item relative to scores for each other item. The manual reports very high test-retest reliability. An overall ICC of .996 for the 20 competence items is reported, as well as an ICC of .952 for the 118 specific problem questions (both $p<.001$).

The manual reports that Pearson correlations reveal good inter-parent agreement, with mean rs for competence scales ranging from .74 to .76 and for problems scales ranging from .65 to .75 for the four sex/age groups. Odds ratios indicate significantly high agreement between mothers' and fathers' ratings of their children as falling within normal versus clinical ranges on the CBCL scales.

CBCL VALIDITY

The CBCL manual (Achenbach, 1991) reports high content validity for the CBCL, supported by the ability of CBCL items to discriminate significantly between demographically matched referred versus non-referred participants. The CBCL manual claims the assessment has high construct validity, as supported by the assessment's correlates with other measures of the same construct. The manual claims high criterion-related validity, citing the quantitative scale scores' abilities to discriminate between referred and non-referred participants after accounting for demographic effects. CBCL scale scores also discriminate significantly between referred and non-referred children who are demographically matched.

The CBCL manual (Achenbach, 1991) reports that results of ANCOVAs reveal significantly ($p<.01$) lower scores for referred children as compared to non-referred children on competence items and scales. The manual reports quite small demographic differences in competence scores. The most consistent demographic effect is the tendency of upper SES parents scoring their children somewhat higher than lower SES parents. Most problem items are scored significantly ($p<.01$) higher for referred than non-referred children.

CBCL APPLICATIONS

Applications of the CBCL (Achenbach, 1991) include intakes and evaluations, clinical interviews, interventions, academic and medical contexts, forensic investigations, and assessments of child-abuse-related problems, as well as case registers detailing all cases entering a system. The CBCL can be used in conjunction with other data sources, such as self-report measures and interviews. Since the CBCL is not linked to one theoretical perspective, it is appropriate for various types of research.

DATA ANALYSIS

SPSS for Windows, Version 10 is the software package that was used for the analysis of data. T-tests compared the means for the participants on each sub-test of the CMS (Cohen, 1997) to the means for the CMS standardization sample for those same sub-tests. Pearson correlations were used to determine any correlations between the CMS results and the results from the CBCL (Achenbach, 1991).

4

RESULTS

NULL HYPOTHESIS 1

There is no difference between the participants' mean scores on the indices of the Children's Memory Scale (CMS) (Cohen, 1997) and the mean scores of the CMS standardization sample on the CMS indices.

ALTERNATIVE HYPOTHESIS 1

There is a difference between participants' mean scores on the indices of the Children's Memory Scale (CMS) (Cohen, 1997) and the mean scores of the CMS standardization sample on the CMS indices.

RESEARCH QUESTION 1

Is there a difference between participants' mean scores on the indices of the Children's Memory Scale (CMS) (Cohen, 1997) and the mean scores of the CMS standardization sample on the CMS indices?

NULL HYPOTHESIS 2

No relationship exists between the scores of the participants on the indices of the Children's Memory Scale (CMS) (Cohen, 1997) and the scores of the participants on the indices of the Child Behavioral Checklist (CBC) (Achenbach, 1991).

ALTERNATIVE HYPOTHESIS 2

A negative correlation relationship exists between the scores of the participants on the Children's Memory Scale (CMS) (Cohen, 1997) and the scores of the participants on the Child Behavioral Checklist (CBC) (Achenbach, 1991).

RESEARCH QUESTION 2

Is there a relationship between memory functioning and reported behaviors of participants?

One-sample T-tests were used to compare the memory performance of the participants (n=22) on the Children's Memory Scale (CMS) (Cohen, 1997) to the CMS standardization sample's performance on the CMS. Results at the two-tailed significance level reveal no significant differences in performance of the participants versus the standardization sample on the following CMS indices: Visual Immediate ($t = -.487$, $p < .631$), Visual Delayed ($t = -.890$, $p < .383$), Verbal Immediate ($t = .949$, $p < .353$), General Memory ($t = -.998$, $p < .330$), Attention/Concentration ($t = -.014$, $p < .989$), Learning ($t = 1.158$, $p < .260$), and Delayed Recognition ($t = .156$, $p < .877$). Results of the participants on the Verbal Delayed index of the CMS were significantly lower than those for the standardization sample ($t = -3.011$, $p < .007$). Results are reported in Tables 1-4 of Appendix B of this thesis.

Pearson r bivariate correlations at two-tailed significance were used to determine whether a relationship exists between the participants' performance on the CMS (Cohen, 1997) and the participants' scores on the Child Behavioral Checklist (CBC) (Achenbach, 1991). No significant correlations were found between the indices of the CMS and CBC. Results are reported in Table 5 of Appendix B of this thesis.

5

DISCUSSION/LIMITATIONS, ADVANTAGES OF THIS STUDY, & RESEARCH RECOMMENDATIONS

DISCUSSION/LIMITATIONS

The results of the T-tests reveal that there is no statistically significant difference between the scores of the participants on the Children's Memory Scale (CMS) indices (Cohen, 1997) and the scores of the CMS standardization sample on the CMS indices, with the exception of the CMS Verbal Delayed index. The scores of the participants were significantly lower than those of the standardization sample on the CMS Verbal Delayed index.

The results of the Pearson r reveal that there is no statistically significant relationship between participants' scores on the Children's Memory Scale (CMS) indices (Cohen, 1997) and their scores on the Child Behavioral Checklist (CBC) (Achenbach, 1991) indices.

Due to the number of comparisons made, the difference in performance of the participants on the Verbal Delayed index of the Children's Memory Scale (CMS) (Cohen, 1997) could have resulted from chance. Therefore, the null hypothesis for the participants' performance on the CMS as a whole is not rejected, although it can be rejected for that one particular index.

This lack of significance could result for several reasons. The obvious problem is the sample size. Sample size for this study was quite small (n = 22). Also, this study lacked control. For instance, the CMS standardization sample may

have been exposed to a great deal of violence, given the likelihood of exposure to violence in our society as discussed in the Literature Review section of this thesis. A different design could help to tease out such confounds.

This research was conducted at one shelter in a small New Jersey town; future research would need to examine the degree to which these findings generalize across other populations in other American cities and towns, as well as across foreign populations.

The sample in this study was not randomly selected, limiting the generalizability of the results. There could be an interaction between selection and assessment, since participants volunteered for assessment. In addition, children exposed to types of trauma other than domestic violence were not assessed for comparison. Due to difficulty scheduling appointments, the children were not assessed at the same times of the day. The sample size as a whole was too small (n=22) for a thorough examination of the hypotheses and relevant issues. Thus, the statistical power was limited for comparison of participants to the standardization sample.

The assessment of memory chosen, The Children's Memory Scale (CMS) (Cohen, 1997), does not evaluate procedural memory, a type of memory associated with skill learning and classical conditioning. In addition, the CMS does not allow for the evaluation of long-term memory greater than 30 minutes.

This study examined the memory functioning of the children at one point in time. However, there could be a delayed onset of certain effects of trauma, or cumulative effects that appear several years after exposure to trauma.

The assessment of memory was limited to one statistically valid and reliable assessment, the CMS (Cohen, 1997). The assessment of behaviors was limited to the CBCL (Achenbach, 1991). It is always best to multiple assessments of a construct that are widely recognized in the field. However, using multiple assessments with children poses additional problems related to attrition, as well as ethical concerns regarding over-taxing children who have been exposed to trauma.

This researcher's appreciation for memory performance could be a personal demand characteristic that might have caused unintentional expectancy effects for the participants to perform at optimum levels on the CMS (Cohen, 1997). If this occurred, external validity for similar results across populations could be jeopardized.

ADVANTAGES OF THIS STUDY

While it is unethical to intentionally expose children to trauma, the children in this study were already exposed to trauma in the form of domestic violence and were to receive individual counseling and other shelter services as treatment for the same. Thus, this is an ethical way to examine the memory functioning in children exposed to trauma.

The population examined is challenging for researchers due to trauma-related and resource obstacles. For this research, services such as babysitting for children too young to participate, were provided by the shelter.

The assessment of memory chosen, The Children's Memory Scale (CMS) (Cohen, 1997), examines multiple aspects of memory and learning of interest in this study, including the following:

- Immediate recall

- Delayed recall
- Auditory versus visual memory
- Encoding (storage) versus retrieval of material
- Free recall versus recognition
- Literal/almost-literal versus thematic recall
- Attention Concentration

Thus, it is a comprehensive assessment of various aspects of children's memory.

Until recently, child neuropsychologists were forced to utilize poorly standardized assessments of memory originally developed for use with adults (Cohen, 1997). However, the CMS (Cohen, 1997) is a well-standardized assessment designed specifically for the evaluation of memory functioning in children.

The CMS (Cohen, 1997) was administered by only one researcher, Deanna Cosgrove, limiting any confounds that could arise from multiple administrators.

RECOMMENDATIONS FOR FUTURE RESEARCH

Continued research efforts are essential to ensure that quality psychological and educational services are provided to children who have been exposed to Type II trauma. The memory functioning in children who are receiving treatment for Posttraumatic Stress Disorder should be examined in relation to those in a non-PTSD matched control group, as well as in groups with disorders comorbid with Posttraumatic Stress Disorder. Longitudinal studies are needed to examine the memory functioning in participants receiving treatment for Posttraumatic Stress Disorder as children to address the effects of trauma on memory functioning over time. In addition, the memory functioning in children with and without Posttraumatic

Stress Disorder who have been exposed to other types of Type II trauma, such as sexual abuse, should be examined. Children who have received on-going treatment for Posttraumatic Stress Disorder symptomatology should be examined at several-year intervals to examine any emergent patterns in memory functioning. The patterns revealed in research with adult participants with a posttraumatic stress disorder diagnosis are not likely to emerge overnight, so it is important to document the changes taking place prior to adulthood over the course of time.

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APPENDIX A

TABLE 1

Brief Description of CMS Subtests

Subtest	Description
Auditory/ Verbal	
Stories (Core)	This subtest assesses the ability to recall meaningful and semantically related verbal material. In the immediate portion, two stories are ready by the examiner, and the examinee is asked to retell the stories from memory. In the delayed portion, the examinee is asked to retell the stories from memory and then to answer factual questions about the stories.
Word Pairs (Core)	This subtest assesses the ability to learn a list of word pairs over three learning trials. In the immediate portion, the examiner reads a list of word pairs aloud, then reads the first word of each pair and asks the examinee to provide the second word from memory. In the delayed portion, the examinee is asked to provide the word pairs from memory and then to indicate whether word pairs read aloud are those he or she was asked to remember earlier.
Word Lists (Supplemental)	This subtest assesses the ability to learn a list of unrelated words over four learning trials. In the immediate portion, after the initial presentation and recall of the list, the examinee is reminded of only those words which he or she forgot. This is followed by a single presentation and recall of a distractor word list, then recall of the first list. In the delayed portion, the examinee is asked to provide the word list from memory and then to indicate whether words read aloud are those he or she was asked to remember earlier.
Visual/Nonverbal	
Dot Locations (Core)	This subtest assesses the ability to learn the spatial location of an array of dots over three learning trials. In the immediate portion, presentation and recall of the learning trials are followed by a single presentation and recall of a distractor array, then recall of the first dot array. In the delayed portion, the examinee is asked to recall the dot array presented earlier.
Face (Core)	The subtest assesses the ability to remember and recognize a series of facts. In the immediate portion, the examinee is presented with a series of faces and asked to remember them. For both the immediate and delayed portions, the examinee is shown the same faces along with distractor faces and asked to identify each face as either a face he or she was asked to remember or a new one.

(Cohen, 1997)

TABLE 2

Brief Description of CMS Subtest (Continued)

Subtest	Description
Family Pictures (Supplemental)	This subtest assesses the ability to remember scenes of family members doing various activities. In the immediate portion, the examinee is shown four different scenes with family members in them an asked to remember each scene. For both the immediate and delayed portions, the examinee is asked to remember which characters were in each scene, where they were positioned, and what they were doing.
Attention/Concentration	
Numbers (Core)	This subtest assesses the ability to repeat random digit sequences of graduated length. This subtest has two portions. In the Forward portion, the examinee is asked to repeat the digits in the same sequence as presented orally by the examiner. In the Backward portion, the examinee is asked to repeat the digits in the reverse order of that presented orally by the examiner.
Sequences (Core)	This subtest assesses the ability to mentally manipulate and sequence auditory/verbal information as quickly as possible. The examinee is asked to perform such tasks as saying the days of the week backward and counting by 4s.
Picture Locations (Supplemental)	This subtest assesses immediate visual/nonverbal memory for spatial location of pictured objects (animals or vehicles). The examinee is shown pictures placed in various locations within a rectangle. The examinee is then asked to recall the locations of the pictures.

(Cohen, 1997)

TABLE 3

Reliability Coefficients of the Supplemental Subtest Scores by Age-Group

	Age 5	Age 6	Age 7	Age 8	Age 9
Dot Locations					
Short Delay[b]	.57	.57	.57	.57	.52
Stories					
Immediate Thematic	.75	.75	.74	.73	.73
Delayed Thematic	.79	.73	.76	.71	.77
Word Pairs					
Immediate[b]	.57	.57	.57	.57	.82
Family Pictures					
Immediate	.69	.67	.68	.71	.61
Delayed	.62	.68	.65	.67	.69
Word Lists					
Learning	.84	.86	.87	.81	.86
Delayed[b]	.66	.66	.66	.66	.77
Delayed Recognition	.78	.78	.79	.64	.77
Numbers					
Forward	.74	.79	.78	.80	.73
Backward	.82	.67	.68	.66	.69
Picture Locations					
Total Scores	.76	.75	.76	.71	.67

(Cohen, 1997)

TABLE 4

Reliability Coefficients of the Supplemental Subtest Scores
(Continued)

	Age 10	Age 11	Age 12	Age 13-14	Age 15-16	Average r_{xx}[a]
Dot Locations						
Short Delay[b]	.52	.52	.52	.52	.52	.54
Stories						
Immediate Thematic	.77	.75	.75	.74	.78	.75
Delayed Thematic	.73	.73	.72	.72	.78	.75
Word Pairs						
Immediate[b]	.82	.82	.82	.76	.76	.73
Family Pictures						
Immediate	.57	.57	.51	.47	.63	.62
Delayed	.61	.66	.62	.53	.62	.64
Word Lists						
Learning	.89	.85	.82	.82	.84	.86
Delayed[b]	.77	.77	.77	.74	.74	.72
Delayed Recognition	.86	.73	.73	.85	.73	.77
Numbers						
Forward	.78	.71	.75	.77	.83	.77
Backward	.72	.80	.76	.79	.80	.74
Picture Locations						
Total Score	.81	.75	.68	.73	.70	.73

(Cohen, 1997)

TABLE 5

Test-Retest Stability Coefficients of Indexes by Age Band

			(Age 5-8)			
	First Testing		**Second Testing**			**Corrected**
	Mean	**SD**	**Mean**	**SD**	**r_{12}**	**r^a**
Indexes						
Visual Immediate	101.8	13.4	113.1	14.7	.66	.69
Visual Delayed	103.0	12.5	109.0	14.5	.66	.70
Verbal Immediate	100.3	16.6	111.3	18.2	.87	.85
Verbal Delayed	100.7	16.2	113.7	15.6	.63	.59
General Memory	102.7	17.3	116.9	16.6	.85	.83
Attention/Concentration	102.0	15.6	107.3	14.4	.84	.85
Learning	100.8	14.1	109.2	14.3	.78	.79
Delayed Recognition	100.7	15.3	105.7	17.0	.57	.54

(Cohen, 1997)

TABLE 6

Test-Retest Stability Coefficients of Indexes by Age Band
(Continued)

			(Ages 9-12)			
	First Testing		**Second Testing**			**Corrected**
	Mean	**SD**	**Mean**	**SD**	r^{12}	**r**
Indexes						
Visual Immediate	103.6	2.1	113.5	4.4	.65	.69
Visual Delayed	102.1	2.3	112.8	3.0	.61	.66
Verbal Immediate	98.7	6.5	109.8	8.3	.82	.82
Verbal Delayed	100.3	5.7	109.9	8.4	.79	.79
General Memory	102.5	6.1	119.0	5.4	.86	.86
Attention/Concentration	98.1	1.5	100.7	6.6	.88	.89
Learning	100.5	5.1	109.9	5.9	.67	.67
Delayed Recognition	100.8	4.7	103.0	6.8	.57	.56

(Cohen, 1997)

TABLE 7

Test-Retest Stability Coefficients of Indexes by Age Band
(Continued)

			(Ages 13-16)			
	First Testing		**Second Testing**			**Corrected**
	Mean	**SD**	**Mean**	**SD**	**r_{12}**	**r**
Indexes						
Visual Immediate	102.6	10.8	115.1	12.2	.26	.29
Visual Delayed	101.0	14.8	110.6	12.6	.40	.38
Verbal Immediate	99.9	18.0	115.8	17.2	.85	.81
Verbal Delayed	101.1	19.0	112.2	17.9	.87	.82
General Memory	102.1	17.7	122.5	15.8	.86	.84
Attention/Concentration	100.6	16.5	104.2	16.1	.86	.85
Learning	100.7	12.6	112.1	14.8	.78	.78
Delayed Recognition	101.1	14.5	105.6	16.8	.56	.52

(Cohen, 1997)

TABLE 8

Decision Consistency Stability Coefficients of Core Battery Subtest Scores and Indexes by Age Band

			(Ages 5-8)		
	First Testing		**Second Testing**		**Decision Consistency**
	Mean	**SD**	**Mean**	**SD**	
Dot Locations					
Learning	10.4	2.7	10.9	3.0	.87
Total Score	10.4	2.8	11.5	3.2	.81
Long Delay	10.4	2.3	10.8	2.5	.85
Stories					
Immediate	9.9	3.0	11.5	3.0	.81
Delayed	10.2	2.8	12.5	2.8	.85
Delayed Recognition	9.9	3.2	11.1	3.3	.81
Faces					
Immediate	10.2	2.8	12.8	3.2	.83
Delayed	10.6	3.2	12.2	3.4	.83
Word Pairs					
Learning	10.2	3.4	12.2	3.5	.81
Total Score	9.9	3.3	12.1	3.4	.79
Long Delay	10.0	3.6	12.0	3.6	.75
Delayed Recognition	10.3	3.2	10.7	3.1	.73
Numbers					
Total Score	10.4	3.0	11.2	2.9	.90
Sequences					
Total Score	10.3	2.8	11.1	2.7	.85
Indexes					
Visual Immediate	101.8	13.4	113.1	14.7	.80
Visual Delayed	103.0	12.5	109.0	14.5	.83
Verbal Immediate	100.3	16.6	111.3	18.2	.77
Verbal Delayed	100.7	16.2	113.7	15.6	.79
General Memory	102.7	17.3	116.9	16.6	.71
Attention/Concentration	102.0	15.6	107.3	14.4	.83
Learning	100.8	14.1	109.2	14.3	.83
Delayed Recognition	100.7	15.3	105.7	17.0	.69

(Cohen, 1997)

TABLE 9

Decision Consistency Stability Coefficients of Core Battery Subtest Scores and Indexes by Age Band (Continued)

			(Ages 9-12)		
	First Testing		**Second Testing**		**Decision Consistency**
	Mean	**SD**	**Mean**	**SD**	
Dot Locations					
Learning	10.4	2.8	11.1	3.0	.87
Total Score	10.5	2.7	11.4	2.7	.87
Long Delay	10.9	2.8	11.5	2.3	.91
Stories					
Immediate	9.6	2.9	11.1	3.1	.73
Delayed	9.9	2.9	11.6	3.3	.71
Delayed Recognition	10.1	3.4	10.8	3.5	.78
Faces					
Immediate	10.7	2.5	13.0	2.9	.87
Delayed	9.8	2.9	12.7	2.8	.80
Word Pairs					
Learning	9.9	3.2	11.9	3.6	.80
Total Score	9.7	3.4	12.1	3.5	.78
Long Delay	10.2	3.0	11.7	3.4	.82
Delay Recognition	10.2	3.1	10.2	3.2	.78
Numbers					
Total Score	9.8	2.7	10.4	3.1	.89
Sequences					
Total Score	9.6	2.9	9.9	3.0	.80
Indexes					
Visual Immediate	103.6	2.1	113.5	4.4	.82
Visual Delayed	102.1	2.3	112.8	3.0	.93
Verbal Immediate	98.7	6.5	109.8	8.3	.73
Verbal Delayed	100.3	5.7	109.9	8.4	.82
General Memory	102.5	6.1	119.0	5.4	.76
Attention/Concentration	98.1	4.5	100.7	6.6	.84
Learning	100.5	5.1	109.9	5.9	.78
Delayed Recognition	100.8	4.7	103.0	6.8	.69

(Cohen, 1997)

TABLE 10

Decision Consistency Stability Coefficients of Core Battery Subtest Scores and Indexes by Age Band (Continued)

			(Ages 13-16)		
	First Testing		**Second Testing**		**Decision Consistency**
	Mean	**SD**	**Mean**	**SD**	
Dot Locations					
Learning	10.7	2.1	12.0	2.1	.93
Total Score	11.0	2.1	11.9	1.9	.89
Long Delay	10.3	2.5	11.1	2.5	.82
Stories					
Immediate	10.4	3.0	12.5	2.9	.75
Delayed	10.5	3.2	12.3	2.7	.82
Delayed Recognition	10.8	2.4	12.4	3.8	.86
Faces					
Immediate	9.9	3.1	13.0	3.1	.82
Delayed	10.0	3.1	12.4	3.3	.79
Word Pairs					
Learning	9.7	3.3	12.0	3.8	.86
Total Score	9.6	3.3	12.0	3.6	.89
Long Delay	9.8	3.5	11.6	3.6	.93
Delayed Recognition	9.6	3.0	9.6	3.1	.82
Numbers					
Total Score	9.5	3.0	10.3	3.3	.75
Sequences					
Total Score	10.7	3.4	11.1	2.9	.89
Indexes					
Visual Immediate	102.6	10.8	115.1	12.2	.93
Visual Delayed	101.0	14.8	110.6	12.6	.79
Verbal Immediate	99.9	18.0	115.8	17.2	.68
Verbal Delayed	101.1	19.0	112.2	17.9	.75
General Memory	102.1	17.7	122.5	15.8	.61
Attention/Concentration	100.6	16.5	104.2	16.1	.79
Learning	100.7	12.6	112.1	14.8	.89
Delayed Recognition	101.1	14.5	105.6	16.8	.75

(Cohen, 1997)

TABLE 11

Decision Consistency Stability Coefficients of the Supplemental Subtest Scores by Age Band

	Ages 5-8				
	First Testing		Second Testing		Decision
	Mean	SD	Mean	SD	Consistency
Dot Locations					
Short Delay	10.4	3.0	11.5	3.3	.73
Stories					
Immediate Thematic	10.2	2.8	11.2	2.7	.88
Delayed Thematic	10.5	2.6	11.7	2.5	.88
Word Pairs					
Immediate	10.1	2.9	11.2	2.9	.79
Family Pictures					
Immediate	9.6	2.8	10.6	3.2	.87
Delayed	9.7	2.6	11.0	3.1	.83
Word Lists					
Learning	10.2	3.0	11.7	2.8	.79
Delayed	10.2	2.6	11.7	2.8	.94
Delayed Recognition	10.3	3.1	10.9	2.9	.85
Numbers					
Forward	10.5	2.9	11.1	2.9	.79
Backward	10.0	3.2	10.8	3.4	.81
Picture Locations					
Total Score	10.5	2.6	11.5	3.1	.92

(Cohen, 1997)

TABLE 12

Decision Consistency Stability Coefficients of the Supplemental Subtest Scores by Age Band (Continued)

	Ages 9-12				
	First Testing		Second Testing		Decision Consistency
	Mean	SD	Mean	SD	
Dot Locations					
Short Delay	10.6	2.9	11.3	2.3	.87
Stories					
Immediate Thematic	10.1	3.2	10.8	2.5	.80
Delayed Thematic	10.0	2.9	11.1	3.1	.78
Word Pairs					
Immediate	10.3	3.1	10.7	3.3	.80
Family Pictures					
Immediate	9.4	2.9	10.6	3.3	.87
Delayed	9.6	3.0	10.6	3.1	.84
Word Lists					
Learning	10.4	3.3	12.2	3.6	.80
Delayed	10.2	3.4	11.6	3.4	.84
Delayed Recognition	10.3	2.8	10.7	2.7	.78
Numbers					
Forward	10.1	2.3	10.6	2.7	.82
Backward	10.2	2.7	10.2	3.2	.87
Picture Locations					
Total Score	10.2	3.1	11.6	2.9	.82

(Cohen, 1997)

TABLE 13

Decision Consistency Stability Coefficients of the Supplemental Subtest Scores by Age Band (Continued)

	Ages 13-16				
	First Testing		Second Testing		Decision Consistency
	Mean	SD	Mean	SD	
Dot Locations					
Short Delay	10.8	1.7	11.1	2.4	.82
Stories					
Immediate Thematic	10.8	3.3	11.4	2.8	.86
Delayed Thematic	10.5	2.8	11.9	2.4	.86
Word Pairs					
Immediate	9.6	3.5	10.8	3.4	.75
Family Pictures					
Immediate	10.1	2.8	11.8	2.6	.82
Delayed	9.7	2.7	11.9	2.2	.82
Word Lists					
Learning	9.2	3.2	11.2	3.3	.75
Delayed	9.9	3.3	10.8	3.4	.68
Delayed Recognition	9.9	3.1	11.2	2.4	.71
Numbers					
Forward	9.7	3.0	10.3	3.2	.93
Backward	9.5	3.5	10.2	3.5	.64
Picture Locations					
Total Score	10.5	2.7	12.4	2.8	.86

(Cohen, 1997)

TABLE 14

Intraclass Correlations for Selected Subtest Scores: Stories by Age Band

	(Ages 5-8)	(Ages 9-12)	(Ages 13-16)
Stories			
Immediate	.99	.99	.98
Delayed	.99	.99	.99
Delayed Recognition	1.00	1.00	1.00
Immediate Thematic	.91	.96	.91
Delayed Thematic	.94	.92	.95

(Cohen, 1997)

TABLE 15

Intraclass Correlations for Selected Subtest Scores: All Ages

	All Ages
Dot Locations	
Learning	1.00
Total Score	.98
Short Delay	1.00
Faces	
Immediate	1.00
Delayed	.97
Word Pairs	
Learning	1.00
Immediate	1.00
Total Score	1.00
Long Delay	1.00
Delayed Recognition	1.00
Family Pictures	
Immediate	.98
Delayed	.98
Word Lists	
Learning	1.00
Delayed	1.00
Delayed Recognition	1.00
Intrusions	1.00
Immediate Recall	
Proactive Score	1.00
Retroactive Score	.88
Numbers	
Total Score	1.00
Forward	1.00
Backward	1.00
Sequences	
Total Score	1.00
Picture Locations	
Total Score	.99

(Cohen, 1997)

TABLE 16

Standard Error of Measurement for Core Battery Subtest Scaled Scores and Index Scores by Age-Group

	Age-Group					
	Age 5	**Age 6**	**Age 7**	**Age 8**	**Age 9**	**Age 10**
Dot Locations						
Learning	1.4	1.3	1.6	1.8	1.9	1.5
Total Score	1.3	1.4	1.5	1.7	1.6	1.3
Long Delay	1.5	1.5	1.5	1.5	1.6	1.6
Stories						
Immediate	1.4	1.6	1.7	1.4	1.6	1.6
Delayed	1.6	1.5	1.6	1.6	1.6	1.6
Delayed Recognition	1.5	1.5	1.5	1.5	1.4	1.4
Faces						
Immediate	1.5	1.6	1.6	1.6	1.7	1.6
Delayed	1.6	1.6	1.7	1.5	1.6	1.6
Word Pairs						
Learning	0.8	1.0	1.0	1.0	1.1	0.9
Total Score	1.0	1.2	1.1	1.2	1.1	1.2
Long Delay	1.6	1.6	1.6	1.6	1.1	1.1
Delayed Recognition	1.2	1.4	1.5	1.0	1.4	1.6
Numbers						
Total Score	1.4	1.4	1.3	1.4	1.6	1.3
Sequences						
Total Score	1.3	1.3	1.4	1.2	1.3	1.2
Indexes						
Visual Immediate	6.2	6.6	7.7	7.3	8.1	6.9
Visual Delayed	6.6	7.4	7.5	6.8	6.9	6.9
Verbal Immediate	4.9	6.4	6.3	5.5	5.7	5.9
Verbal Delayed	7.2	7.3	7.6	7.5	5.8	5.8
General Memory	4.0	4.7	5.1	4.6	4.4	4.7
Attention/Concentration	5.3	5.7	5.6	5.4	6.0	5.1
Learning	5.1	5.0	6.2	6.3	6.9	5.9
Delayed Recognition	5.9	6.9	7.3	5.9	6.4	7.1

(Cohen, 1997)

TABLE 17

Standard Error of Measurement for Core Battery Subtest Scaled Scores and Index Scores by Age-Group (Continued)

	Age-Group				
	Age 11	**Age 12**	**Age 13-14**	**Age 15-16**	**Average a SemS**
Dot Locations					
Learning	1.8	1.5	1.3	1.7	1.6
Total Score	1.6	1.4	1.4	1.4	1.5
Long Delay	1.6	1.6	1.6	1.6	1.5
Stories					
Immediate	1.4	1.5	1.4	1.6	1.5
Delayed	1.3	1.4	1.3	1.5	1.5
Delayed Recognition	1.4	1.4	1.6	1.6	1.5
Faces					
Immediate	1.6	1.7	1.8	1.5	1.6
Delayed	1.6	1.6	1.6	1.6	1.6
Word Pairs					
Learning	1.0	0.8	0.7	0.9	0.9
Total Score	1.0	1.0	0.9	1.0	1.1
Long Delay	1.1	1.1	0.8	0.8	1.3
Delayed Recognition	1.6	1.3	1.6	1.5	1.4
Numbers					
Total Score	1.2	1.3	1.3	1.2	1.4
Sequences					
Total Score	1.2	1.3	1.3	1.4	1.3
Indexes					
Visual Immediate	7.4	8.1	8.2	7.6	7.4
Visual Delayed	7.3	9.1	7.5	7.7	7.4
Verbal Immediate	5.3	5.3	5.0	5.7	5.6
Verbal Delayed	5.4	5.5	4.6	5.0	6.3
General Memory	4.0	5.0	4.4	4.1	4.5
Attention/Concentration	5.0	5.6	5.3	5.5	5.5
Learning	6.1	5.3	5.3	6.4	5.9
Delayed Recognition	7.1	6.1	8.0	6.9	6.8

(Cohen, 1997)

TABLE 18

Standard Error of Measurement for Supplemental Subtest Scores by Age-Group

	Age 5	Age 6	Age 7	Age 8	Age 9	Age 10
Dot Locations						
Short Delay	2.0	2.0	2.0	2.0	2.1	2.1
Stories						
Immediate Thematic	1.5	1.5	1.5	1.6	1.6	1.4
Delayed Thematic	1.4	1.6	1.5	1.6	1.4	1.6
Word Pairs						
Immediate	2.0	2.0	2.0	2.0	1.3	1.3
Family Pictures						
Immediate	1.7	1.7	1.7	1.6	1.9	2.0
Delayed	1.8	1.7	1.8	1.7	1.7	1.9
Word Lists						
Learning	1.2	1.1	1.1	1.3	1.1	1.0
Delayed	1.7	1.7	1.7	1.7	1.4	1.4
Delayed Recognition	1.4	1.4	1.4	1.8	1.4	1.5
Numbers						
Forward	1.5	1.4	1.4	1.3	1.6	1.4
Backward	1.3	1.7	1.7	1.7	1.7	1.6
Pictures Locations						
Total Score	1.5	1.5	1.5	1.6	1.7	1.3

(Cohen, 1997)

TABLE 19

Standard Error of Measurement for Supplemental Subtest Scores by Age-Group (Continued)

	Age 11	Age 12	Age 13-14	Age 15-16	Average[a] SE_m [s]
Dot Locations					
Short Delay	2.1	2.1	2.1	2.1	2.0
Stories					
Immediate Thematic	1.5	1.5	1.5	1.4	1.5
Delayed Thematic	1.6	1.6	1.6	1.4	1.5
Word Pairs					
Immediate	1.3	1.3	1.5	1.5	1.6
Family Pictures					
Immediate	2.0	2.1	2.2	1.8	1.9
Delayed	1.7	1.8	2.1	1.8	1.8
Word Lists					
Learning	1.0	1.2	1.3	1.2	1.2
Delayed	1.4	1.4	1.5	1.5	1.6
Delayed Recognition	1.1	1.6	1.2	1.6	1.4
Numbers					
Forward	1.6	1.5	1.4	1.2	1.4
Backward	1.3	1.5	1.4	1.3	1.5
Picture Locations					
Total Score	1.5	1.7	1.6	1.6	1.6

(Cohen, 1997)

TABLE 20

Intercorrelations of Core Battery Subtests: All Ages

	Dot Locations Learning	**Dot Locations Long Delay**	**Dot Locations Total Score**	**Stories Immediate**	**Stories Delayed**	**Stories Del Rec**
Dot Locations						
Learning						
Long Delay	.46					
Total Score	.91	.62				
Stories						
Immediate	.18	.13	.18			
Delayed	.15	.11	.14	.88		
Delayed Recognition	.14	.10	.14	.56	.59	
Faces						
Immediate	07	.16	.11	.12	.09	.16
Delayed	.06	.15	.10	.11	.10	.15
Word Pairs						
Learning	.22	.17	.20	.40	.39	.30
Long Delay	.18	.15	.18	.29	.29	.22
Delayed Recognition	.19	.08	.16	.20	.20	.19
Total Score	.20	.15	.19	.39	.38	.29
Numbers						
Total Score	.22	.14	.22	.22	.23	.15
Sequences						
Total Scores	.27	.14	.26	.30	.31	.26

(Cohen, 1997)

TABLE 21

Intercorrelations of Core Battery Subtests: All Ages (Continued)

	Faces Immediate	Faces Delayed	Word Pairs Learning	Word Pairs Long Delay	Word Pairs Del Rec	Word Pairs Total Score	Numbers Total Score	Sequences Total Score
Dot Locations								
Learning								
Long Delay								
Total Scores								
Stories								
Immediate								
Delayed								
Delayed Recognition								
Faces								
Immediate								
Delayed	.59							
Word Pairs								
Learning	.16	.14						
Long Delay	.13	.15	.55					
Delayed Recognition	.08	.06	.50	.36				
Total Score	.16	.13	.96	.44	.48			
Numbers								
Total Score	.08	.12	.31	.24	.23	.26		
Sequences								
Total Score	.13	.13	.36	.35	.25	.31	.47	

(Cohen, 1997)

TABLE 22

Correlations Between Memory and Intellectual Functioning for CMS and DAS

		DAS				
CMS Indexes	**Verbal**	**Nonverbal**	**Spatial**	**GCA**	**Mean**	**SD**
Visual Immediate	.41**	.22	.43*	.34*	101.8	16.4
Visual Delayed	.33*	.14	.43*	.38*	103.3	14.8
Verbal Immediate	.50**	.32	.52**	.52**	106.6	13.2
Verbal Delayed	.51**	.41*	.44*	.52**	99.9	14.4
General Memory	.59**	.40*	.65**	.65**	105.9	15.3
Attention/Concentration	.36*	.47**	.40*	.51**	104.9	15.3
Learning	.40*	.38*	.62**	.58**	104.3	14.9
Delayed Recognition	.40*	.24	.28	.36*	104.4	12.5
Mean	114.3	106.8	105.3	111.5		
SD	16.7	17.6	18.8	17.1		

(Cohen, 1997)

TABLE 23

Correlations Between Memory and Intellectual Functioning for CMS and OLSAT

		OLSAT			
CMS Indexes	**Verbal**	**Non-Verbal**	**Total**	**Mean**	**SD**
Visual Immediate	.31	.55	.43	98.4	8.0
Visual Delayed	.08	.33	.09	95.7	12.3
Verbal Immediate	.52*	.51*	.55	103.7	15.9
Verbal Delayed	.72**	.61*	.72**	102.4	12.1
General Memory	.64**	.72**	.73**	99.8	12.4
Attention/Concentration	.72**	.84**	.82**	102.3	14.6
Learning	.40	.58**	.45	101.3	15.7
Delayed Recognition	.55	.40	.28	100.4	16.7
Mean	56.5	57.8	63.5		
SD	22.1	25.4	21.2		

(Cohen, 1997)

TABLE 24

Correlations Between Memory and Academic Achievement for CMS and WIAT

CMS Indexes	Mean	SD	Reading	Math	Language	Writing	Total
Visual Immediate	101.2	13.4	.24**	.29**	.14*	.22**	.27**
Visual Delayed	100.4	14.4	.08	.08	.04	.07	.14
Verbal Immediate	100.0	17.2	.51**	.55**	.52**	.50**	.61**
Verbal delayed	100.1	17.2	.54**	.55**	.52**	.52**	.42**
General Memory	101.3	17.2	.49**	.51**	.43**	.47**	.56**
Attention/Concentration	102.4	16.5	.58**	.58**	.48**	.57**	.62*
Learning	101.5	15.4	.37**	.39	.34**	.39**	.42**
Delayed Recognition	99.5	16.0	.38**	.38**	.42**	.38**	.47**
Mean			101.2	99.0	101.7	107.9	102.7
SD			16.1	16.5	15.7	16.0	16.3

(Cohen, 1997)

TABLE 25

Correlations Between Memory and Executive Functioning for CMS and WCST

CMS Indexes	Mean	SD	Categories	% Errors	% Persev Res	% Concept
Visual Immediate	96.9	14.1	.37**	.34*	.32	.38**
Visual Delayed	95.6	18.2	.24	.41**	.18	.28
Verbal Immediate	98.1	16.0	.37**	.46**	.38**	.47**
Verbal Delayed	98.0	16.3	.44**	.59**	.50**	.56**
General Memory	95.5	18.2	.42**	.50**	.40**	.46**
Attention/Concentration	94.6	18.5	.51**	.39**	.27*	.36**
Learning	97.1	14.5	.31*	.40**	.35	.38**
Delayed Recognition	91.9	21.5	.42**	.35	.39**	.36**

(Cohen, 1997)

TABLE 26

Correlations Between Memory and Executive Functioning for CMS and CCT

CMS Indexes	Level 1	Mean	SD	Level 2	Mean	SD
Visual Immediate	.43	104.4	18.4	.26	98.3	14.8
Visual Delayed	.33	97.0	14.4	.31	95.6	17.9
Verbal Immediate	.09	93.5	14.9	.66**	103.1	20.8
Verbal delayed	.22	96.6	16.2	.63**	99.2	20.0
General Memory	.25	99.2	18.4	.59**	99.6	22.6
Attention/Concentration	.06	83.2	14.7	.30	98.5	18.1
Learning	.28	95.5	14.4	.40*	101.4	19.2
Delayed Recognition	.31	86.5	23.9	.43**	94.0	20.1
Mean	44.4			50.1		
SD	18.8			10.1		

(Cohen, 1997)

TABLE 27

Correlations Between Memory and Language Processing for CMS and CLEF-3

CMS Indexes	Mean	SD	Expressive	Receptive	Total Language
Visual Immediate	101.8	13.6	.08	.31	.22
Visual Delayed	101.4	11.7	.12	.33	.26
Verbal Immediate	105.0	11.8	.40	.42*	.46*
Verbal delayed	105.1	12.1	.29	.27	.31
General Memory	108.0	11.2	.40	.51*	.52*
Attention/Concentration	103.3	14.9	.43*	.39*	.47*
Learning	100.3	11.4	.22	.38*	.35
Delayed Recognition	104.7	15.2	.35	.27	.35
Mean		109.2	103.9	106.4	
SD		11.8	12.4	11.3	

(Cohen, 1997)

TABLE 28

Correlations Between Memory Measures for CMS and WMS-III (Continued)

CMS Indexes	Auditory Delayed	Visual Delayed	Auditory Recognition Delayed	General Memory	Working Memory
Visual Immediate	.35	.46	.26	.43	.33
Visual Delayed	.24	.26	.13	.26	.23
Verbal Immediate	.63	.35	.56	.63	.36
Verbal Delayed	.65	.41	.56	.66	.44
General Memory	.64	.46	.52	.67	.44
Attention/Concentration	.41	.17	.48	.43	.68
Learning	.58	.40	.44	.59	.48
Delayed Recognition	.48	.36	.44	.52	.21
Mean	94.66	103.17	99.48	98.92	98.36
SD	18.52	14.90	16.82	17.02	14.70

(Cohen, 1997)

TABLE 29

Correlations Between Memory Measures for CMS and WRAML

CMS Indexes	Mean	SD	General	Learning	Verbal	Visual
Visual Immediate	99.4	13.5	.41*	.43*	.27	.26
Visual Delayed	103.9	14.0	.55**	.34	.40*	.50**
Verbal Immediate	105.4	13.9	.58**	.48**	.60***	.41*
Verbal Delayed	102.2	14.7	.56**	.44**	.51**	.44*
General Memory	102.8	18.4	.64***	.53**	.55**	.50**
Attention/Concentration	98.8	17.0	.64***	.20	.70***	.43*
Learning	102.4	14.5	.59***	.50*	.48**	.40*
Delayed Recognition	97.7	17.0	.60***	.40*	.49**	.53**
Mean			97.5	100.9	104.9	102.2
SD			13.9	13.5	13.3	13.3

(Cohen, 1997)

TABLE 30

Correlations Between Memory Measures for CMS and CVLT-C

CMS Indexes	Mean	SD	Trial 1	Trial 5	Trial 1-5	Long Del	Rec
Visual Immediate	102.5	13.4	.40	.35	.50*	.31	.41
Visual Delayed	111.6	11.6	-.10	-.01	-.04	.27	-.11
Verbal Immediate	109.8	15.0	.08	.11	.18	.36	.04
Verbal Delayed	108.6	18.1	.38	.31	.49*	.47*	.36
General Memory	112.0	16.1	.32	.29	.42	.47*	.29
Attention/Concentration	101.6	16.4	.55**	.32	.51*	.17	.10
Learning	104.6	12.6	.15	.09	.20	.33	.15
Delayed Recognition	106.9	16.5	.50*	.35	.52*	.32	.35
Mean			.61	.25	54.7	.41	.41
SD			.90	1.0	10.9	1.0	.96

(Cohen, 1997)

TABLE 31

Correlations Between CMS Word Lists and CVLT-C

CMS Word Lists	Trial 1	Trial 5	Trial 1-5	Short Delay	Long Delay	Recognition	Intrusions	Mean
Learning	.32	.60**	.70**	.61**	.70**	.47*	-.18	10.7
Short Delay	.32	.65**	.73*	.52*	.70**	.43*	-.48*	8.9
Long Delay	.61**	.56*	.78**	.30	.78**	.50*	-.05	11.6
Delayed Recogniton	.26	.39	.43*	.45*	.43*	.47*	-.08	11.3
Intrusions	-.26	-.52**	-.44*	-.48*	-.47*	-.47*	-.03	1.2
Mean	.61	.25	54.7	.39	.41	.41	-.27	
SD	.90	1.0	10.9	1.0	1.0	.96	.37	

(Cohen, 1997)

APPENDIX B

TABLE 1, APPENDIX B

Children's Memory Scale (CMS) Research Participants'
Frequencies/Statistics
Child's Age in Months

N	Valid	22
	Missing	0

	Child's Age in Months	Frequency	Percent	Valid Percent	Cumulative Percent
Valid	60	1	4.5	4.5	4.5
	62	1	4.5	4.5	9.1
	71	1	4.5	4.5	13.6
	76	1	4.5	4.5	18.2
	81	1	4.5	4.5	22.7
	84	1	4.5	4.5	27.3
	85	1	4.5	4.5	31.8
	88	1	4.5	4.5	36.4
	92	1	4.5	4.5	40.9
	102	1	4.5	4.5	45.5
	103	1	4.5	4.5	50.0
	105	1	4.5	4.5	54.5
	107	2	9.1	9.1	63.6
	110	1	4.5	4.5	68.2
	115	1	4.5	4.5	72.7
	119	1	4.5	4.5	77.3
	120	1	4.5	4.5	81.8
	122	1	4.5	4.5	86.4
	126	1	4.5	4.5	90.9
	129	1	4.5	4.5	95.5
	132	1	4.5	4.5	100.0
	Total	22	100.0	100.0	

TABLE 2, APPENDIX B

Children's Memory Scale (CMS) Research Participants'
Scores in Comparison to Scores for CMS Standardization Sample
T-Test One-Sample Statistics
N = 22 for All Measures
Bonferroni Correction Was Applied

CMS Index Score	Mean	Std. Deviation	Std. Error Mean
Visual Immediate	98.09	18.39	3.92
Visual Delayed	97.27	14.37	3.06
Verbal Immediate	102.91	14.37	3.06
Verbal Delayed	91.73	12.89	2.75
General Memory	96.95	14.31	3.05
Attention/Concentration	99.95	15.78	3.36
Learning	103.77	15.29	3.26
Delayed Recognition	100.64	19.11	4.07

None of the comparisons met a Bonferroni corrected probability of .05

TABLE 3, APPENDIX B

Children's Memory Scale (CMS) Research Participants' Scores in Comparison to Scores for CMS Standardization Sample
T-Test One-Sample Results
N = 22 and Degrees of Freedom = 21 for All Measures
Bonferroni Correction Was Applied

CMS Index Score	Test Value = 100			95% Confidence Interval of the Difference	
	t	Significance (2-tailed)	Mean Difference	Lower	Upper
Visual Immediate	-.487	.631	-1.91	-10.06	6.25
Visual Delayed	-.890	.383	-2.73	-9.10	3.64
Verbal Immediate	.949	.353	2.91	-3.46	9.28
Verbal Delayed	-3.011	.007	-8.27	-13.99	-2.56
General Memory	-.998	.330	-3.05	-9.39	3.30
Attention/ Concentration	-.014	.989	-4.55E-02	-7.04	6.95
Learning	1.158	.260	3.77	-3.01	10.55
Delayed Recognition	.156	.877	.64	-7.84	9.11

None of the comparisons met a Bonferroni corrected probability of .05

TABLE 4, APPENDIX B

Pearson Correlation
With Bonferroni Correction
Children's Memory Scale (CMS) Indices
N = 22 for all Measures
Significance = 2-tailed for all Measures

	CMS Vis. Imm.	CMS Vis. Del.	CMS Verb. Imm.	CMS Verb. Del.	CMS Gen. Mem.	CMS Att./ Con.	CMS Learn.
CMS Visual Delayed	.760 (*)						
Significance	.000						
CMS Verbal Immediate	.051	.111					
Significance	.823	.623	.				
CMS Verbal Delayed	-.222	-.088	.629				
Significance	.322	.698	.002	.			
CMS General Memory	.700 (*)	.750 (*)	.646	.434			
Significance	.000	.000	.001	.043	.		
CMS Attention/ Concen-tration	.473	.282	.457	.096	.520		
Significance	.026	.203	.033	.670	.013		
CMS Learning	.615	.399	.431	-.203	.529	.699 (*)	
Significance	.002	.066	.045	.364	.011	.000	
CMS Delayed Recognition	-.057	.219	.592	.591	.475	.317	.135
Significance	.802	.328	.004	.004	.025	.151	.548

* Indicates a Bonferroni corrected probability of < .05

TABLE 5, APPENDIX B

Pearson Correlation
With Bonferroni Correction
Children's Memory Scale (CMS) and
Child Behavioral Checklist 4-11 (CBC) Indices
N = 22 for all Measures
Significance = 2-tailed for all Measures

	CMS Vis. Imm.	CMS Vis. Del.	CMS Verb. Imm.	CMS Verb. Del.	CMS Gen. Mem.	CMS Att./ Con.	CMS Learn.	CMS Del. Rec.
CBC Withdrawn	-.338	-.255	.044	.116	-.209	-.310	-.328	.095
Significance	.124	.252	.846	.608	.351	.160	.136	.673
CBC Somatic Complaints	-.017	-.057	-.055	-.022	-.070	-.314	-.169	-.247
Significance	.942	.799	.807	.921	.756	.155	.452	.267
CBC Anxious/ Depressed	-.381	-.339	.065	.257	-.208	-.141	-.308	.122
Significance	.080	.123	.773	.248	.352	.532	.164	.588
CBC Social Problems	.047	-.027	-.059	-.062	-.033	-.188	.083	-.277
Significance	.836	.906	.795	.784	.885	.401	.714	.211
CBC Thought Problems	-.188	-.230	-.224	-.108	-.308	-.242	-.280	-.028
Significance	.402	.302	.316	.632	.163	.279	.207	.900
CBC Attention Problems	-.085	.016	-.131	-.156	-.152	-.098	-.103	-.022
Significance	.708	.944	.561	.488	.501	.663	.647	.922
CBC Delinquent Behavior	-.123	-.284	-.113	.021	-.209	-.234	-.288	-.195
Significance	.587	.201	.618	.927	.350	.294	.194	.385
CBC Aggressive Behavior	.017	.031	.236	.242	.176	.035	-.037	.200
Significance	.939	.892	.290	.278	.434	.876	.871	.372

TABLE 5 CONTINUED, APPENDIX B

Pearson Correlation
With Bonferroni Correction
Children's Memory Scale (CMS) and
Child Behavioral Checklist 4-11 (CBC) Indices
N = 22 for all Measures
Significance = 2-tailed for all Measures

	CMS Vis. Imm.	CMS Vis. Del.	CMS Verb. Imm.	CMS Verb. Del.	CMS Gen. Mem.	CMS Att./ Con.	CMS Learn.	CMS Del. Rec.
CBC Sex Problems	-.143	-.242	-.023	-.245	-.258	.160	.242	-.320
Significance	.525	.277	.919	.273	.246	.478	.279	.146
CBC Other Problems	-.132	-.072	-.153	-.116	-.197	-.306	-.220	-.194
Significance	.558	.751	.496	.606	.380	.166	.326	.387
CBC Internalizing	-.287	-.263	.027	.140	-.190	-.259	-.293	-.010
Significance	.195	.236	.904	.533	.397	.245	.186	.965
CBC Externalizing	-.021	-.057	.147	.189	.074	-.038	-.109	.097
Significance	.925	.800	.515	.399	.744	.866	.630	.669
CBC Total Score	-.138	-.129	-.003	.057	-.110	-.204	-.189	-.029
Significance	.540	.567	.989	.801	.625	.362	.400	.898

None of the correlations met a Bonferroni corrected probability of .05