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Memory Consolidation in Developmental Disorders

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by

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Abstract

The relationship between memory and adaptive functioning was studied in sample of 268 children with attention deficit/hyperactivity disorder (ADHD, *n* = 83), autism spectrum disorder (ASD, *n* = 62), velo-cardio-facial syndrome (VCFS, *n* = 21), and low birth weight (LBW, *n* = 38) and neurotypicals (*n* = 64). Children with ASD and VCFS demonstrated a relative weakness in facial and visual memory, while no between-group differences were found during the auditory verbal learning task of the TOMAL. Learning curve analyses showed that after the first trial of the visual span test, all groups performed at the same level, but the performance of the clinical samples dropped after each subsequent trials. However, during the delayed recall, no between-group differences were evident. On the word memory test, the groups were significantly different after the first trial, but during delayed recall their performance converged. When memory functioning was used to predict academic achievement, TOMAL scores explained 37% of the variance in math scores, 22% in reading, and 13% in spelling scores. The same models did not predict social skills as measured by the CBCL. When age, gender, and FSIQ were added to memory scores to the regression model, the adjusted R^2 value doubled for achievement scores, with IQ clearly driving the age effect. However, IQ was not a significant predictor of social skills. With that criterion, age became the only significant predictor, explaining 39% of the variance. The clinical implications of the findings on diagnostic (non-verbal memory and math seem to be a relative weakness in neurodevelopmental disorders; repeated learning trials may be needed to allow diagnosis-specific deficits to emerge; cognitive variables predict academic, but not social functioning; consolidation could be treated as a separate, emergent variable and normed separately to enhance its diagnostic utility) and treatment (visual cuing may be less effective in ASD and especially VCFS than a verbally mediated one) considerations are discussed.

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Memory: Function, Biology, Phenomenology

Memory as an ability of an organism can be defined as the process of recording experiences and adjusting behavior based on that stored information (McGaugh, 1966). As a mental product, memory can be described as a lasting representation of the perceived reality reflected in cognitive, affective, and behavioral changes (Moscovitch, Chein, Talmi, & Cohn, 2007). From a biological perspective, memory can be defined as a relatively enduring change in the organism's neural architecture and cell physiology as a result of experience (Alberini, 2005; Chklovskii, Mel, & Svoboda, 2004; Dudai, 2000; Haist, Bowden Gore, & Mao, 2001; Malenka & Nicoll, 1999; Trachtenberg et al., 2002).

Phenomenologically, memory is the formidable capacity of the brain to recreate the awareness of a past event in the absence of the external stimulus that led to the original experience (Sheslow & Adams, 2003). Memory preserves the individual's identity by building and maintaining mental connections between the past and present (Reynolds & Bigler, 1994). Memory allows for both a comprehensive perception of reality at any given time and the ability to preserve a personal chronology and mental inventory of past events. As such, it is a building block for higher-order cognitive and motor skills as well as a prerequisite for normal daily functioning. Memory helps organize the sensory input and enables experience to accumulate, thus increasing the individual's problem-solving efficiency over time.

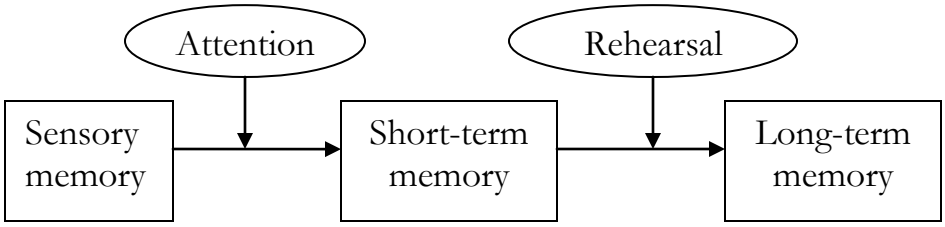
Brief History of Memory Theories

Traditionally, memory is conceptualized in an information-processing framework as a direct one-to-one correspondence between the initial learning and later recall: stimuli are captured, stored, and retrieved in the same format. This positivistic view is questioned by a century of memory research, which made it clear that the original stimulus, its immediate

perception, and the retrieval of its long-term mental representation are far from being equivalent. Accumulating disconfirming evidence led to the formulation of the *process view*, which emphasizes the dynamic nature of memory, recognizing that remembering is a reconstructive mechanism dependent on a cascade of molecular events (Frankland, O'Brien, Ohno, Kirkwood, & Silva, 2001; Izquierdo et al., 2002; Lisman & Morris, 2001), and thus prone to several sources of distortion (Loftus, 2005; Loftus & Davis, 2006; Moscovitch et al., 2007).

The idea of multiple memory systems has also become widely accepted over time. Conceptualizing memory as a unitary system with multiple processes accounting for the multiple facets of memory encoding and retrieval is unnecessarily abstract and detached from the accumulating neurobiological data (Squire, 2004). This distinction is relevant beyond a mere theoretical argument: emerging models of memory can explain an increasingly wider range of experimental and clinical data, thus contributing to our better understanding of this fascinating cognitive domain.

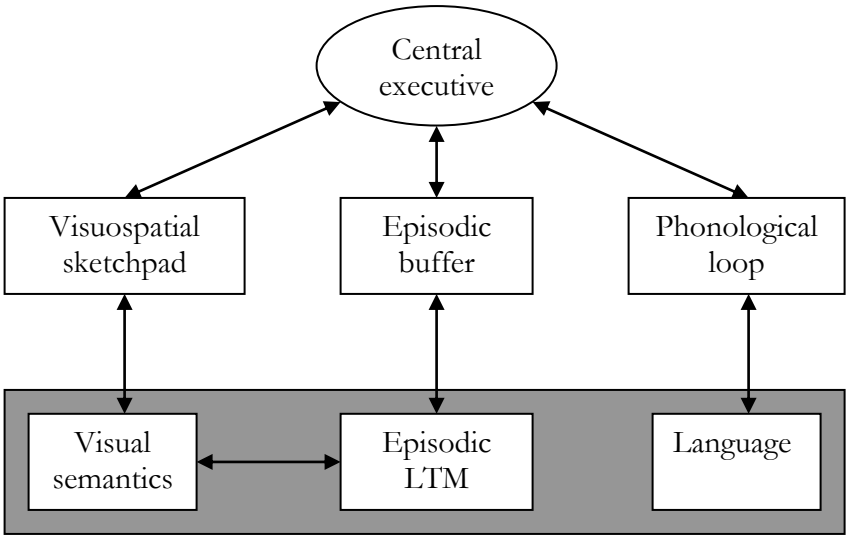
Although it had been introduced previously (Broadbent, 1958; Waugh & Norman, 1965), Atkins and Shiffrin (1968) were the ones who formalized the theory of short-term memory (STM) in its most systematic version.



As Figure 1 illustrates, prior to entering STM, information is first captured in the sensory register, a transient memory system with very limited capacity (can last a maximum of 5-10 seconds). Sensory input is constantly and rapidly deleted to make room for new incoming sensations. The visual sensory system processes the raw material for iconic memories, whereas

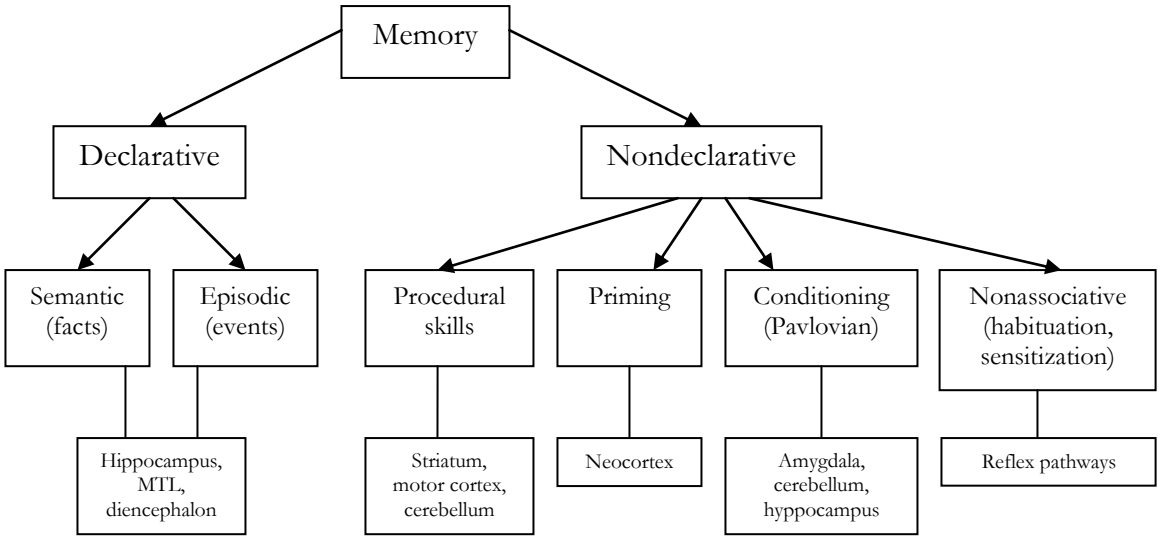
the antecedents of echoic memory are housed in the auditory sensory system. According to their theory, only information consciously attended to enters STM, an intermediate storage system where it can be kept longer through rehearsal. Also referred to as memory span, STM has a capacity limited to about seven elements. Through several repetitions, the information stored in STM is encoded into long-term memory (LTM), a relatively permanent storage system.

Although few researchers accept the STM model as it was originally presented, it has been a powerful influence on subsequent theories of memory functioning (Anderson, 2005). The three-component model of working memory (WM) introduced in 1974 by Baddeley and Hitch builds upon the initial conceptualization of the STM systems, but emphasizes a different function: a temporary storage system that enables the simultaneous access and processing of information (Baddeley, 1992). It preserves and accentuates the distinction between a visual and auditory input and the importance of attention as a mediating process but does not posit that rehearsal is necessary for information to reach LTM. Later, a new component was introduced to improve the explanatory power of the model. As shown by Figure 2, the episodic buffer is conceptualized as an interface between the transient (unshaded boxes) and long-term (shaded box) memory systems (Baddeley, 2000).



Using the high positive correlation among the above discussed measures of memory as an argument, Engle (2002) pointed out that at a practical level, WM is difficult to distinguish from executive attention and is perhaps isomorphic to fluid intelligence. Despite the apparent convergence of these cognitive constructs on current measurement instruments, the models from which they arise do have heuristic value, and thus the potential to advance our understanding of the main underlying phenomena: the encoding, storage, and retrieval of information.

Although the above models of memory offer intuitive and phenomenologically congruent conceptualizations of information storage and processing and are consistent with clinical and experimental data, they are biased toward cognition in humans and are not explicit about the neural substrates underlying their central constructs. In contrast, Squire's (1987) taxonomy includes long-term memory systems that apply to all mammalian species and specifies the locus of each form of memory in the brain (see Figure 3).



By differentiating among salient subtypes of memory based on function and neural correlates, his model allows for more specific predictions in experimental studies of memory. Patterns of interaction among the type of information to be learned, the neurophysiology of the

learner, and type and context of encoding and retrieval have been reliably reported in the memory literature (Alvarez & Squire, 1994; Cipolotti & Bird, 2006; Eichenbaum, Yonelinas, & Ranganath, 2007; Frankland & Bontempi, 2005; Izquierdo et al., 2002; McGaugh, 1966; Meeter & Murre, 2004; Murray & Bussey, 2001; Nadel, Samsonovich, Ryan, & Moscovitch, 2000; Yonelinas et al., 2007).

Consolidation: History and Conceptualization

The temporal nature of memory formation was first systematically studied and described by Müller and Pilzecker (1900). They introduced the term *consolidation* into the literature to label the phenomenon through which newly learned material initially vulnerable to disruption is gradually transferred into a more permanent storage. Some of their methodological innovations (e.g. fixing the number of learning trials, and using percent retention as a dependent measure) are standard psychometric practices today (Lechner, Squire, & Byrne, 1999). Müller and Pilzecker were the first to observe retroactive interference: they noticed that the temporal processing of successive trials of a list learning task was reliably disrupted when a distractor (a different list to memorize) was introduced. This is the first known experiment to demonstrate that silent information processing occurred between the first exposure to a stimulus and a later recall. Essentially, all consolidation paradigms developed since are complex variations of this basic design.

The first attempt to coalesce the consolidation phenomena into a coherent theory was made by Hebb (1949). His dual-trace theory suggested that the perseveration of the initially fragile memory trace over time eventually translates into lasting synaptic changes, which he considered the neural representation of learning. Hebb's structural theory of learning conceptualizes memory as a time-dependent process – a notion that remains the core assumption of modern consolidation hypotheses.

In his seminal paper McGaugh (1966) reviewed the cumulative evidence for time-dependent processes in memory and argued that if the existing body of research continued to expand in the same directions, Lashley's (1930) prophecy that understanding the neuroplasticity underlying learning is beyond human reach could eventually be falsified. His review culminates in a few important conclusions: 1) the trace of an experience is not fixed immediately after the initial exposure – consolidation takes time; 2) consolidation is susceptible to both facilitating and impairing influences; and 3) there are several distinct memory processes.

Consolidation: Neurophysiology

Recent discoveries in molecular biology support the hypothesis of time-dependent changes in cell physiology that translate into functional alterations in the neuron. Nayak and Sikdar (2007) described a form of *molecular memory*: after sustained depolarization, the voltage-gated sodium channels exhibit a *novel* pattern of activation, which ultimately influences the excitability of the neuron. Although it is not yet clear to what extent this phenomenon depends on external input (i.e. learning), it offers a plausible mechanism through which experience is encoded and stored at a basic level. In fact, Gilboa, Chen, and Brenner (2005) showed that ion channel functioning depends on recent activation history by using mathematical modeling of single neuron activity. This interdependence of function and usage in molecular neurophysiology provides indirect evidence that experience modulates an organism's nervous system at the lowest observable levels. Similarly, Eguia, Rabinovich, and Abarbanel (2000) proposed a model of neural connectivity in which multiple signal transmission represents an opportunity for self-correction: neurons give priority to incoming signals that show consistency over time. This preferential processing of electrical impulses results in improving the overall signal-to-noise ratio. Such molecular processes serve as plausible physiological mechanisms underlying the phenomenon of consolidation.

Contemporary research on memory is characterized by a proliferation of novel designs and technologies as well as a convergence of several fields: cognitive sciences, neuropsychology, psychopharmacology, computer data modeling, and neuroimaging. This multidisciplinary approach to memory opened horizons to innovative approaches and created a multitude of methods that can be used to cross-validate findings (Tulving, 1995).

Consolidation has many definitions, each one having a slightly different theoretical emphasis. Although most theorists agree on the basic mechanisms, many of its details are topics for ongoing speculations (Meeter & Murre, 2004). Consolidation has been described as a gradual reorganization of the stored information with an implied shift in the locus of storage from the medial-temporal structures to the neocortex (Medina, Bekinschtein, Cammarota, & Izquierdo, 2008; Murray & Bussey, 2001; Squire & Alvarez, 1995; Squire & Bayley, 2007; Tse et al., 2007) as well as a progressive, domain-specific stabilization of memory traces (Moscovitch et al., 2007; Moscovitch, Nadel, Winocur, Gilboa, & Rosenbaum, 2006; Nadel, Samsonovich, Ryan, & Moscovitch, 2000; Sutherland, Lehmann, Spanswick, Sparks, & Melvin, 2006). That is, the definition could be condensed into a single notion: consolidation is a time-dependent reorganization of the brain structures underlying the ability to store a specific type of information (McGaugh, 2000). This implies that new memories are fragile, vulnerable to forgetting, and are gradually transformed into a more permanent state that is resistant to decay (Frankland & Bontempi, 2005). Central to consolidation theory is the hypothesized underlying neural mechanism through which memory traces are gradually transferred from the hippocampus into the neocortex, to the point where they can be retrieved without hippocampal mediation (Alvarez & Squire, 1994).

The concept that post-experience processes can make memories more resistant to forgetting was first developed as an explanation for the retrograde amnesia gradient named after

Theodule Ribot, who was the first to report that the most recent experiences decay at the highest rate. After a century of evolving controversy about the ontological status and underlying neural mechanisms, the Ribot gradient (the *last in, first out* doctrine) remains the strongest empirical support for consolidation theory (Meeter & Murre, 2004).

Memory consolidation appears to be an adaptive process, as 1) It is present in a variety of species, and 2) It is clearly not a mere compensatory mechanism for a biologically constrained processing speed. Human working memory is capable of holding and simultaneously manipulating large amounts of sensory data instantly. What could then be the function of the slow processing of memories? McGaugh (2000) suggested that the time-dependency of long-term memory formation allows for selection among the myriad of information: previous experience as well as built-in sensitivities modulate the processing of incoming memories, determining which ones are worthy of permanent storage and which ones can left to fade away. A prominent example of such a regulatory mechanism is the role of the amygdala in differentially enabling emotionally arousing events to enter the long-term memory systems. Allowing the significance of an experience to determine its *memory-worthiness* is an effective way of managing an otherwise overwhelming sensory load.

Based on the neurophysiology of learning and the observable changes in behavior resulting from it, it appears that "the brain prefers plasticity at the expense of stability" (Yadin, 2000). In other words, information taken in through the sensory systems are subjected to a selective pressure (i.e. forgetting), and only the most salient ones are allowed to survive (i.e. enter the long-term memory), while the majority of them slide into the oblivion. As such, the process of consolidation represents an opportunity to truncate or undo synaptic changes that encode new learning before irrelevant information becomes permanently stored, and thus overburdening the memory system. On the negative side, this astonishing experience-dependent

flexibility (Chklovskii et al., 2004; Frankland et al., 2001; Trachtenberg et al., 2002) means that initial learning is highly vulnerable to automatic deletion. In other words, the fragility of the memory trace is the cost of a more efficient, malleable storage system (Yadin, 1996).

Consolidation: Timeline

On a surface level, the temporality inherent in consolidation seems to be its most salient dimension. The earliest attempts to develop coherent explanatory models for consolidation proposed a neural mechanism that enabled the stabilization of immediate sensory-based mental representations into long-term memories. Hebb's (1949) dual-trace theory of memory posits that *neurons that fire together, wire together*. In other words, temporary changes in cell-level neurophysiology (biochemical representations of short-term memory) over time evolve into lasting reconfigurations of synaptic connections. Although this seems a plausible explanation with some existing empirical support, Moscovitch et al. (2007) point out that the Hebbian memory trace remains an inferential construct until the change in long-term neural connectivity resulting from consolidation can be observed directly.

Moreover, consolidation theory continues to be vague about the specific time line that new memories follow during their journey from the hippocampus to the neocortex. McGaugh (2000) introduced a three-stage model of consolidation, distinguishing among 1) short-term (seconds to hours), 2) long-term (hours to months) and 3) long-lasting (months to lifetime) memories. This classification system is sound and grounded in empirical research, yet not very specific. Nadel and Moscovitch (1997) criticized the extremely long time frame (as long as 25 years) classical consolidation theory uses, stating that it was hard to see how a cognitive strategy that takes two thirds of the lifetime of the medieval man would serve any meaningful adaptive function.

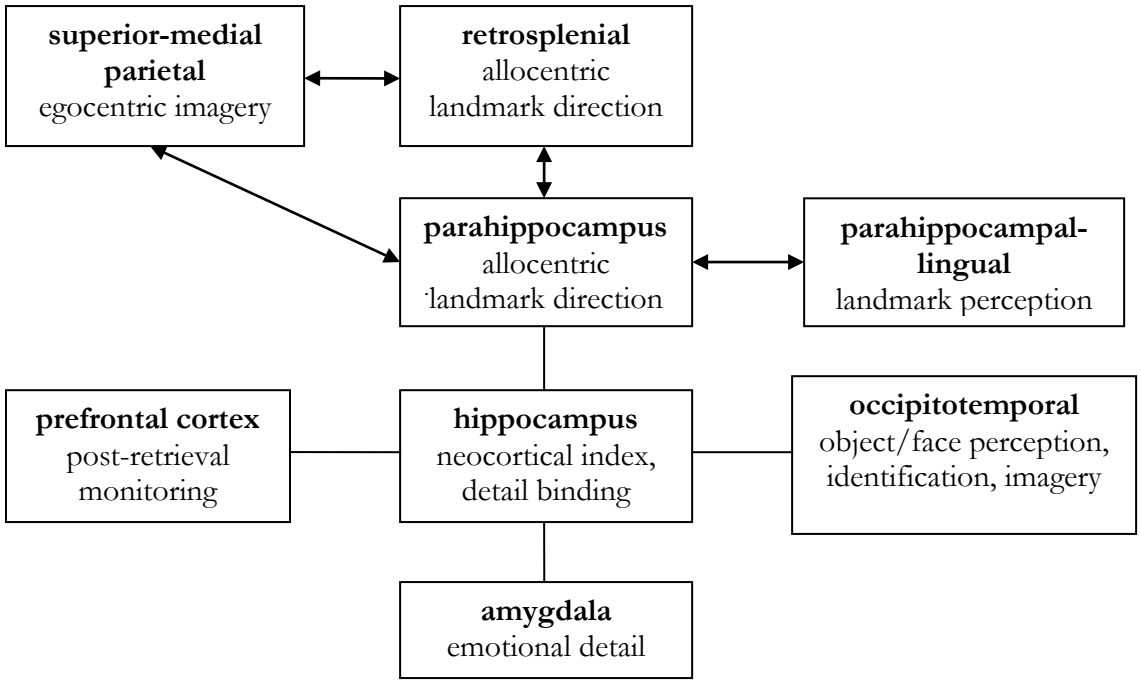
The debate whether stages of consolidation were sequentially linked or independent parallel processes were settled by the mounting evidence that physiologically, there are clearly two distinct phases underlying memory formation. During *cellular consolidation*, transient memory traces are captured by changing the efficiency of local synaptic connectivity, also known as long-term potentiation or depression (Malenka & Nicoll, 1999). During *systems consolidation*, initial memories are transferred into long-term storage requiring large-scale reorganization of the neural networks underlying the targeted memory traces. The physiological marker of this latter process is protein synthesis: selective gene expression is needed to retain information in the long run, but not for the initial learning (Frankland & Bontempi, 2005; Moscovitch et al., 2007; Squire & Bayley, 2007).

Consolidation: Contemporary Theories

A century-old theory invites challenges, revisions, and alternative conceptualizations. Since consolidation theory was first presented, there were several attempts to refine or refute it. Three distinct yet overlapping versions of the basic consolidation theory have emerged: connectionist models, multiple trace theory (MTT), and semantization.

Connectionist models retain the theoretical framework of the original consolidation hypothesis. They postulate a fast-learning hippocampal memory system and a neocortical one that is gradually developed through consolidation. The hypothesized theoretical mechanism is a loosely interpreted rehearsal, through which old representations are strengthened or repaired (McLelland, McNaughton, & O'Reilly, 1995). In this sense, consolidation is the gradual strengthening of memory traces within the neocortex through reactivation initiated in the hippocampus. In computer simulations of this model, consolidation was functionally equivalent to new learning (Meeter & Murre, 2004). A puzzling feature of the model is "runaway consolidation," when occasionally a pattern becomes so strong that it monopolizes

consolidation (Meeter, 2003). To save the model, this phenomenon requires the introduction of new restrictions, such as reduced plasticity of the hippocampus during consolidation or tighter hippocampal control over the process.



The multiple trace theory introduced by Moscovitch and Nadel (1997) presents a competing view on the neurophysiology of consolidation. The core assumption of MTT is that the hippocampal complex continues to have an instrumental role in the retrieval of episodic and autobiographical memories even after they have been transferred to the neocortical memory systems (Moscovitch et al., 2005). They hypothesize that memories are replicated over time in the hippocampus. Older memories have more copies than newer ones; therefore they are easier to retrieve. Phenomenologically, this manifests itself as the Ribot gradient. Figure 4 offers a visual summary of the model proposed by MTT to describe the neural correlates of the system of memory functions implicated in consolidation processes.

Although the MTT explains certain findings better than consolidation theory (a flat gradient in retrograde amnesia; hippocampal activation in the retrieval of old memories observed

in imaging studies by Maguire, Henson, Mummery and Frith [2001] and Ryan et al. [2001]), it also has its own flaws. Specifically, the exact mechanism beyond trace replication is not any clearer than that of consolidation. Moreover, MTT cannot adequately address some data in memory research: the limited hippocampal activation during accessing remote memories (Bontempi et al., 1999; Frankland et al., 2001; Niki & Luo, 2002) or the reverse memory gradient observed in patients with semantic dementia (Graham, 1999) posit serious challenges to the theory.

Finally, semantization offers a conceptual alternative to memory consolidation. According to this theory, remote memories are qualitatively different from recent ones. The former are semantic, whereas the latter are episodic. In other words, memory traces metamorphose from a context-bound state to a language-mediated one, thus becoming part of the individual's general knowledge rather than a homologous representation of an event (Frankland & Bontempi, 2005; Squire & Bayley, 2005). In this view, consolidation is progressive semantization or decontextualization, resulting in the Ribot gradient (Schooler, Shiffrin, & Raajimakers, 2001). Although semantization may be difficult to distinguish from consolidation, they do differ in two ways. First, semantization is thought to result from explicit repetition and relearning, as opposed to the automatic (and largely unexplained) strengthening of memories during consolidation. Second, a central tenet of semantization is the qualitative difference between recent and old memories (episodic and semantic, respectively), an idea that is compatible with consolidation theory but has not been explicitly developed (Meeter & Murre, 2004).

Despite a century of theorizing and research, the exact nature and mechanism of memory consolidation is still poorly understood. The major explanatory systems thus developed (consolidation, MTT, semantization) are essentially speculative, and their central claims are

difficult to test empirically. No single view of consolidation can explain all the phenomena observed in memory research. Moreover, each theory is vague about the time frame within which stages of consolidation occur. However, the rapidly expanding knowledge base in the neurophysiology of learning has the potential to clarify some of the inconsistencies resulting in the highly inferential nature of consolidation theories (Cipolotti & Bird, 2006; Chklovskii et al., 2004; Lisman & Morris, 2001; Sutherland et al., 2006; Tse et al., 2007).

Clinical Implications of Memory Functioning

The intricate neurological mechanism underlying learning and remembering is sensitive to global or partial disruptions. In turn, memory deficits translate into or accompany a divergence of psychiatric problems (Izquierdo et al., 2002). The resulting functional impairment depends on the specificity and severity of the deficit. Understanding the exact role of memory problems in the larger context of the disorder offers a prospect of a more focused, hence more effective intervention (Reynolds & Bigler, 1994). The accurate evaluation of memory functioning is the first step toward this goal and thus often part of a comprehensive clinical assessment of patients with a variety of disorders. Memory impairment of some sort is the defining characteristic of several psychiatric diagnoses; therefore its accurate assessment is critical for a valid diagnosis and treatment planning.

A frequently faced dilemma in diagnosing and treating mental illness is the coexistence of multiple disorders in the same individual. In patients with memory deficit, comorbidity is the rule rather than the exception. This phenomenon is predicted by both biological and cognitive theories. Memory systems are intimately related with other cognitive domains, thus impairment in any given module affects the integrity of not only other types of memory, but overall mental functioning. Although developmental disorders with a strong genetic etiology tend to converge into more or less consistent neurocognitive profiles, there is a high level of interindividual

variability awaiting an explanation. Given that acquired lesions usually affect multiple brain areas in random patterns, the resulting behavioral deficits are also expected to be variable and to overlap with other sets of symptoms.

Static vs. Dynamic Measures of Memory in Clinical Assessment

Considerable research has been done on cognitive profiles associated with different psychiatric diagnoses, in a search for answers to a variety of diagnostic questions. More recently, researchers investigating the consistency of neurocognitive profiles associated with certain disorders pointed out the potential value of memory profiles in diagnostics (Bental & Tirosh, 2007; Dowson et al., 2007; Liddel & Rasmussen, 2005). Although the idea is not new (Kay & Bellak, 1986), the benefit of considering cognitive data in diagnostic decision-making is an active area of research.

Table 1

Selected Tests of Memory

Test	Age Range	Immediate vs. delay	Free recall vs. recognition
Benton Revised Visual Retention Test	8-adulthood	NO	NO
California Verbal Learning Test	5-89 years	YES	YES
Children's Memory Scale	5-16 years	YES	NO
Rey Complex Figure Test	6-89 years	YES	NO
Test of Memory and Learning	5-19 years	YES	NO
Wechsler Memory Scale, 3rd Ed.	16-89 years	YES	YES
Wide Range Assessment of Memory and Learning, 2nd Ed.	5-90 years	YES	YES

Given that consolidation as a process is not universally accepted as a salient cognitive variable that accounts for unique variance in memory functioning, it is not commonly measured by standard assessment instruments. Most tests sample memory performance at two different points in time (immediate vs. delay), others aggregate performance within a larger memory domain and compute an index of acquisition rate, but some tests only measure the target constructs at a single time (Table 1). Similarly, although the distinction between recall and

recognition has been shown to have known neural correlates (Eichenbaum, et al., 2007; Yonelinas et al., 2007) and clinical validity in diagnosing dementia (Arnaiz & Almkrist, 2003; Hutchinson & Mathias, 2007; Levy & Chelune, 2007;), it is not explicitly measured in every test of memory and learning.

The present study hypothesized that the extension of this principle to neurodevelopmental disorders would have clinical utility. In other words, a measure of consolidation would contribute to the current understanding of the cognitive phenotype of certain disorders beyond the general memory indices taken at a single point in time. It was suggested that analyzing the discrepancy between immediate and delayed recall or recognition confers additional specificity to diagnosing and the overall conceptualization of the central deficit in a given diagnostic category. Although to my knowledge this hypothesis has not been tested in the children with attention-deficit/hyperactivity disorder (ADHD), autism spectrum disorder (ASD), velo-cardio-facial syndrome (VCFS), or low birth weight (LBW), there is ample empirical support for its heuristic value in the Alzheimer's disease (AD) literature. In fact, free delayed recall is considered to be the most sensitive measure for diagnosing dementia (Ivanoiu et al., 2005).

Research in geriatric populations has shown that sampling memory performance after a time delay versus immediately following the stimulus is often a better estimator of the presence of absence of AD. Graham et al. (2003) reported that of a long list of neuropsychological tests, the Logical Memory Delay of the WMS-R and a silhouette naming test differentiated between patients with vascular dementia and AD with an 89% accuracy. Likewise, Wicklund, Johnson, Rademaker, Weitner, and Weintraub (2006) found that on Logical Memory Immediate of the WMS-R, there were no differences between patients with frontotemporal dementia and those with AD. However, after the delay, the former category outperformed the latter.

Bondi et al. (2003) found different between-group effects on immediate and delayed recall of list learning tests when normal controls were compared to AD groups. Interestingly, the direction of consolidation was instrument-specific: in the WMS-R, group differences were magnified after delay, whereas the opposite trend was observed using the CVLT. Similarly, the rate of decline over time was found to discriminate between AD and subcortical vascular dementia in the study conducted by Kramer et al. (2004), even though there was no difference in the rate of acquisition between the two groups. These findings resonate with the meta-analysis by Hutchinson and Mathias (2007) who summarized neuropsychological deficits in frontotemporal dementia and AD. They reported invariably larger effect sizes during the delayed recall for each of the measure included in their analysis. These data further argue that memory measures after a delay are more sensitive to between-group differences than measures of immediate recall, even within the same test.

If measures of consolidation have the potential to reliably differentiate among diagnostic categories in one population, it is worth investigating whether the same effect would generalize to other disorders. Although at first it may seem strange to compare disorders typically diagnosed during childhood to dementias, in reality they both are developmental disorders – just on the opposite ends of the life span. Moreover, until a clear biological etiology is established, health care and educational professionals often have to partially rely on neurocognitive measures in diagnosing (and treating) these populations in addition to the Diagnostic and Statistical Manual (DSM) criteria. Further, in the light of the most recent advances in decoding the molecular mechanisms underlying memory, neurocognitive profiles have the potential to guide research on the neurobiology of developmental disorders by providing salient, behaviorally based, and ecologically valid outcome measures.

Operationalizing Consolidation

Although the construct itself seems face-valid (a change in memory performance after a specified time period), the measurement of consolidation raises some important epistemological questions. What is the best way to capture the phenomenon? Are different ways to operationalize consolidation functionally equivalent? Is consolidation a unitary construct, or does it have several distinct, measurable facets, each important in its own way?

From a pragmatic perspective, there are a couple of convenient answers. First, consolidation could be expressed as a difference score between performance at Time 2 (delayed recall) and Time 1 (immediate recall). A negative number means forgetting; a positive one means additional *off-line* learning. Second, the delayed recall score alone could represent consolidation, in an implicit comparison to the immediate recall score.

Consolidation can also be operationalized as learning curve. Although in a multiple-trial learning task the rate of acquisition is conceptualized as the cumulative effect of the repeated exposure to the original stimulus, consolidation is also likely to operate as a parallel process: time elapsed between two trials provides an opportunity to strengthen the memory trace. Given the compound effect of time-dependent neural reorganization occurring after the initial exposure to a stimulus (i.e. consolidation) and its subsequent repetitions, learning curves may not be the best way to capture consolidation in its purest form. However, learning curve analysis could provide important insights into the nature of information processing in the school-aged target population, thus offering practical suggestions about the nature of the core deficits and what type of interventions would produce the best results. Specifically, the selective reminding paradigm employed by certain memory tests has the potential to be used as a "response to intervention" index: How well does the individual benefit from extra instruction compared to controls and other children with the same diagnosis?

Consolidation can also be conceptualized as a forgetting curve: after the last presentation of the original stimulus, performance is sampled during at least one more point in time. Intraindividual differences between immediate and delayed free recall or several delayed recalls (i.e. short and long) can be considered measures of consolidation. This approach has the advantage of eliminating the effect of additional exposure to the initial stimulus: the observed changes in performance can be attributed to neurophysiological changes underlying memory performance (i.e. the classical definition of consolidation).

Although measures of immediate and delayed recall are routinely taken and used in clinical and research settings, there is no formal, universally agreed-upon methodology to operationalize consolidation. Therefore, in this study all the competing conceptualizations will be used and compared to one another. Evaluating the most salient method to operationalize consolidation will thus become an auxiliary goal of the study.

The Clinical Populations and Measures of Memory in the Present Study: A Brief Overview

A review of the recent research on neurocognitive profiles associated with ADHD, ASD, LBW, and VCFS is presented below. These disorders were chosen because they have important similarities and differences; hence the analysis capitalizes on the advantages of a comparative approach. They are all developmental disorders diagnosed in childhood. On the other hand, the core deficit is different in each of them: attention/executive functioning (ADHD), communication disorder (ASD), diffuse, global impairments with high inter-individual variability (LBW), and a combination of well-defined medical problems and variable cognitive deficits with high incidence of a non-verbal learning disability (VCFS). Therefore, these four diagnoses represent a wide spectrum of disorders, providing a theoretical and empirical ground for identifying patterns of similarities and differences in memory functioning – patterns that

could potentially contribute to a better understanding of these populations in themselves and in relation to one another.

Four domains of memory functioning were selected as the main outcome variables: word list, story, design, and facial memory. Two of these domains (list and design learning) are assessed using the selective reminding paradigm, where the examiner repeats the original stimulus for a fixed number of trials, focusing on the items the examinee has previously missed. These domains of learning and remembering are constructs routinely assessed during neuropsychological evaluation. Moreover, they have been shown in the literature to be instrumental in establishing disorder-specific neurocognitive profiles (Antshel, Kates, Roizen, Fremont, & Shprintzen, 2005; Crafword, Kaplan, & Dewey, 2006; Dowson et al., 2007; Geva, Eshel, Leitner, Fattal-Valevski, & Harel, 2006; Muller et al., 2007; Williams, Goldstein, & Minshew, 2005; 2006) or detecting developmental trends in the interaction between diagnosis and symptoms (Happe, Booth, Charlton, & Hughes, 2006; Salsavini et al., 2006). Given the focus of the investigation, a measure of each domain during both immediate and delayed recall will be used in the final data analysis.

Memory consolidation in ADHD. Martinussen, Hayden, Hogg-Johnson, and Tannock (2005) included 26 empirical studies published between 1997 and 2003 in their meta-analysis. In contrast with normal controls, verbal memory systems were less impaired in the ADHD samples (medium effect) than visual-spatial systems (large effect). The results of this study emphasize the importance of examining memory functioning separately across domains. Given the specific focus on consolidation in the present study, papers reporting both immediate and delayed recall data were given preference over those limited to a single time sampling.

On list learning tasks, individuals with ADHD typically performed more poorly than controls during at least one of the measured time points (Leitner, Doniger, Barak, Simon, &

Hausdorff, 2007; Mealer, Morgan, & Luscomb, 1996; Øie, Sundet, & Rund, 1999; Roth et al., 2004; Seidman, Biederman, Weber, Hatch, & Faraone, 1998). Lazar and Frank (1998) found that the ADHD group performed at or below the level of learning disabled and comorbid group on a selective reminding test. However, several studies found no difference between ADHD and control groups on certain aspects of the list learning paradigm (Burden & Mitchell, 2005; Cutting, Koth, Mahone, & Denckla, 2003; Leitner et al., 2007; Øie et al., 1999; Seidman et al., 1998). Data are similarly contradictory regarding retention scores. There is evidence both for (Roth et al., 2004) and against (Burden & Mitchell, 2005; Leitner et al., 2007; Slomine et al., 2005) consolidation. Interestingly, Øie et al. (1999) found no between-group differences in recognition memory, suggesting that the memory deficits observed in ADHD are more pronounced when measuring retrieval rather than familiarity.

Data on story memory are more consistent: the ADHD group was more impaired than controls both at immediate and delayed recall. The performance of both groups deteriorated over time at comparable rates (Johnson et al., 2001; Mealer et al., 1996; Muller et al., 2007). However, in the study by Kaplan, Dewey, Crawford, and Fisher (1998), there was no difference in story memory between the ADHD and the control groups.

Results from studies on visual-spatial memory are again divergent. Some authors found normative performance in children and adults with ADHD (Crawford et al., 2007; Kaplan et al., 1998; Mealer et al., 1996). Others found evidence for a significant impairment (Øie et al., 1999; Shin, Kim, Cho, & Kim, 2003). There was more agreement on patterns of temporal processing. The data presented by Burden and Mitchell (2005) and Johnson et al. (2001) show that initially, the ADHD group performs similarly to controls, but after a delay their score is more impaired. Although the performance for both groups decays over time, individuals with ADHD have a steeper forgetting curve.

The persistent inconsistency in neuropsychological data in this population could be attributed to a number of factors such as sampling error (varying severity of ADHD, pediatric vs. adult samples) and method variance (different tests, research designs, administration sequences, data analytic methods). Some of the findings are in line with dominant conceptualizations of ADHD (the central deficit is inefficient initial encoding); some of them (e.g. normative performance in ADHD) are somewhat surprising. If the pattern of symptom covariation can be established through more research, that could serve two important purposes. First, it would aid diagnostic decisions by providing alternative sources of data to complement the categorical nature of the DSM. Second, it might shape our definition of the disorder by providing insights into its nature.

In summary, on list learning tasks the ADHD groups generally performed more poorly than controls, although exceptions were noted. Learning and forgetting curves were similar, but the clinical group tended to lag behind controls. Similarly, on story memory the ADHD groups generally performed more poorly than controls. There was no evidence for consolidation in either group: performance was poorer during delayed recall. The findings were similar in the visual-spatial domain, except that the two groups were often identical during immediate recall, but the performance of the ADHD groups tended to deteriorate faster over time. The observed deficits seemed to reflect an encoding rather than a retrieval issue.

Memory consolidation in autism. Even though in current clinical conceptualizations memory deficit is not considered a central feature of autism, the hypothesis has been formulated (and refuted) in the past (Minshew & Goldstein, 1993). The salience of memory functioning in diagnosing and treating autism remains equivocal (Ambery, Russel, Perry, Morris, & Murphy, 2006; Blair, Frith, Smith, Abell, & Cipolotti, 2002; Browning, 1983; Dawson et al., 2001; Happe, Booth, Charlton, & Hughes, 2006; Hill & Russel, 2002; Ozonoff & Strayer, 2001; Reed, 2002;

Smith, 1994; Szelag, Kowalska, Galkowski, & Poppel, 2004). However, there are a number of cognitive features that are consistently reported. Short-term and recognition memory, as well as paired associates, were normal in all studies reviewed. On the other hand, recall, story, and spatial working memory as well as list learning were impaired. A lack of modality specificity and organizing strategies was also noted as a pattern that transcended the variability in samples, instruments, and designs.

Research on list learning performance in autism produced fairly consistent results. Most studies did not find significant overall differences between individuals on the ASD and normal controls (Ambery et al., 2006; Minshew & Goldstein, 1993; 2001; Toichi & Kamio, 2002; Williams et al., 2005; 2006). Some of the same studies found significant between-group differences on certain measures, such as the Trial 5 and List B of the California Verbal Learning Test (CVLT, Minshew & Goldstein, 1993), without altering the overall conclusions. Bowler, Gardiner, and Grice (2000) reported better recognition relative to recall performance. These findings suggest that rote memorization in an auditory verbal domain is an intact cognitive skill in autism.

Results regarding temporal processing are divergent. Ambery et al. (2006) found the same rate of acquisition, recall, recognition, and forgetting in ASD and controls. Williams et al. (2005) found slight consolidation, whereas Williams et al. (2006) found slight decay in performance over time in both groups. Data from Minshew and Goldstein (1993) suggest that although individuals with ASD learn and consolidate slower than controls, their performance does improve over time.

Data on story memory are both scarce and contradictory. Minshew and Goldstein (2001) and Williams et al. (2006) found that the ASD group was more impaired than the control group both on immediate and delayed recall. The two groups did not differ in the study by Williams et

al. (2005). Similarly, the evidence is split between consolidation (Williams et al., 2005; 2006) and decay (Minshew & Goldstein, 2001). In all three studies both groups changed in the same direction.

Three studies found equal performance in ASD and control groups in temporal processing of visual-spatial memory (Ambery et al., 2006; Renner, Klinger, & Klinger, 2000; Williams et al., 2006). The data from Minshew and Goldstein (2001) and Williams et al. (2005), however, showed that the ASD groups were more impaired. There was congruence among studies regarding the direction of change over time: performance deteriorated in both groups.

Studies that reported data on facial memory in ASD found that the clinical group performed more poorly at both immediate and delayed recall (Lajiness-O'Neill et al., 2005; Williams et al., 2005). A slight consolidation was observed in controls.

In summary, the group with ASD performed as well as controls on list learning tasks. Both groups consolidated over time; if there was a difference, it was in the favor of the ASD group. In the domain of story memory, findings are more equivocal: in two studies the clinical sample performed more poorly than controls, but one study found no differences between them. Similarly, there was no agreement whether participants improved or decayed over time. In the visual-spatial domain, two-thirds of the comparisons found no differences between ASD groups and controls. In the remaining studies, the ASD groups performed more poorly. All groups decayed over time.

Memory consolidation in low birth weight. Infants born below a body weight of 2,500 grams are classified as having low birth weight (LBW). The group is further divided into very low birth weight (VLBW, below 1,500 grams) and extremely low birth weight (ELBW, below 1,000 grams). There is evidence that suggests that LBW places children are at risk for a host of cognitive impairments, including visuo-spatial working memory (Anderson & Doyle,

2004; Espy et al., 2002; Richards, Hardy, Kuh, & Wadsworth 2001, Rickards et al., 1993; Saavalainen et al., 2007; Woodward, Edgin, Thompson, & Inder, 2005), episodic memory (Briscoe, Gathercole, & Marlow, 2001) and verbal memory (Mikkola et al., 2005). LBW has also been found to negatively affect school achievement, language skills, overall intellectual ability, and various aspects of adaptive functioning (Haack et al., 2002; Indredavik et al., 2005; Rose, Feldman, Jankowski, & Van Rossem, 2005).

Research findings on memory consolidation on list learning tasks are inconclusive. Geva, Eshel, Leitner, Fattal-Valevski, and Harel (2006) reported the same learning curve between a group of children with intrauterine growth restriction (IUGR) and controls matched on gestational age. Similarly, when comparing preterm and full-term children (all with VLBW), Isaacs et al. (2000) found no difference in their performance in either immediate or delayed recall. The results of the latter study might be confounded by low power due to small sample size and the fact that the two groups were identical on one important variable: birth weight. However, Naberhaus et al. (2007) also failed to find significant differences on a list learning task even with robust, matched samples.

Rose and Feldman (1996) compared the cognitive performance of two samples of preterm and full-term children. The clinical group was more impaired on all measured variables, including recognition memory. Their results thus suggest a global deficit that affects multiple areas of cognitive functioning. Haack et al. (1992) collected data on a very large sample of children with VLBW and normal controls using a verbal selective reminding paradigm. They also found significant deficits in the VLBW group in both long-term recall and selective reminding scores; however, the performance discrepancy was less pronounced on the latter task. This finding may suggest an initial encoding deficit that is offset to some extent by focused, successive repetitions of the salient components of the initial learning trial. Interestingly, when

the analysis was restricted to neurologically normal participants, children with VLBW performed as well as controls on long-term recall but showed deficits on selective reminding tasks. This finding can lead to two related conclusions: 1) significant consolidation was masked by other comorbid disorders in the first analysis; and 2) the label 'VLBW' may often be a mere covariate of other neurological conditions rather than being the primary cause of the observed impairments. Similar results were reported by Dewey, Crawford, Creighton, and Sauve (1999). They divided the VLBW sample into a normally developing group (ND) and one with suspected neurodevelopmental disorders (SDD). On the majority of verbal memory tasks, ND performed as well as controls and significantly better than SDD. In other words, VLBW status in itself did not translate into lower scores; impairment was associated with being at risk for neurological dysfunction. In the visual domain differences were generally less pronounced.

The only study identified that reported sequential data on story memory found that initially the clinical group was impaired compared to controls, but the performance gap closed during delayed recall (Isaacs et al., 2000). As mentioned above, this evidence for consolidation in this memory domain should be interpreted with caution given the small samples.

There is more agreement among the studies in the visual-spatial domain: the clinical samples are consistently impaired across a range of instruments and designs. In addition to the fact that the intrauterine growth restriction sample scored below controls on all trials of a complex figure test, their performance continued to decay during the delayed recall, whereas controls consolidated slightly (Geva et al., 2006). Vicari, Caravale, Carlesimo, Casadei, and Allemand (2004) reported a similar pattern when contrasting the performance of preterm children with that of age-matched controls. One noteworthy exception in this study was that both groups performed worse as the latency of recall increased. Nevertheless, the clinical group had a higher rate of decay. Likewise, Isaacs et al. (2000) found a significant difference in

performance in the favor of controls during immediate recall. However, in their study the preterm group improved after a delay and performed as well as the controls. Again, the small sample probably prevents far-reaching generalization, yet these results argue for a compensatory plasticity that manifests differentially over time in the clinical group.

Luciana, Lindeke, Georgieff, Mills, and Nelson (1999) studied the performance of preterms against age-matched controls on a spatial memory span. The clinical group performed consistently below controls, and the discrepancy increased as the task became more complex. However, no significant difference was found on spatial recognition, while the pattern recognition performance of the preterm group continued to be impaired. These findings suggest a task specificity of memory ability in addition to the effect of neurological impairment and that of temporal processing.

The study by Haack et al. (1992) observed essentially the same pattern in the visual-spatial domain as they did with list learning. As a group, children with VLBW performed more poorly than controls on both long-term recall and selective reminding. However, when the data analysis excluded children with known neurological impairments, the difference disappeared. Again, this phenomenon suggests that VLBW as a condition may not be the primary cause of the cognitive deficits. Instead, it might be a risk factor for other disorders (ADHD, LD), the presence of which mediates the constellation of neurocognitive impairments typically observed in this population.

Curtis, Zhuang, Townsend, Hu, and Nelson (2006) reported no difference between a small sample of adolescents born preterm and age-matched controls on either immediate or delayed recall. Performance decayed in both groups over time at comparable rates. This study is an outlier in the distribution of memory consolidation data in the LBW population, as it is the only paper found that failed to detect a difference between the clinical and control group. Given

the small sample size, the negative findings should be interpreted with caution as they may simply be caused by low power. Nevertheless, the study was included in the current analysis because of its sound methodology and because other researchers (Vicari et al., 2004) did find an effect using samples of comparable size.

The study by Curtis et al. (2006) was the only one found that reported time series data on facial memory in LBW children. They found no difference between the clinical and control group during either immediate or delayed recall. Both groups decayed over time, but the performance of preterms deteriorated at a higher rate. Again, as described above, low power affects the generalizability of these findings. Esbjorn, Hansen, Greisen, and Mortensen (2006) studied a cohort of ELBW children and found no memory impairment at age five. Although both the clinical sample and controls performed more poorly after a delay and the rates of decay were similar, immediate and delayed recall data on faces and names were collapsed and only averages were reported. Therefore, the actual trends in consolidation within each subtest cannot be reconstructed.

In summary, findings are inconclusive on list learning performance. However, the evidence suggests that after controlling for the presence of neurological disorders, LBW status alone does not result in impaired scores. In the visual domain, the LBW group showed consistent impairments. Patterns of consolidation remain equivocal.

Memory consolidation in VCSF. Velo-cardio-facial syndrome (VCFS) is a disorder with a known genetic etiology, a 22q11.2 deletion. As such, it provides a unique opportunity to study the link between genetics and clinical prototypes (Woodin et al., 2001; van Amelsvoort et al., 2004). VCFS is accompanied by a distinctive yet broad neurocognitive phenotype: subaverage IQ, better verbal than visual-spatial skills, and poor attention and concentration (Swillen et al., 1997; 1999). VCFS carries an elevated risk for schizophrenia: more than half of

the affected individuals report transient psychotic episodes and a third of them develop schizophrenia later in life (Debbane, Glaser, & Eliez, 2008; Eliez et al., 2001). Certain aspects of the VCFS cognitive profile (facial memory, delayed recall) resemble that of the autism (Lajiness-O'Neill et al., 2005).

VCFS is highly comorbid with learning disabilities. Over 80% of this population qualifies for a general diagnosis, and around half displays the symptom constellation commonly referred to as non-verbal learning disability (NLD; Rourke, 1995): relative impairment in visual-spatial performance in the face of well-preserved verbal abilities, and social skills deficits (Bearden et al., 2001; Swillen et al., 1999). The relatively well-preserved verbal domain in contrast to markedly impaired visual skills supports the NLD classification; however, given the occasional presence of language impairment, researchers are cautious to automatically categorize all instances of VCFS as a form of NLD (Swillen et al., 1999).

Besides consistently observed impairment in facial memory and working memory in general as well as preserved rote verbal memory, Majerus and colleagues (2006, 2007) reported a new feature of VCFS. Although short-term recall of words and non-words was normal, the serial order was impaired. This finding is still awaiting replication before advancing to be a candidate for another identifying clinical feature of VCFS.

Brain imaging studies provide another rich source of data to describe the neuro-behavioral phenotype of VCFS. A series of anatomical abnormalities have been linked to this genotype. Besides an overall lower brain volume (Eliez, Schmitt, White, & Reiss, 2000; Eliez et al., 2001), region-of-interest analyses identified several reliable differences that have the potential to explain the neurological basis of some of the symptoms associated with this condition. Eliez et al. (2000) described an asymmetrical tissue organization in the parietal lobe. This finding was replicated by Barnea-Goraly et al. (2003) who reported aberrant white matter tracts in this area,

as well as reduced white matter anisotropy in the frontal and temporal regions. Eliez et al. (2001) found that children with VCFS had smaller temporal lobe, superior temporal gyrus and hippocampal volumes compared to controls, but commensurate with their overall brain size. On the other hand, researchers found enlarged basal ganglia (Eliez, Barnea-Goraly, Schmitt, Liu, & Reiss, 2002) and right caudate nucleus (Campbell et al., 2006). Aberrant fronto-temporal (Barnea-Goraly et al., 2003; Campbell et al., 2006; van Amelsvoort et al., 2001) and fronto-striatal (Campbell et al., 2006) connectivity also surfaced during imaging studies.

Time series memory data are scarce in the VCSF literature. Moreover, the existing studies are difficult to aggregate given the heterogeneity of samples, instruments, and designs. In addition, many studies are limited by low power due to small samples with high relative variability on the dependent measures. Therefore, population level causal inferences should only be made with caution.

Research on list learning performance in VCSF is inconclusive. The majority of the studies found scores within the normal range, either by comparison to a control group (Henry et al., 2002; Lajiness-O'Neill et al., 2005; Majerus, Glaser, van der Linden, & Eliez, 2006) or to a normative sample (Swillen et al., 1999; Woodin et al., 2001). Van Amelsvoort et al. (2004) contrasted the performance of schizophrenic and non-schizophrenic adults with VCFS on verbal memory and reported equivalent performance. However, both groups were significantly impaired compared to the normal population. When delayed recall data were available, the trend was slight decay over time (van Amelsvoort et al., 2004; Henry et al., 2002; Woodin et al., 2001). In contrast to this body of evidence, Bearden et al. (2001) found that compared to the normative sample, the children with VCFS performed substantially worse during the initial learning trials, but this difference decreased considerably during delayed recall. In other words, consolidation allowed the clinical group to improve performance to near-normal levels over time.

There was more agreement in the story memory domain. All studies identified reported similar performance in the clinical sample and controls. In the majority of groups, performance declined between immediate and delayed recall (Henry et al., 2002; van Amelsvoort et al., 2004).

In the only study found that reported facial memory data, the VCFS group was impaired relative to controls and siblings (Lajiness-O'Neill et al., 2005). This finding may be related to the high rate of NLD profile in the VCFS population.

An important fact must be pointed out regarding the comparisons described above. Many studies used IQ- or disorder-matched controls. Although this practice is justified within the original research question, when taken out of context, the data are hard to interpret and even harder to compare. For example, it is unclear whether negative results from a VCFS sample versus an IQ-matched control group contrast mean that the clinical sample is unimpaired or that IQ mediates memory performance.

In summary, the VCFS group performed as well as controls in the list learning domain. Performance tended to decline in both groups, but it was more pronounced in VCFS. The groups were equal on story memory and declined at comparable rates. In the visual domain there was no evidence of impairment in the VCFS group.

Ecological validity of memory profiles. Most frequently, memory functioning is studied in the context of other cognitive domains (intelligence, attention, executive function). The relationship between memory performance and adaptive functioning (scholastic and occupational achievement, independent living, mental health, social skills) is rarely investigated explicitly. Although the diagnostic utility of memory profiles is independent of their ability to predict functional outcome, they are both important in their own way. Even though empirical measures of adaptive functioning (GPA, income, direct observation of social interaction) would be the preferred method of investigation, the logistical burden of acquiring such data is often too

great to undertake. As a compromise, parent rating scales of children's functioning were used as outcome data in this study. Despite the obvious shortcomings of relying on self-report measures of this kind (dependence in the data set, reporter bias, inter-individual differences in interpreting questionnaire items), it is an important step in asserting the importance of integrating cognitive and functional data. Moreover, given the target population (children and teenagers), it is argued that the perception of the parents and teachers, although perhaps distorted in some ways, is nevertheless a salient and socially valid indicator of the individual's general adaptability. Finally, collateral informants familiar with the child's typical behavior represent a rich source of ecological data that provided a meaningful context in which otherwise ambiguous test scores can be interpreted with more confidence (Lajiness-O'Neill & Beaulieu, 2006).

Little research has been done to systematically investigate the relationship between memory and adaptive functioning. The paucity of correlational analyses focused on cognitive and functional outcome is puzzling given the large amounts of rich clinical data that have been collected and reported in the scientific literature. Researchers frequently collect data on functional outcomes but rarely incorporate them in the main analyses (Dawson, Osterling, Rinaldi, Carver, & McPartland, 2001; Gerdes et al., 1999; Slomine et al., 2005; Niklasson, Rasmussen, Oskarsdottir, & Gillberg, 2001; 2002; Sonuga-Barke, Dalen, Daley, & Remington, 2002; Stevens, Quittner, Zuckerman, & Moore, 2002). Even when functional outcome becomes a variable of interest, subsequent data analysis is limited to describing basic relationships. For example, Crawford, Kaplan, and Dewey (2006) studied the additive effect of cognitive disorders comorbid with ADHD and reported a weak positive correlation between self-reported symptom severity indices and number of diagnoses. Other studies explored more complex patterns of covariance between cognitive and functional data. Dowson et al. (2007) found that self-reported measures of adaptive functioning in a sample of adults with ADHD correlated more strongly

with spatial working memory than data on the same variables provided by collateral informants. In the autism literature Klin et al. (2007) reported a weak relationship between cognitive ability (IQ) and social functioning. The authors explored the interactions among age, symptom severity, and intelligence and found complex relationships they thought to be driven by developmental trajectories. Notably, communication impairment was independent of IQ, and social skills decreased markedly over time. Using a similar between-group comparison design with measures of adaptive functioning as correlates, Happe et al. (2006) found that diagnosis (ADHD or ASD) mediated the relationship between executive and adaptive functioning. Specifically, mental flexibility predicted communication skills in both groups and emotional symptoms in ADHD. All measured aspects of executive functioning were related to social skills in the ASD but not the ADHD group. In general, the association between the cognitive and functional data was stronger in the ASD group.

As the sporadic reports sampled above attest, there seems to be a relationship between cognitive and functional data. Establishing reliable neurocognitive profiles associated with certain disorders has the potential to aid in diagnosing and may allow for increased understanding of the pathophysiology and aberrant neural connectivity in these disorders. Exploring the shared variance between cognitive and adaptive functioning would help determine the salience of cognitive data in conceptualizing the disorder in terms of core deficits, developmental trends, and treatment planning.

Purpose of Study

The current study was designed to investigate two diagnostic issues. First, whether patterns of memory consolidation add discriminative power to static (i.e. one-time) measures of memory in the neurocognitive profiles of the selected disorders and second, to what extent do

measures of memory consolidation predict functional outcome? The present analysis focused on academic achievement and social skills.

Hypotheses

Two major hypotheses were proposed. First, it was hypothesized that measures of memory consolidation would contribute significant information to the neurocognitive profiles of the studied diagnostic groups beyond a single-time sampling of a given memory domain. Specifically, it was predicted that between-group differences will change (in magnitude or direction) as the analysis focuses on the shift from immediate to delayed recall within the same instrument. In other words, measures of consolidation will discriminate among the studied diagnostic categories better than memory performance assessed at one time only.

Second, it was hypothesized that consolidation indices will perform better at predicting functional outcome than static measures of memory. This prediction follows logically from the first one: if measures of consolidation contain unique variance above single-time sampling of memory functioning, they are expected to perform better at accounting for the variability in adaptive functioning as well.

Method

Participants

The project was approved by the Human Subject Review Board (IRB) of both Henry Ford Health System and Eastern Michigan University (see Appendix). Proper ethical and research guidelines were followed throughout the study. Participants with ADHD and ASD were diagnosed using criteria outlined in the DSM-IV (American Psychiatric Association, 1994). ASD also diagnosis was also confirmed with the Childhood Autism Rating Scale (CARS; Schopler, Reichler, DeVellis & Daly, 1980) or the Autism Diagnostic Interview, Revised (ADI-R; Lord, Storoschuck, Rutter & Pickles, 1993) in some cases in addition to DSM criteria. Children were assigned to diagnostic groups based on their primary diagnoses. Children with VCFS were diagnosed using fluorescent in situ hybridization (FISH; Hou, Wang, Tsai, Chou & Wang, 1997). Children with LBW will not be included in the main between-group analyses for two reasons: 1) The high rate of comorbidity with the ADHD would violate the assumption of independence underlying inferential statistical procedures; and 2) LBW is not a diagnostic category per se; rather, it is a condition that increases vulnerability to a host of neurological disorders. Instead of a diagnostic group in its own right, LBW children will serve as clinical controls. Finally, a group of children with no known neurological disorder recruited from scout troops and schools served as normal controls. The clinical data were collected at the medical archives of the Henry Ford Hospital in Detroit, MI, following confidentiality guidelines outlined in the research protocol approved by the IRB.

The final sample contains data on 268 individuals. Age (M = 10.0, SD = 3.6, range: 5-27) is normally distributed with a slight positive skew (1.18) and positive kurtosis (2.4). This latter deviation from the standard normal curve is due to the overrepresentation of young and the presence of a few older children in the current sample compared to the Gaussian distribution

that sharply and symmetrically tapers off at the tails. This deviation reflects the rectangular age distribution in clinical assessment due to the fact that children of all ages are equally likely to be referred to psychological testing. Thus, there is no reason to expect a bell-shaped age distribution in this particular situation. Table 2 lists distributional variables (mean, standard deviation, range, skew and kurtosis) and the results of a one-way ANOVA separately for each subsample. The groups differed on age [$F(4, 263) = 4.94$, $p < .01$], with a medium effect (partial $\eta^2 = .07$). This difference is likely driven by the ADHD group that is slightly younger, and the VCFS group that is older than the grand mean.

Table 2

The Distribution of Age (Years) and Gender within the Diagnostic Groups

	Age								
	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2
Control	64	10.8	4.2	5-25	.67	.47	4.94	.001	.07
ADHD	83	8.8	2.6	5-16	.56	-.37			
ASD	62	10.0	2.8	6-16	.39	-.78			
VCFS	21	12.0	5.7	6-27	1.34	1.21			
LBW	38	9.7	3.5	5-17	.57	-.73			
Total	268	10.0	3.6	5-27	1.18	2.4			

	Gender								
	Males		Females		Within contrasts*		Overall contrast**		
	n	%	*n*	%	χ^2	Φ	χ^2	*p*	Φ
Control	32	50.0	32	50	0.00	.00	29.8	<.001	.33
ADHD	59	71.1	24	28.9	14.8	.42			
ASD	55	88.7	7	11.3	37.2	.77			
VCFS	11	52.4	10	47.6	.05	.05			
LBW	18	47.4	20	52.6	.11	.05			
Total	175	65.3	93	34.7	25.1	.31			

* Tests the null hypothesis that gender is evenly distributed within each diagnostic category: $\chi^2_{crit(df = 1)} = 3.84$, one-tailed

** Tests the null hypothesis that gender is evenly distributed across diagnostic categories

Gender is unevenly distributed in the sample (175 males, 93 females; $\chi^2 = 25.1$, $p < .01$, $\Phi = .31$). This imbalance is driven by the ADHD and ASD groups. Within controls, the VCFS and LBW groups there was an even split between males and females. Again, the observed

deviation from the 50/50 split reflects true population level gender imbalances within ADHD and ASD where males are known to be overrepresented. The diagnosis X gender cross-tabulation was also significant ($\chi^2 = 29.8, p < .01, \Phi = .33$) reflecting group-specific patterns discussed above. The lower half of Table 2 shows descriptive statistics for this main analysis along with the results of individual within-group contrasts.

Materials

Selected neuropsychological standard and raw scores and demographic variables served as dependent variables. Specifically, intellectual (Wechsler Intelligence Scale for Children, Third and Fourth Edition [WISC-III; Wechsler, 1991 and WISC-IV; Wechsler, 2003], Wechsler Abbreviated Scale of Intelligence [WASI; Wechsler, 1999]), memory (Test of Memory and Learning [TOMAL], Reynolds & Bigler, 1994), academic (Woodcock-Johnson, Third Edition [WJ-III; Woodcock, McGrew, & Mather, 2001]), and adaptive functioning (Child Behavior Checklist [CBCL; Achenbach, 1991], Conners Rating Scales-Revised [CRS-R; Conners, 1997]) were assessed. All these are standardized instruments normed on very large (N > 1 000), nationally representative stratified samples with excellent reliability indices (typically in the .90s).

The Wechsler intelligence scales measure several cognitive domains and produce four composite scores: a verbal, visual, working memory and processing speed index. The short form (WASI) was used to estimate the full scale (FSIQ), verbal (VIQ), and performance IQ (PIQ) of the participants in the control group. Distributional variables (mean, standard deviation, range, skew, and kurtosis) as well as the results of a one-way ANOVA are presented separately for each subsample in Table 3. Effect sizes associated with between-group contrasts were quite large (partial η^2 ranging from .20 to .23), suggesting that diagnostic groups differ systematically on IQ. Given this finding, IQ was not used as a covariate for any of the main analyses, as it violates

basic statistical and theoretical assumptions underlying the analysis of covariance as applied to cognitive data (Dennis et al., 2009).

Table 3

The Distribution of IQ Scores within the Diagnostic Groups

					FSIQ				
	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2
Control	62	102.3	13.4	74-136	.52	.60	18.1	<.001	.23
ADHD	78	92.5	18.3	47-132	-.21	-.02			
ASD	59	84.7	20.1	45-149	.50	.50			
VCFS	21	69.1	11.9	40-86	-.69	.00			
LBW	31	84.9	18.6	52-137	.57	.82			
Total	251	90.2	19.5	40-149	.01	-.14			
					VIQ				
	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2
Control	19	112.4	17.0	83-140	-.17	-.61	12.7	<.001	.21
ADHD	79	94.5	17.7	54-129	-.25	-.47			
ASD	46	86.4	19.9	54-135	.08	-.52			
VCFS	21	74.0	12.6	46-91	-.52	-.68			
LBW	32	89.6	20.1	54-146	.59	.71			
Total	197	91.4	20.2	46-146	.01	-.41			
					PIQ				
	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2
Control	19	103.3	12.8	83-140	-.12	-.61	11.9	<.001	.20
ADHD	75	91.2	17.7	48-139	.22	.61			
ASD	46	89.2	20.1	41-151	.36	1.01			
VCFS	21	68.8	10.7	46-84	-.53	-.69			
LBW	31	82.1	17.3	47-118	-.23	.03			
Total	192	88.0	19.1	41-151	.16	.24			

The WJ-III samples the most salient domains of scholastic achievement: single word reading, spelling, and arithmetic. On occasion, where WJ-III scores were not available, they were substituted with scores on the same domains generated by the Wide Range Achievement Test (WRAT, Third Edition; Jastak & Wilkinson, 1995). Distributional variables (mean, standard deviation, range, skew, and kurtosis) as well as the results of the one-way ANOVAs are presented separately for each subsample in Table 4.

Table 4

The Distribution of Achievement Scores within the Diagnostic Groups

	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2	*Post hocs**
Reading										
Control	50	104.8	14.5	72-147	.37	.56	6.40	<.001	.10	1, 2, 3, 4, 6
ADHD	78	95.7	16.1	45-126	-1.02	1.20				
ASD	58	93.5	17.5	45-147	-.38	1.90	Age-corrected ANOVA			
VCFS	21	85.4	18.2	53-116	-.18	-1.00	5.28	<.001	.10	1, 2, 3, 4, 6
LBW	38	93.4	16.3	70-136	.75	.11	Gender-corrected ANOVA			
Total	245	95.8	17.1	45-147	-.33	.82	5.61	<.001	.11	1, 2, 3, 4, 6
Spelling										
Control	49	101.9	12.7	66-126	-.45	.20	8.27	<.001	.12	1, 2, 3, 4, 6, 8
ADHD	82	91.7	16.1	48-129	-.76	.70				
ASD	51	93.3	15.4	63-139	.45	.35	Age-corrected ANOVA			
VCFS	21	82.7	15.5	52-113	-.06	-.25	7.01	<.001	.13	1, 2, 3, 4, 6, 8
LBW	37	87.4	14.6	57-133	.57	1.50	Gender-corrected ANOVA			
Total	240	92.7	15.9	48-139	-.25	.22	6.64	<.001	.12	1, 2, 3, 4, 6, 8
Math										
Control	50	103.4	13.4	63-131	-.20	.66	18.7	<.001	.23	1, 2, 3, 4, 5, 6, 8, 10
ADHD	82	90.3	16.4	45-120	-.48	.04				
ASD	60	83.1	18.4	45-131	.09	-.21	Age-corrected ANOVA			
VCFS	21	69.1	19.5	45-117	.53	.08	15.0	<.001	.23	1, 2, 3, 4, 5, 6, 8, 10
LBW	38	85.9	18.7	50-137	.75	1.29	Gender-corrected ANOVA			
Total	251	88.8	19.2	45-137	-.19	-.21	16.1	<.001	.25	1, 2, 3, 4, 5, 6, 8, 10

* Only contrasts significant at $\alpha = .05$ were recorded (LSD)

1. Control – ADHD
2. Control – ASD
3. Control – VCFS
4. Control – LBW
5. ADHD – ASD
6. ADHD – VCFS
7. ADHD - LBW
8. ASD - VCFS
9. ASD – LBW
10. VCFS - LBW

The TOMAL is a comprehensive assessment instrument tapping into various domains of verbal and visual memory. Each of the core subtests includes an immediate and a delayed recall task, thus allowing the investigation of memory consolidation. This study used four of its subtests: 1) Word Selective Reminding, a free-recall list learning task that requires the examinee to repeat a string of unrelated words; only items missed by the examinee during a given trial are repeated. The subtest continues for 8-12 trials, but stops earlier if mastery (perfect score on two consecutive trials) is achieved. 2) Memory for Stories, a subtest requiring the memorization of a short story. 3) Facial Memory, a subtest that requires the recognition of pictures of individuals representing a wide age range and various ethnic backgrounds. 4) Visual Selective Reminding, a visual analogue of the verbal list learning task that requires the examinee to point at dots on a card in the same sequence the examiner did during a demonstration. The subtest contains 8-12 trials but stops earlier if mastery is achieved.

Data on adaptive functioning were also collected using standardized parent rating scales. The Child Behavior Checklist (CBCL; Achenbach, 1991) was designed to measure behavioral and social competence in children between ages of 1.5 and 18 years. With its robust internal consistency and temporal stability indices (all composites exceeding .89), the CBCL is the gold standard against which other instruments are compared. Inter-rater reliability, as with all rating scales, depends on observer training, and for the CBCL it is lower than ideal (.66) when it is based on between-parents agreement after a two-week latency (Sajatovic & Ramirez, 2003). The low interrater reliability (parent-parent, parent-teacher, teacher-teacher) was apparent during data collection. Therefore, when CBCL data were available from multiple raters, only the form completed by the child's mother was used in the analysis. Although descriptive data as well as between- and within-group contrasts are reported on all CBCL subtests (Tables 10-13) to show

basic relationships between diagnosis and adaptive functioning, only the Social Composite was used as a criterion variable during the regression analyses.

The other sources of data on adaptive functioning were the Conners Rating Scales-Revised (CRS-R; Conners, 1997). These scales were developed to be completed by collateral observers familiar with the child's daily routine (parents, teachers), and measure a variety of cognitive, social, emotional, and behavioral aspects of functioning. They are intended for use with children from ages 6 through 14. Test-retest reliability over a two-week period ranges between .84 and .97. The standard and raw scores of these tests were used as the outcome variables during the regression analyses along with academic achievement scores. Apparent disagreements among different raters on the CRS-R were handled in the same way as described above with the CBCL. Two of the CRS-R subscales were used in the present analyses: the Social Index and the Global Index, which is construed as an overall impairment composite. Tables 10 through 13 display descriptive data as well as between- and within-group contrasts on these two subtests to show basic relationships between diagnosis and adaptive functioning. The Social Index was intended to serve as an alternative measure of the same latent construct to control for instrument-specific biases.

Table 9 lists distributional variables (mean, standard deviation, range, skew, and kurtosis) for academic achievement scores for each subsample as well as between-group contrasts (one-way ANOVAs) with and without age and gender as covariates. Tables 10 through 12 provide the results of between-group contrasts with and without age and gender as covariates on adaptive functioning scores (CBCL and CRS-R). Table 13 lists the results of the within-group contrasts on adaptive functioning scores. ANOVAs are given both with the CBCL Social Competence composite included and excluded, as this index is reverse scored (i.e. lower scores

mean less skill, more impairment); therefore it could confound the interpretation of the overall within-group differences.

Dependent Variables

Memory performance was measured across four domains (story and facial memory, visual span and word list learning) using TOMAL scaled scores during both immediate and delayed recall. Academic achievement was measured across three domains (single word reading, spelling, and mathematics) using WJ-III and WRAT-3 standard scores. Adaptive functioning was measured using the Social Composite of the CBCL as well as the Social and Global Indices of the CRS-R.

Results

Hypothesis 1. Memory Profiles

The first level of analysis used TOMAL scaled scores (SS). This metric uses a standard scale with preset parameters (M = 10.0, SD = 3.0) to correct raw score distributions for age. Essentially, SSs are indices that reference individuals with different demographic characteristics (most often age) against the most appropriate normative sample. Thus, SSs have the same meaning regardless of the actual performance (raw scores) and demographics of the individual; hence they are directly comparable. The analyses performed were one-way ANOVAs with the diagnostic groups as the independent variable and TOMAL scores as dependent variables.

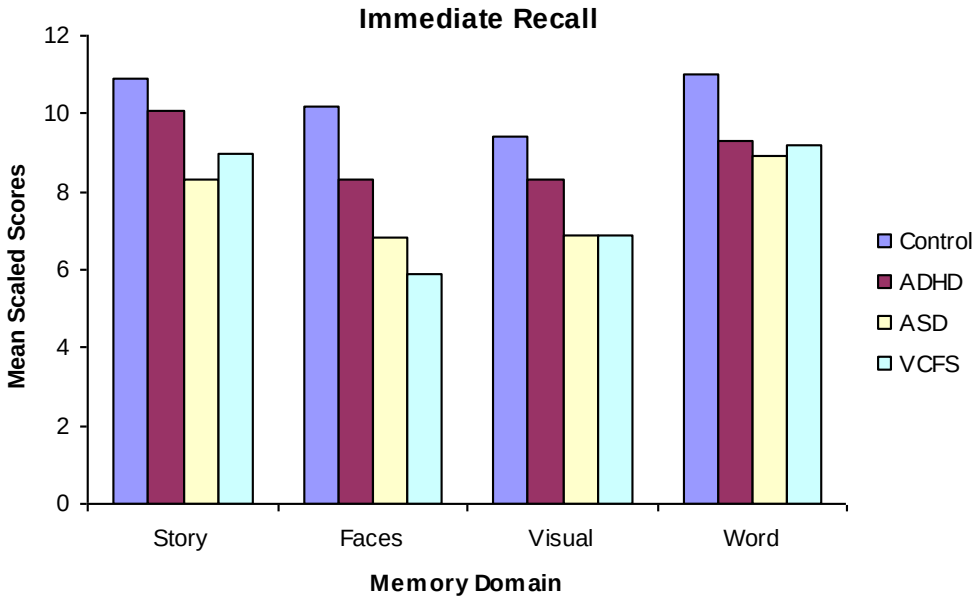
During immediate recall, there was a significant difference among the groups across all four domains of memory: story [$F(4, 221) = 7.75, p < .001$], facial [$F(4, 205) = 17.4, p < .001$], visual [$F(4, 217) = 5.78, p < .001$] and word [$F(4, 256) = 5.80, p < .001$]. On story memory, group membership accounted for 12% of the variance in scores (medium-large effect). The ASD group performed the most poorly, followed by VCFS and LBW. All clinical groups except ADHD recalled significantly fewer details of an orally presented short story than normal controls. The ASD group produced a mean significantly below the mean of the normative sample (M = 10.0), whereas the control mean was superior to that parameter. This finding suggests that within the organizing framework of a narrative, children with ADHD perform at a level comparable to the population average, but the other diagnostic groups are impaired in this memory domain – not just compared to the controls used in this study, but in reference to the normative samples in general.

On a facial memory test, between-group differences were more pronounced: group membership accounted for 25% of the variance in scores (very large effect). This two-fold increase in effect size seems to be driven by the remarkably low performance of the VCFS and

ASD groups. All clinical groups performed significantly more poorly than the controls and below the mean of the normative sample, but better than both VCFS and ASD. This finding suggests that facial memory deficit may be a defining component of these diagnoses and an area of general weakness in developmental disorders.

On a visual span test, all clinical groups were outperformed by controls, although this difference was not statistically significant for the ADHD group. All clinical groups performed significantly below the mean of the normative sample. Group membership accounted for 10% of the variance in scores (medium effect), suggesting that this task may not discriminate well among the clinical groups but seems to be a defining deficit in ASD and VCFS. Also, this task seems to be a general weakness in developmental disorders.

On a word list learning task, the overall effect size was even smaller (group membership accounted for 8% of the variance in scores - medium effect) and apparently driven by controls, who performed at a higher level than the estimated population average, against which all scores are referenced. No difference was found within the clinical groups, although they all produced means significantly below that of the normative sample. This finding suggests that rote verbal learning is relatively well-preserved in developmental disorders; hence it may be concluded that this task does not discriminate well among these diagnostic categories, as it was not sensitive to deviations from estimated overall population means in this sample. Figure 5 provides a visual summary of the analyses presented above.



Statistically controlling for the effect of age or gender did not change the results of between-group comparisons, despite the fact that gender was a significant covariate while comparing story and word memory. This finding may be another piece of evidence for gender-based differences in verbal ability. Overall, however, these variables seem to have a weak relationship with memory performance. Detailed results are summarized in Table 5.

Table 5

The Distribution of TOMAL Scaled Scores during Immediate Recall within the Diagnostic Groups

					Story Memory Immediate					
	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2	*Post hocs***
Control	63	10.9*	2.0	6-16	-.22	.32	7.75	<.001	.12	2, 3, 4, 5
ADHD	63	10.1	2.8	5-16	-.02	-.76				
ASD	54	8.3*	2.9	3-14	.02	-.96	Age-corrected ANOVA			
VCFS	17	9.0	2.8	2-13	-.96	1.07	6.29	<.001	.13	2, 3, 4, 5
LBW	29	9.1	2.9	4-16	.08	-.17	Gender-corrected ANOVA***			
Total	226	9.7	2.8	2-16	-.28	-.36	7.17	<.001	.14	2, 3, 4, 5
					Facial Memory Immediate					
	N	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2	*Post hocs***
Control	63	10.2	2.3	5-16	-.01	-.09	17.4	<.001	.25	1, 2, 3, 4,
ADHD	58	8.4*	2.6	2-15	-.02	.36				5, 6, 9, 10
ASD	47	6.9*	2.7	2-12	.22	-1.02	Age-corrected ANOVA			1, 2, 3, 4,
VCFS	17	5.9*	2.6	1-11	-.10	.08	14.2	<.001	.26	5, 6, 9, 10
LBW	24	8.3*	2.0	5-12	-.29	-.40	Gender-corrected ANOVA			1, 2, 3, 4,
Total	209	8.4*	2.8	1-16	-.10	-.26	14.0	<.001	.26	5, 6, 9, 10
					Visual Memory Immediate					
	N	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2	*Post hocs***
Control	63	9.4	3.0	1-14	-.51	-.13	5.78	<.001	.10	2, 3, 4, 5
ADHD	61	8.3*	3.1	2-16	.04	-.15				
ASD	47	6.9*	3.1	1-15	.58	.14	Age-corrected ANOVA			
VCFS	16	6.9*	3.3	1-13	-.16	-.50	4.61	<.001	.10	2, 3, 4, 5
LBW	25	7.1*	3.3	3-15	.77	.04	Gender-corrected ANOVA			
Total	212	8.1*	3.2	1-16	.40	-.59	4.71	<.001	.10	2, 3, 4, 5
					Word Memory Immediate					
	N	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2	*Post hocs***
Control	63	11.0*	2.4	6-19	.76	1.16	5.80	<.001	.08	1, 2, 3, 4
ADHD	82	9.2*	3.2	1-16	-.14	-.20				
ASD	61	8.9*	2.8	2-14	-.20	-.15	Age-corrected ANOVA			
VCFS	17	9.2	3.3	3-14	-.34	-1.05	4.63	<.001	.08	1, 2, 3, 4
LBW	38	8.7*	3.2	1-16	.47	-.08	Gender-corrected ANOVA***			
Total	261	9.5*	3.1	1-19	-.14	.19	5.93	<.001	.10	1, 2, 3, 4

* Marks a sample mean significantly different from a population mean of 10.0 at $\alpha = .05$ (LSD)

** Only contrasts significant at $\alpha = .05$ were recorded

1. Control – ADHD
2. Control – ASD
3. Control – VCFS
4. Control – LBW
5. ADHD – ASD
6. ADHD – VCFS
7. ADHD - LBW
8. ASD - VCFS
9. ASD – LBW
10. VCFS - LBW

*** Marks a covariate significant at $\alpha = .05$

During delayed recall, there were some notable changes from the patterns described above. On story memory, the between-group contrast was statistically significant [$F(4, 219) = 6.68$, $p < .001$], although all groups obtained lower scores compared to immediate recall. This effect was the strongest among the clinical groups. Group membership accounted for 11% of the variance in scores (medium effect). All diagnostic categories performed significantly below controls, but not differently from each other. They all produced means significantly below that of the normative sample. The ADHD and VCFS groups decayed the most, suggesting that deficits in story memory are more pronounced in developmental disorders after a delay than they are during immediate recall.

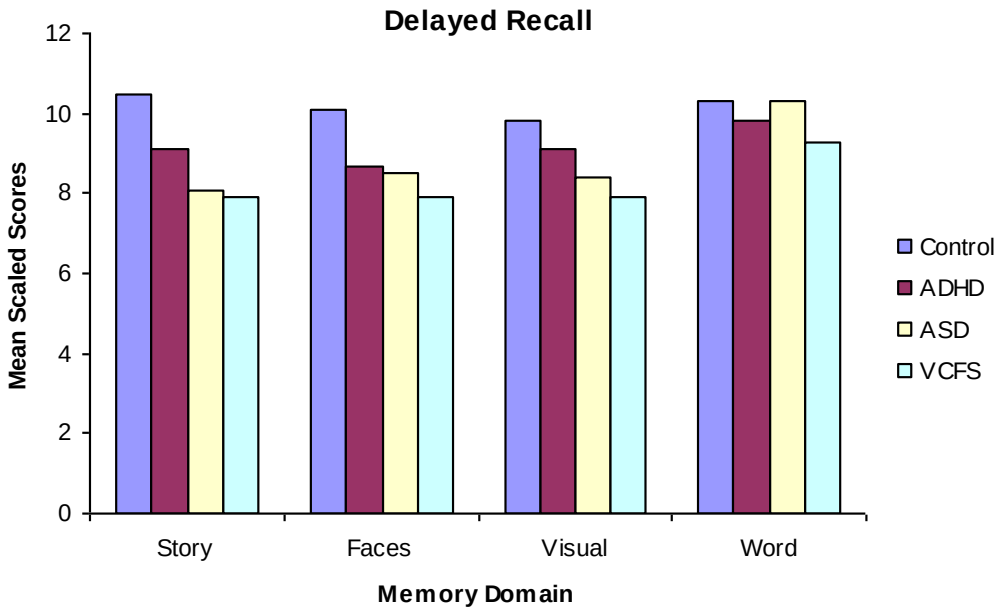
The opposite trend was observed on the facial memory task: all groups performed better than they did during immediate recall. Between-group contrasts were statistically significant: $F(4, 202) = 8.74$, $p < .001$. The performance gap between the ASD and VCFS groups and the controls diminished, causing the overall effect size to drop substantially from the between-group contrasts on immediate recall scores, but still staying large: group membership accounted for 15% of the variance in scores. All groups except LBW performed significantly below controls. All clinical groups performed significantly below the mean of the normative sample. This change after the delay is somewhat difficult to interpret. It is unclear whether it means that children with ASD and VCFS consolidate their facial memory traces more efficiently or that they perform so poorly initially that they are likely to perform better later simply due to chance. Given the binary code (yes/no) unique to the recognition paradigm employed in this subtest, correct answers can be expected to occur at random, in the absence of any meaningful memory traces. In other words, some of this improvement might be accounted for by a regression towards the mean. However, this statistical phenomenon alone cannot explain why these diagnostic groups performed consistently poorly during immediate recall and consistently better during delayed

recall. Overall, consolidation seems to have an equalizing effect: after a time delay, group performances tend to converge, but despite that trend, facial memory remains an area of weakness in developmental disorders.

The trend described above was also observed for visual memory. Between-group contrasts were statistically significant: $F(4, 217) = 5.70$, $p < .001$. All groups except the controls performed better than immediate recall. The performance gap between the ASD and VCFS groups and the controls diminished, although the overall effect size has not changed from immediate recall: group membership still accounted for 10% of the variance in scores (medium effect). This is due mainly to the simultaneous drop in within-group variability. Thus, even though between-group differences decreased, this change was offset by the concurrent shrinkage in standard deviations, producing the same effect size estimate. All clinical groups performed significantly below controls and below the mean of the normative sample. The VCFS group was also inferior to ADHD. It appears that non-verbal visual stimuli take time to form lasting memory traces in both healthy controls and children with a variety of neurological disorders, although time-related changes are much more pronounced in clinical populations.

On word memory, for the first time, no significant difference was found among the groups [$F(4, 254) = 1.23$, $p = .30$], due mostly to gains made by the clinical group and the simultaneous deterioration of the controls' performance. The test used had more than adequate power, so the probability of Type II error is low. Therefore, the negative findings are unlikely to be artifactual but a true reflection of the absence of between-group differences. Further support for the validity of the non-significant result comes from the one-sample *t* tests comparing each group mean to the mean of the normative sample. All these contrasts were non-significant as well, suggesting that all groups produced a mean close to the estimated population average. This equalizing effect is remarkable given the medium effect observed during immediate recall, and

again suggests that memory performance converges after a time delay to the point were controls become undistinguishable from clinical populations. Therefore, these scores have little clinical utility in terms of differential diagnosing. Figure 6 provides a visual summary of the analyses presented above.



Statistically controlling for the effect of age or gender did not change the results of between-group comparisons. Nevertheless, age was a significant covariate during the facial memory contrast. This isolated finding may be an artifactual one: the main effect in that analysis is driven by the low VCFS average. This group also happens to be the most heterogeneous in terms of age; thus a chance covariation between age and memory performance may have triggered the age to become a significant covariate for the entire analysis. Overall, age and gender do not seem to be related to memory performance. Detailed results are presented in Table 6.

Table 6

The Distribution of TOMAL Scaled Scores during Delayed Recall within the Diagnostic Groups

					Story Memory Delayed					
	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2	*Post hocs***
Control	63	10.5	2.2	13-72	.23	.06	6.68	<.001	.11	1, 2, 3, 4
ADHD	62	9.1*	3.0	2-16	-.18	-.05				
ASD	53	8.1*	3.0	2-15	.17	-.40	Age-corrected ANOVA			
VCFS	17	7.9*	3.1	1-13	-.89	.96	5.78	<.001	.12	1, 2, 3, 4, 5
LBW	29	8.5*	3.0	3-15	.09	-.07	Gender-corrected ANOVA			
Total	224	9.1*	2.9	1-18	-.21	.12	6.13	<.001	.12	1, 2, 3, 4, 5
					Facial Memory Delayed					
	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2	*Post hocs***
Control	63	10.1	1.9	7--14	-.09	-.76	8.74	<.001	.15	1, 2, 3, 7, 9, 10
ADHD	57	8.7*	1.8	3-12	-1.04	1.98				
ASD	46	8.5*	1.9	4-13	-.04	-.19	Age-corrected ANOVA***			1, 2, 3, 7, 9, 10
VCFS	17	7.7*	2.4	2-11	-.84	.69	8.36	<.001	.17	
LBW	24	9.6	1.5	7-13	.46	-.35	Gender-corrected ANOVA			1, 2, 3, 7, 9, 10
Total	207	9.1*	2.0	1-14	-.58	1.14	7.15	<.001	.15	
					Visual Memory Delayed					
	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2	*Post hocs***
Control	63	9.8	1.7	3-8	-.16	-.93	5.70	<.001	.10	1, 2, 3, 4, 6
ADHD	61	9.1*	2.0	4-12	-.72	-.31				
ASD	47	8.4*	2.0	3-12	-.32	.14	Age-corrected ANOVA			
VCFS	16	7.9*	2.2	4-12	-.11	-.65	4.62	.001	.10	1, 2, 3, 4, 6
LBW	25	8.4*	2.1	4-11	-.34	-.79	Gender-corrected ANOVA			
Total	212	9.0*	2.0	3-12	-.60	-.39	4.73	<.001	.10	1, 2, 3, 4, 6
					Word Memory Delayed					
	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2	*Post hocs***
Control	63	10.3	1.8	5-13	-1.14	1.17	1.23	.30	.02	
ADHD	81	9.8	2.4	3-13	-1.02	.29				
ASD	60	10.3	1.9	5-13	-.79	.25	Age-corrected ANOVA			
VCFS	17	9.3	3.1	3-13	-.60	-.84	1.30	.27	.03	
LBW	38	10.0	2.3	5-13	-.71	-.54	Gender-corrected ANOVA			
Total	259	10.0	2.2	3-13	-1.00	.43	1.32	.26	.03	

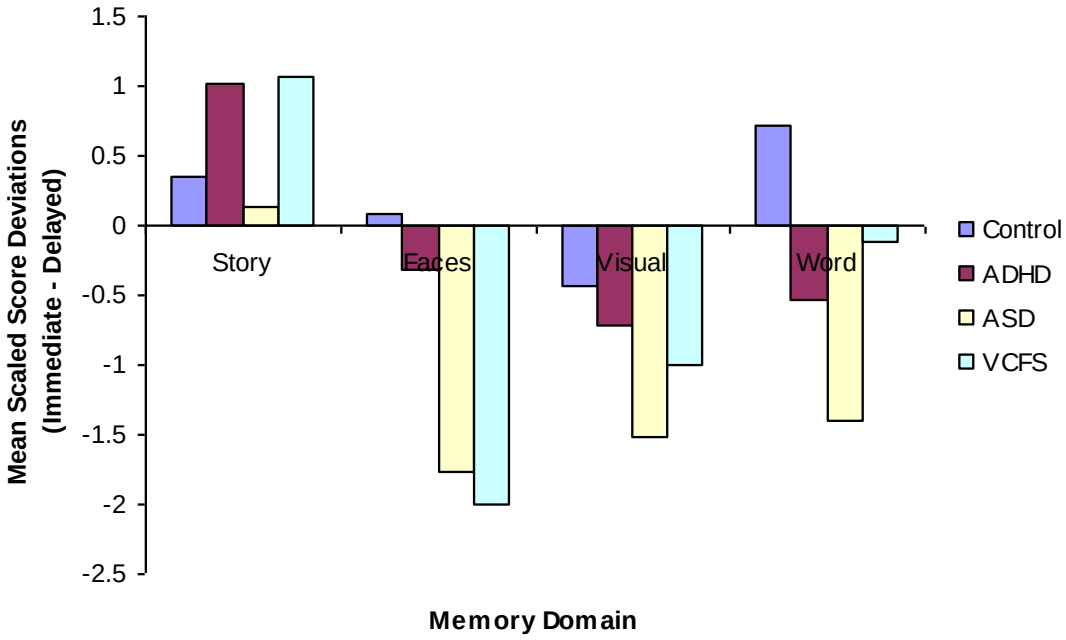
* Marks a sample mean significantly different from a population mean of 10.0 at $\alpha = .05$ (LSD)

** Only contrasts significant at $\alpha = .05$ were recorded

1. Control – ADHD
2. Control – ASD
3. Control – VCFS
4. Control – LBW
5. ADHD – ASD
6. ADHD – VCFS
7. ADHD - LBW
8. ASD - VCFS
9. ASD – LBW
10. VCFS - LBW

*** Marks a covariate significant at $\alpha = .05$

Norm-referenced consolidation was specifically measured by creating a new dependent variable: *scaled score deviations* (SSD) - a change score that was computed by subtracting the delayed recall SSs from the immediate recall SSs. Thus, a positive number means deterioration of performance over time, whereas a negative number means improvement. Using SSD as the outcome measure, no between-group difference was detected on story memory: $F(4, 219) = 2.26$, $p = .06$. Although all mean SSDs were positive, suggesting that all groups retained fewer details of an orally presented narrative as the normative sample, this change was relatively small (less than a third of a standard deviation) and highly variable within the groups. On facial memory, there was a significant between-group difference [$F(4, 202) = 5.14$, $p < .001$] driven by the fact that controls produced a small positive mean SSD, while all clinical groups had higher and negative SSDs. Figure 7 provides a visual summary of the results presented above.



Group membership accounted for 9% of the variance in scores (medium effect). Age was a significant covariate, boosting effect size to 13% of variance in SSDs explained by group membership. On visual span, there was no significant difference on SSDs [$F(4, 217) = 1.89$, $p = .11$], and all groups produced negative means, indicating deterioration in performance from immediate to delayed recall. On word memory, the pattern described with facial memory re-emerged: there was a significant between-group difference [$F(4, 254) = 6.14$, $p < .001$] driven by the fact that controls produced a positive mean SSD, while all clinical groups had higher and negative SSDs. Group membership accounted for 11% of the variance in scores (medium effect).

The analysis of SSDs suggests that norm-referenced consolidation is both domain- and diagnosis-specific. As such, it is more likely to occur on facial and word memory and is generally the least pronounced in controls. Somewhat surprisingly, marked improvement from immediate to delayed recall is more likely to happen in children with developmental disorders than to neurotypicals. As an endnote, the very large relative variability (standard deviation/mean ratio) must be pointed out, which signals highly platokurtic distributions. In other words, a substantial proportion of the data are scattered far away from the mean. The most important implication of this finding is that the mean SSD is not a stable descriptor of overall trends in consolidation, as many participants deviated from the centroid in both directions. The data underlying these analyses are summarized in Table 7.

Table 7

The Distribution of TOMAL Scaled Score Deviations within the Diagnostic Groups

						Story Memory				
	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2	*Post hoc*s*
Control	63	.35	1.37	-3, 5	.50	1.46	2.26	.06	.04	1, 5
ADHD	62	1.02	1.85	-2, 9	1.66	4.97				
ASD	53	.13	2.45	-4, 10	1.24	4.14	Age-corrected ANOVA			
VCFS	17	1.06	1.39	-2, 4	.52	2.23	2.05	.07	.05	5
LBW	29	.62	1.35	-2, 3	.01	-.30	Gender-corrected ANOVA			
Total	224	.57	1.83	-4, 10	1.08	4.74	1.81	.11	.04	1, 5
						Facial Memory				
	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2	*Post hoc*s*
Control	63	.08	2.50	-7, 6	-.27	.54	5.14	.001	.09	2, 3, 4, 5, 6
ADHD	57	-.32	2.40	-6, 5	-.04	-.27				
ASD	46	-1.76	2.59	-8, 3	.14	-.41	Age-corrected ANOVA**			
VCFS	17	-2.00	3.35	-7, 8	1.54	4.34	5.77	<.001	.13	2, 3, 4, 5, 6
LBW	24	-1.25	2.29	-6, 2	-.52	-.59	Gender-corrected ANOVA			
Total	207	-.76	2.65	-8, 8	.04	.11	4.75	<.001	.11	2, 3, 4, 5, 6
						Visual Memory (Immediate – Delayed Recall)				
	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2	*Post hoc*s*
Control	63	-.44	2.26	-7, 3	-.55	.12	1.89	.11	.04	2
ADHD	61	-.71	2.19	-5, 5	.81	.52				
ASD	47	-1.51	1.97	-5, 3	.42	.24	Age-corrected ANOVA			
VCFS	16	-1.00	1.59	-4, 1	-.23	-.88	1.62	.16	.04	2
LBW	25	-1.32	2.93	-8, 5	.18	.60	Gender-corrected ANOVA			
Total	212	-.91	2.25	-8, 5	.15	.30	1.51	.19	.04	2
						Word Memory (Immediate – Delayed Recall)				
	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2	*Post hoc*s*
Control	63	.71	2.76	-4, 8	.95	.23	6.14	<.001	.09	1, 2, 4
ADHD	81	-.53	2.64	-7, 8	.36	.74				
ASD	60	-1.40	2.34	-8, 7	.40	2.51	Age-corrected ANOVA			
VCFS	17	-.12	3.37	-6, 6	.46	.13	5.38	<.001	.10	1, 2, 4
LBW	38	-1.29	2.34	-6, 4	.42	.24	Gender-corrected ANOVA			
Total	259	-.51	2.72	-8, 8	.60	.90	5.57	<.001	.10	1, 2, 4, 5, 8

* Only contrasts significant at $\alpha = .05$ were recorded (LSD)

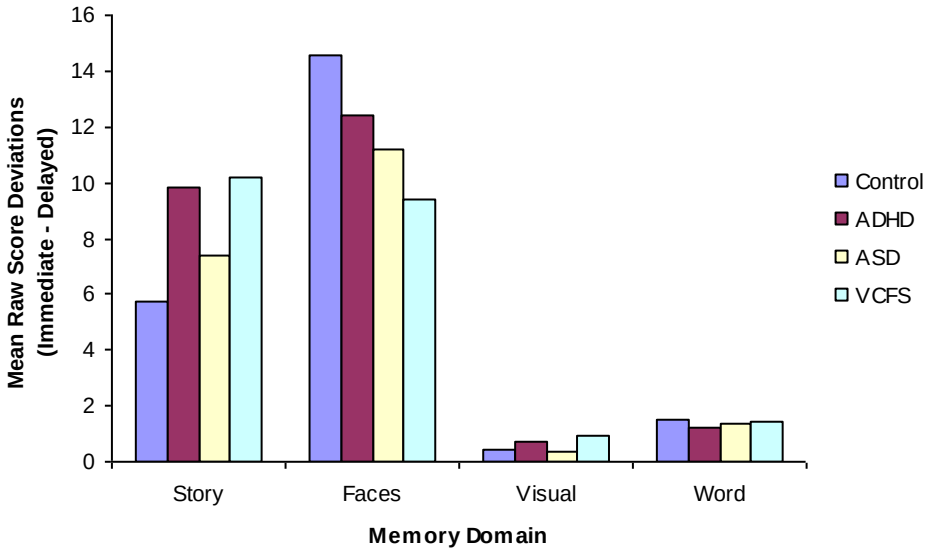
1. Control – ADHD
2. Control – ASD
3. Control – VCFS
4. Control – LBW
5. ADHD – ASD
6. ADHD – VCFS
7. ADHD - LBW
8. ASD - VCFS
9. ASD – LBW
10. VCFS - LBW

* * Marks a covariate significant at $\alpha = .05$

A possible disadvantage of using SSs instead of raw scores (RSs) is that norm referencing can mask trends occurring at the level of absolute values. For example, if the performance of a sample of individuals is measured at SSs level, it may be noted that during delayed recall they obtained higher scores than they did during immediate recall. This could lead to the interpretation that consolidation took place, where in fact it may just be that they *forgot less* than the normative sample, but still performed more poorly at a RS level after a delay. In other words, SSs and RSs capture different, equally relevant aspects of memory functioning. Therefore, the above analyses were repeated using RSs.

Mirroring SSDs, a new dependent variable was created: *raw score deviations* (RSD) - a change score that was computed by subtracting the delayed recall RSs from the immediate recall RSs (or the Trial 8 score in case of Visual and Word Selective Reminding). Thus, a positive number means deterioration of performance over time, whereas a negative number means improvement. RSDs have the advantage of being free of (most often adaptive) distortions that occur during re-scaling, and therefore offers a pure measure of time-related changes in performance. Using RSDs, the only significant difference was found on facial memory [$F(3, 134) = 7.52$, $p < .001$], where group membership accounted for 14% of the variance in scores (large effect). All groups lost less information than controls after a delay. This matched the pattern described with SSDs: although controls outperform the clinical groups during both immediate and delayed recall, they lose the most information over time. Age was a significant covariate, boosting effect size to 22% of variance in SSDs explained by group membership (very large effect). A quick analysis of the group means reveals that on average, subjects performed more poorly after a delay in every memory domain, but this effect was the least pronounced in visual span. Just as it was mentioned with SSDs, the very large relative variability (standard deviation-to-mean ratio) signals highly platokurtic distributions. In other words, a substantial proportion

of the data are scattered far away from the mean. The most important implication of this finding is that the mean RSD is not a reliable representation of overall trends in consolidation, as many participants deviated from the centroid in both directions. The data underlying these analyses are summarized in Table 8 and Figure 8.



This finding provides an important contrast to the SSD analysis, which suggested that on several occasions, certain groups made gains after a delay. The RSD analysis creates a point of reference for those results and clarifies that those apparent gains are to be interpreted in the context of re-scaling. In other words, change scores expressed in SSDs speak to performance compared to the normative sample, which is arguably the clinically most salient reference. However, at the same time they may create the appearance of performance changes in real sense (i.e. higher or lower absolute scores at delayed recall), which is not the case. Thus, a simultaneous analysis of both SDSs and RSDs is needed to accurately describe and interpret patterns of changes in memory performance over time.

Table 8

The Distribution of TOMAL Raw Score Deviations within the Diagnostic Groups

					Story Memory (Immediate – Delayed)					
	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2	*Post hoc*s*
Control	37	5.8	5.35	-3, 21	.82	.66	2.50	.06	.05	1, 3
ADHD	61	9.8	8.00	-2, 41	1.48	3.23	Age-corrected ANOVA			
ASD	36	7.4	10.40	-7, 46	1.78	4.51	1.87	.12	.05	1, 3
VCFS	17	10.2	6.29	-2, 24	.55	1.18	Gender-corrected ANOVA			
Total	151	8.29	8.08	-7, 46	1.54	4.18	1.91	.11	.05	1
					Facial Memory (Immediate – Delayed)					
	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2	*Post hoc*s*
Control	37	14.6	3.72	7-22	.27	-.09	7.52	<.001	.14	1, 2, 3, 5
ADHD	56	12.4	3.71	2, 20	-.20	.43	Age-corrected ANOVA**			
ASD	29	11.2	4.34	5, 21	.43	-.52	9.65	<.001	.22	2, 3, 4, 5
VCFS	17	9.4	5.17	0-24	1.04	3.44	Gender-corrected ANOVA			
Total	139	12.4	4.32	0-24	4.32	.19	5.92	<.001	.15	1, 2, 3, 5
					Visual Memory (Trial 8 – Delayed Recall)					
	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2	*Post hoc*s*
Control	38	.40	1.10	-2, 3	.41	.88	1.31	.27	.03	
ADHD	61	.74	1.30	-3, 5	.60	1.92	Age-corrected ANOVA			
ASD	30	.33	1.56	-3, 4	.39	.84	1.03	.39	.03	
VCFS	15	.93	1.03	0, 3	1.05	.32	Gender-corrected ANOVA			
Total	144	.58	1.29	-3, 5	.44	1.34	1.06	.38	.03	
					Word Memory (Trial 8 – Delayed Recall)					
	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2	*Post hoc*s*
Control	38	1.50	1.98	-2, 6	.55	-.04	.16	.93	.00	
ADHD	81	1.23	2.23	-2, 9	1.31	2.12	Age-corrected ANOVA			
ASD	41	1.34	1.92	-2, 7	.87	.40	.50	.74	.01	
VCFS	17	1.47	2.58	-2, 8	1.22	1.21	Gender-corrected ANOVA			
Total	177	1.34	2.13	-2, 9	1.07	1.24	.13	.97	.00	

* Only contrasts significant at $\alpha = .05$ were recorded

1. Control – ADHD
2. Control – ASD
3. Control – VCFS
4. ADHD – ASD
5. ADHD – VCFS
6. ASD - VCFS

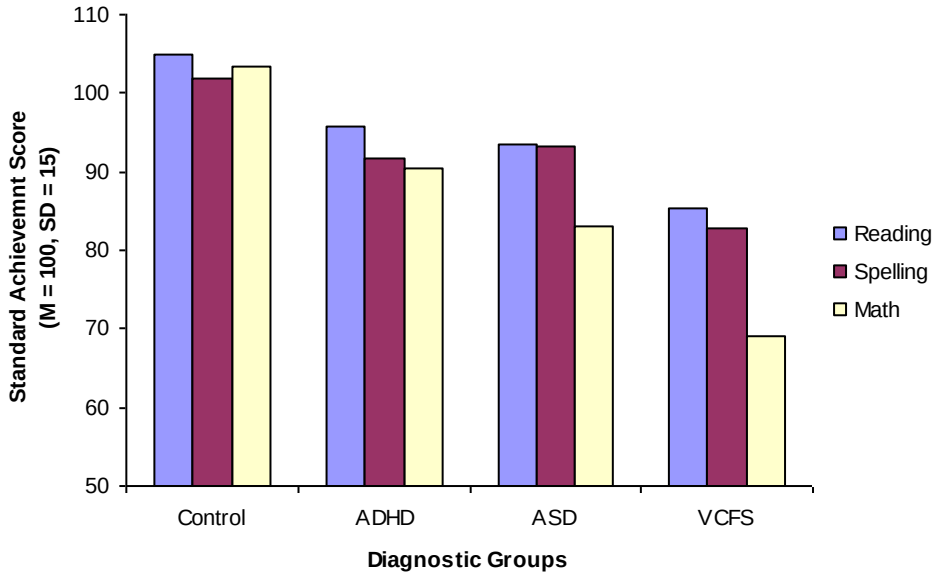
* * Marks a covariate significant at $\alpha = .05$

Since all clinical groups changed in the same direction, the direction of consolidation *per se* is not useful in differential diagnosis. Also, given the high variability in both SSDs and RSDs, the average deviation score is not a good descriptor of overall trends. However, consolidation could be treated as an emergent variable of its own merit, and normative distributions could be developed for change scores, thus allowing comparing an individual's consolidation to that observed in a selected target group. In other words, deviation scores themselves could be scaled. The relative standing on this variable may have the potential to refine the existing cognitive profiles and enhance confidence in differential diagnosis. If consolidation is a salient variable itself, it should be normed separately to allow for a more meaningful nomothetic referencing, which could improve discriminant validity. This method could reduce within-group variability by age-correcting deviation scores, or at the very least standardize deviation scores to make them readily interpretable. The complexity of the present data analysis and interpretation of the multiple within- and between-group comparisons with often diverging conclusion serves as a compelling argument for the practical – and potentially clinical – utility of such norm-referencing and standardization effort.

Hypothesis 2. Memory as Predictor of Adaptive Functioning

Academic achievement. One-way ANOVAs with diagnostic groups as independent variable and scaled scores on the WJ-III as dependent variables revealed that clinical groups were significantly inferior to controls in all assessed domains. The effect size was large to moderately large and independent of the effect of age and gender, two commonly used covariates in cognitive data. In the domain of reading [$F(4, 240) = 6.40$, $p < .001$, partial $\eta^2 = .10$], the VCFS and LBW groups performed the most poorly (Low Average). The other clinical groups all produced means in the Average range. The same pattern was observed with spelling scores [$F(4, 235) = 8.27$, $p < .001$, partial $\eta^2 = .12$]. The between-group differences were

magnified within the domain of mathematics [$F(4, 246) = 18.7$, $p < .001$, partial $\eta^2 = .23$]. The average of the VCFS group fell in the Extremely Low range, with the LBW and ASD in the Low Average range. The ADHD group produced an average at the lower end of the Average range. The effect size was nearly double that of the prior two analyses. Figure 9 provides a visual summary of the group means.



In a search for a potential background variable, the effect of IQ was removed to statistically control for its potential mediating effects: $F(5, 227) = 60.5$, $p < .001$, partial $\eta^2 = .57$. Surprisingly, the main effect was even larger after the effect of IQ was accounted for, suggesting that the observed between-group differences are not driven by general cognitive ability. Rather, they may be specific manifestations of the neurocognitive phenotypes associated with these diagnoses. The same finding was replicated with reading [$F(5, 222) = 28.3$, $p < .001$, partial $\eta^2 = .39$] and spelling [$F(5, 219) = 19.7$, $p < .001$, partial $\eta^2 = .31$] scores. Details of these analyses are presented in Table 9.

Table 9

The Distribution of Academic Achievement within the Diagnostic Groups

					Reading					
	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2	*Post hoc*s*
Control	50	104.8	14.5	72-147	.37	.56	6.40	<.001	.10	1, 2, 3, 4,
ADHD	78	95.7	16.1	45-126	-1.02	1.20				6
ASD	58	93.5	17.5	45-147	-.38	1.90	Age-corrected ANOVA			1, 2, 3, 4,
VCFS	21	85.4	18.2	53-116	-.18	-1.00	5.28	<.001	.10	6
LBW	38	93.4	16.3	70-136	.75	.11	Gender-corrected ANOVA			1, 2, 3, 4,
Total	245	95.8	17.1	45-147	-.33	.82	5.61	<.001	.11	6
					Spelling					
	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2	1, 2, 3, 4,
Control	49	101.9	12.7	66-126	-.45	.20	8.27	<.001	.12	6, 8
ADHD	82	91.7	16.1	48-129	-.76	.70				
ASD	51	93.3	15.4	63-139	.45	.35	Age-corrected ANOVA			1, 2, 3, 4,
VCFS	21	82.7	15.5	52-113	-.06	-.25	7.01	<.001	.13	6, 8
LBW	37	87.4	14.6	57-133	.57	1.50	Gender-corrected ANOVA			1, 2, 3, 4,
Total	240	92.7	15.9	48-139	-.25	.22	6.64	<.001	.12	6, 8
					Math					
	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2	1, 2, 3, 4,
Control	50	103.4	13.4	63-131	-.20	.66	18.7	<.001	.23	5, 6, 8, 10
ADHD	82	90.3	16.4	45-120	-.48	.04				
ASD	60	83.1	18.4	45-131	.09	-.21	Age-corrected ANOVA			1, 2, 3, 4,
VCFS	21	69.1	19.5	45-117	.53	.08	15.0	<.001	.23	5, 6, 8, 10
LBW	38	85.9	18.7	50-137	.75	1.29	Gender-corrected ANOVA**			1, 2, 3, 4,
Total	251	88.8	19.2	45-137	-.19	-.21	16.1	<.001	.25	5, 6, 8, 10
					ACHIEVEMENT Composite					
	n	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	Partial η^2	1, 2, 3, 4,
Control	49	103.0	11.2	67-121	.39	.88	12.7	<.001	.18	6, 8, 10
ADHD	77	93.2	14.4	47-117	-1.10	1.59				
ASD	48	89.9	14.0	64-133	.37	.90	Age-corrected ANOVA			
VCFS	21	79.1	16.5	50-115	.07	-.20	10.3	<.001	.19	1, 2, 3, 4, 6, 8, 10
LBW	37	89.1	15.1	66-135	.88	1.13	Gender-corrected ANOVA			1, 2, 3, 4,
Total	232	92.7	15.4	47-135	-.34	.34	10.5	<.001	.19	6, 8, 10

* Only contrasts significant at $\alpha = .05$ were recorded

1. Control – ADHD
2. Control – ASD
3. Control – VCFS
4. Control – LBW
5. ADHD – ASD
6. ADHD – VCFS
7. ADHD - LBW
8. ASD - VCFS
9. ASD – LBW
10. VCFS – LBW

** Marks a covariate significant at $\alpha = .05$

The above findings seem to converge in a couple of main conclusions. First, between-group differences among the studied diagnostic categories are the least likely to emerge in reading scores, followed by spelling. Conversely, of the three main domains of academic functioning, math scores have the most potential to differentiate among these diagnoses. Second, children with VCFS seem to be at a high risk for low scholastic achievement across all three measured domain, but especially in math. Third, low achievement scores in the clinical groups cannot be explained by IQ alone. On the contrary, controlling for the effect of intellectual functioning accentuated the between-group differences.

Table 10

Between-group Contrasts on Adaptive Functioning Comparing the ADHD, ASD and ADHD Samples

Measure	Scale	*F*	*p*	Partial η^2	*Post hoc* contrasts*
CBCL	Withdrawn	14.1	<.001	.16	1, 3
	Somatic Complaints	.49	.62	.01	
	Anxious/Depressed	3.35	.04	.04	1, 3
	Social Problems	7.30	.001	.09	1
	Thought Problems	12.1	<.001	.14	1
	Attention Problems	2.34	.10	.03	
	Delinquent Behavior	1.23	.30	.02	
	Aggression	.08	.92	.00	
	Social Competence	4.91	.01	.08	1, 2
Conners**	Social Index	15.3	<.001	.25	1
	Global Index	.56	.46	.01	

* Only contrasts significant at α = .05 were recorded
1. ADHD – ASD
2. ADHD – VCFS
3. ASD – VCFS
** No data on the VCFS group

Psychopathology. One-way ANOVAs were performed to compare the three main clinical groups (ADHD, ASD, and VCFS) on several measures of psychopathology. Significant differences emerged on three of the eight commonly assessed domains of psychopathology on the CBCL: depression/anxiety [$F(2, 147) = 14.1, p < .001$, partial $\eta^2 = .16$] social impairment [$F(2, 147) = 3.35, p = .04$, partial $\eta^2 = .04$], and thought problems [$F(2, 147) = 12.1, p < .001$,

partial η^2 = .14]. On Conners' Social Index, a structured parent-report on selected child behaviors, data were available only on the ADHD and ASD groups. The contrast was significant [$F(1, 48) = 15.3$, $p < .001$, partial $\eta^2 = .25$], with a very large effect. This was expected given that social problems are the defining characteristics of ASD and to a lesser extent of VCFS and ADHD. Results are summarized in Table 10. The between-group comparisons were recomputed with age as a covariate. The overall findings remained the same, but the effect sizes increased, suggesting that age is a significant moderator of the between-group differences. Results are summarized below in Table 11.

Table 11

Between-group Contrasts on Adaptive Functioning Comparing the ADHD, ASD and ADHD Samples with Age as a Covariate

Measure	Scale	*F*	*p*	Partial η^2	*Post hoc* contrasts*
CBCL	Withdrawn	10.0	<.001	.17	1, 3
	Somatic Complaints	.67	.57	.01	
	Anxious/Depressed	2.50	.06	.05	1, 3
	Social Problems	7.60	.001	.14	1
	Thought Problems	9.67	<.001	.17	1, 3
	Attention Problems	2.04	.11	.04	
	Delinquent Behavior	1.23	.30	.02	
	Aggression	.08	.92	.00	
	Social Competence	4.90	.01	.11	1
Conners**	Social Index	10.7	<.001	.32	1
	Global Index	.98	.38	.04	

* Only contrasts significant at α = .05 were recorded
1. ADHD – ASD
2. ADHD – VCFS
3. ASD – VCFS

** No data on the VCFS group

The between-group comparisons were then recomputed with gender as a covariate. The most noticeable change was that the inattention scale of the CBCL became a significant contrast, suggesting that gender is a significant moderator of the between-group differences. Results are summarized in Table 12.

Table 12

Between-group Contrasts on Adaptive Functioning Comparing the ADHD, ASD and ADHD Samples with Gender as a Covariate

Measure	Scale	*F*	*p*	Partial η^2	*Post hoc* contrasts*
CBCL	Withdrawn	9.30	<.001	.16	1, 3
	Somatic Complaints	.35	.79	.01	
	Anxious/Depressed	2.89	.04	.06	
	Social Problems	5.35	<.01	.10	1
	Thought Problems	8.13	<.001	.14	1
	Attention Problems	2.79	.04	.06	1
	Delinquent Behavior	1.23	.30	.02	
	Aggression	.08	.97	.00	
	Social Competence	3.31	.02	.08	1, 2
Conners**	Social Index	12.4	<.001	.36	1
	Global Index	1.76	.18	.07	

* Only contrasts significant at α = .05 were recorded
1. ADHD – ASD
2. ADHD – VCFS
3. ASD – VCFS

** No data on the VCFS group

To assess the relative standing of each diagnostic group on different subscales of psychopathology, repeated measures ANOVAs were performed. The test was significant within all three groups, suggesting a differential level of impairment across the eight subscales of the CBCL. Within the ADHD group [$F(7, 79) = 21.4$, $p < .001$, partial $\eta^2 = .21$], the most severe symptoms were reported to be inattention (mean T-score = 68.9, SD = 9.6) and social problems ($M = 62.1$, $SD = 11.5$), followed by aggression ($M = 61.0$, $SD = 12.2$). Within the ASD group [$F(7, 49) = 23.6$, $p < .001$, partial $\eta^2 = .33$], the highest scores were obtained on the attention ($M = 72.8$, $SD = 10.6$) and social problems ($M = 69.7$, $SD = 10.4$) scale, followed by the internalizing ($M = 68.7$, $SD = 11.6$) scale. It is somewhat surprising that parents report attention problems to be the most severe symptoms in this population. In fact, the ASD group obtained a higher symptom severity index than the ADHD group on the Attention Problems scale of CBCL. This difference was statistically significant: $t(128) = 2.17$, $p = .03$. One explanation may

be that parents misperceive the manifestation of ASD as primarily an attention deficit. This phenomenon was also observed in the VCFS group [$F(7, 16) = 7.61$, $p < .001$, partial $\eta^2 = .32$], where attention problems ($M = 69.4$, $SD = 12.0$) were rated as the most pronounced symptom, followed by social problems ($M = 66.2$, $SD = 12.9$) and thought problems ($M = 63.4$, $SD = 10.2$). Table 13 provides a detailed summary of the analyses described above. Every mean (across domains and functioning and diagnostic groups) was significantly different from the population mean of 50 ($\alpha = .01$), indicating the clinical populations are reported to have elevations on all symptom scales of the CBCL and CRS-R. Results are summarized in Table 13.

Table 13

The Distribution of Adaptive Functioning within the Clinical Groups

		ADHD Group								
Measure	Scale	*n*	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	η^2
CBCL	Withdrawn	80	58.6	10.1	50-85	1.19	.54	73.9	<.001	.52
	Somatic Complaints	80	58.4	7.7	50-83	.73	.07			
	Anxious/Depressed	80	58.7	8.8	50-81	1.07	.02			
	Social Problems	80	62.1	11.5	50-92	.94	.02		*Without Social Competence*	
	Thought Problems	80	60.2	7.8	50-76	.12	-1.00	21.4	<.001	.21
	Attention Problems	80	68.9	9.6	50-92	.12	-.23			
	Delinquent Behavior	80	58.5	8.9	50-84	.80	-.29			
	Aggression	80	61.0	12.2	50-93	1.13	.23			
	Social Competence	*70*	*39.6*	*7.6*	*24-55*	*-.17*	*-.73*			
Conners'	Social Index	28	60.9	13.5	45-90	.70	-.31	5.74	.03	.18
	Global Index	28	68.3	13.6	46-90	-.03	-1.05			
		ASD Group								
Measure	Scale	*n*	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	η^2
CBCL	Withdrawn	53	68.7	11.6	50-100	.41	-.48	64.7	<.001	.65
	Somatic Complaints	53	59.9	10.2	50-88	1.22	1.18			
	Anxious/Depressed	53	62.7	10.9	50-89	.77	-.11			
	Social Problems	53	69.7	10.4	53-91	.39	-.67		*Without Social Competence*	
	Thought Problems	53	68.2	11.0	50-109	.66	2.51	23.6	<.001	.33
	Attention Problems	50	72.8	10.6	54-97	.34	-.50			
	Delinquent Behavior	53	58.9	8.2	50-85	.89	.50			
	Aggression	50	61.3	9.3	50-88	.73	.13			
	Social Competence	*37*	*35.0*	*8.3*	*20-51*	*.07*	*-.81*			
Conners'	Social Index	20	76.4	13.6	45-90	-1.21	1.03	12.3	<.01	.39
	Global Index	20	65.5	12.0	40-90	-.11	.13			
		VCFS Group								
Measure	Scale	*n*	*M*	*SD*	Range	Skew	Kurtosis	*F*	*p*	η^2
CBCL	Withdrawn	17	59.5	10.7	50-82	1.00	-.13	16.9	<.001	.63
	Somatic Complaints	17	59.1	9.5	50-78	.67	-.78			
	Anxious/Depressed	17	57.2	6.7	50-74	1.02	.98			
	Social Problems	17	66.2	12.9	50-94	.57	-.23		*Without Social Competence*	
	Thought Problems	17	63.4	10.2	50-79	-.02	-1.48	7.61	<.001	.32
	Attention Problems	17	69.4	12.0	50-95	.34	-.06			
	Delinquent Behavior	17	55.3	7.1	50-70	1.25	.26			
	Aggression	17	60.0	12.5	50-93	1.38	1.61			
	Social Competence	*11*	*34.4*	*10.4*	*22-54*	*.44*	*-.53*			

Memory as predictor of adaptive functioning. Several multiple regression models were built and tested, with memory functioning (the four main domains of memory measured by TOMAL subtests, immediate and delayed recall) used as predictor variables of adaptive functioning. Given the exploratory nature of the investigation, predictors were entered using the stepwise backward method. This procedure was chosen because it handles suppressor effects better than the forward method as it is less likely to eliminate predictors that account for unique variance in the outcome measures, but only after the effect of other variables has been partialed out. Therefore, this method is more powerful than its alternatives (Field, 2005).

SSs from all four memory domains during either immediate or delayed recall were entered at once. Next, SSDs were used as a new set of predictors. The final model was made of retention rates (RRs) associated with each memory domain. RRs are ratios of delayed to immediate recall raw scores. Therefore, a value below one indicates decay, while a value above one indicates consolidation over time. A RR = 1.00 means that that the individual remembered the same amount of information during both immediate and delayed recall. Significant between-group differences in RRs were found on story [$F(3, 146) = 5.9$, $p < .001$, partial $\eta^2 = .11$] and visual memory [$F(3, 140) = 4.2$, $p < .01$, partial $\eta^2 = .08$].

The same four regression models defined by these sets of predictors were tested on each of the outcome variables. As is apparent from the description above, the first two models use static measures (i.e. one-time sampling) of memory, while the latter two use a combination of immediate and delayed recall, thus capturing a more dynamic aspect of memory. These derived variables were created as a consolidation index that captures the shift in memory performance from Time 1 to Time2.

Regression diagnostics. To evaluate external validity, the assumptions underlying multiple regression were checked. This procedure is necessary to ensure that the findings are not

artifactual, but would generalize to other samples. First, the variables entered in the regression equation were checked to ensure that they are quantitative (interval scale) or dichotomous categorical, and the range is unrestricted. Second, the non-zero variance assumption in each predictor was tested. Third, predictors were evaluated for multicollinearity. Interpredictor correlations of .80 or above should be flagged for further analysis, and those .90 or above excluded (Field, 2005). As shown in Table 14, the highest predictor intercorrelation was .38, thus demonstrating a high degree of independence of each other.

Table 14

Multicollinearity Diagnostics: Intercorrelations between Predictor Variables

	TOMAL Scaled Scores - Immediate Recall				
	n	Story	Facial	Visual	Word
Story	163	1	.25**	.24**	.33**
Facial	146		1	.23**	.22**
Visual	148			1	.17*
Word	161				1
	TOMAL Scaled Scores - Delayed Recall				
	n	Story	Facial	Visual	Word
Story	161	1	.22**	.19**	.38**
Facial	144		1	.11	.23**
Visual	148			1	.23**
Word	159				1
	TOMAL Scaled Score Deviations				
	n	Story	Facial	Visual	Word
Story	161	1	.12	.04	.13
Facial	144		1	.08	.17*
Visual	148			1	.04
Word	159				1

* Significant at $\alpha = .05$
** Significant at $\alpha = .01$

Formal tests of multicollinearity were also performed using critical values by Myers (1990) and Menard (1995). None of the regression models included in the final analyses had excessive levels of collinearity among the predictor variables. Fourth, the assumption of

independent errors was evaluated using the Durbin-Watson test (Durbin & Watson, 1951) that is designed to detect the presence of autocorrelation. Given that cognitive data are virtually invariably positively correlated, the alternative hypothesis for these analyses was $\rho > 0$ using the decision rule outlined by Kutner, Nachysheim, Neter, and Li (2005). Based on these criteria and using the $\alpha = .05$ criterion for the autoregression statistic, all of the significant regression models discussed here met the assumption of uncorrelated errors. Fifth, the assumption of independence was ensured by assigning each participant to only one diagnostic group. Sixth, the assumption of linearity was evaluated by a visual examination of the scatter plots, and no apparent pattern of heteroscedasticity was detected. Finally, the data were screened for extreme data points using both the Mahalanobis distance and Cook's *D* as indicators of multivariate outliers. Critical values for the Mahalanobis distance were obtained from Barnett and Lewis (1978). Critical values for Cook's *D* were obtained from Cook and Weisberg (1982). Although a number of the regression models contained one or two data points with Mahalanobis distances exceeding the critical value, they were not excluded from the analysis, as they did not exert undue influence on the model as measured by Cook's *D*.

Predicting academic functioning. Both immediate and delayed recall SSs explained 22% of the variability in reading scores. The only significant predictors were story ($\beta = .40$ - $.43$) and visual span ($\beta = .18$ - $.21$). The hypothesis that measures of consolidation will be better predictors of performance was not supported: the regression model using SSDs was not significant, and the one based on RRs explained only 5% of the variance in the criterion variables, with story memory being the only significant predictor ($\beta = .25$).

Less variability (11-13%) in spelling scores was captured by SSs. The same two domains of memory (story [$\beta = .32$] and visual [$\beta = .20$]) were the significant predictors using immediate recall scores. In the model based on delayed recall scores, story memory ($\beta = .30$) was the only

significant predictor. As described above, measures of consolidation performed poorly at predicting outcome compared to static measures of memory: both SSDs and RRs failed to produce significant regression models.

All four models performed significantly better in the domain of mathematics. All four predictors were significant at immediate recall (story memory β = .37, facial memory β = .14, visual memory β = .21, word memory β = .14) together accounting for 37% of the variance in the criterion. At delayed recall, the model was still significant, although less predictive overall (adjusted R^2 = .26). This time, the only significant predictors were story memory (β = .38) and visual span (β = .20). The consolidation based models performed even more poorly. SSDs predicted only 11% of the variance in standard math scores using three significant predictors: facial (β = .15), visual (β = .21) and word memory (β = .19). The model using RRs was the lowest performing (adjusted R^2 = .06) with story memory RR as the only significant predictor (β = .18). It is somewhat surprising that a verbal memory domain (story) is the most significant predictor of math skills, about twice as influential as visual span.

Predicting social functioning. None of the four models reached significance predicting the Social Composite on the CBCL. These negative findings are reliable: based on the power analysis tables developed by Miles and Shevlin (2001), assuming a medium effect size, the models had a power of .85 – exceeding the benchmark .80 value proposed by Cohen (1992). Therefore, it is reasonable to assume that the predictive power of memory in social functioning is less than medium.

When Conners' Social Index was used as an outcome measure, the model based on SSs during immediate recall produced an adjusted R^2 of .18, with visual span as the only significant predictor (β = -.37). This finding is puzzling for a couple of reasons. First, the same model with more power was non-significant when the same construct was measured with CBCL, an

instrument that is thought to be an equivalent index of social functioning. Second, the model seems to be a valid one, as it meets all underlying assumptions of regression; therefore, the unusual finding cannot be discounted on grounds of model bias. The other three models failed to reach significance.

Table 15 provides a visual summary of the regression analyses discussed above. Two major conclusions are apparent from this overview. First, memory performance is clearly a better predictor of academic than social functioning. In fact, with the unusual exception described above, none of the models could account for a significant amount of variance in social skills. Second, dynamic measures of memory are consistently inferior to static measures. Therefore, the second major hypothesis in this study is not supported by the data. In fact, the opposite seems to be the case (Type III error): overall, consolidation has a poorer predictive power than performance measured either immediately or after a delay.

Table 15

Summary of Regression Models Predicting Adaptive Functioning using TOMAL Subtests

Models	Criterion Variables														
	Reading			Spelling			Math			CBCL Social			Conners' Social		
TOMAL Subtests	df_2	*Sig.*	*Adj.* R^2	df_2	*Sig.*	*Adj.* R^2	df_2	*Sig.*	*Adj.* R^2	df_2	*Sig.*	*Adj.* R^2	df_2	*Sig.*	*Adj.* R^2
SS Immediate	176	<.01	.22	175	<.01	.13	182	<.01	.37	79	.11	.04	31	.04	.18
Significant predictors	Story, Visual			Story, Visual			Story, Facial, Visual, Word			None			Visual		
SS Delayed	176	<.01	.22	175	<.01	.11	182	<.01	.26	79	.17	.03	31	.13	.10
Significant predictors	Story, Visual			Story			Story, Visual			None			None		
SS Difference	176	.40	.00	175	.25	.01	182	<.01	.11	79	.66	-.02	31	.29	.03
Significant predictors	None			None			Facial, Visual, Word			None			None		
Retention Rate	123	.03	.05	122	.33	.01	127	.02	.06	67	.84	-.04	30	.73	-.06
Significant predictors	Story			None			Story			None			None		

In a further attempt to explore the covariation between the targeted criterion variables and memory functioning, new hierarchical regression models were developed that entered the four memory domains first, followed by gender, age, and FSIQ – each in a separate block. Table 16 displays the results of the analyses. Not surprisingly, IQ was a significant predictor of academic achievement in every model even though it was entered last, causing the adjusted R^2 to increase substantially and changing the significance and β-values of other predictors. Age and gender were unrelated to outcome. The mirror image of these findings describes the models predicting social functioning: age was a significant predictor in all cases (although had a much stronger influence on Conners' Social Index than on CBCL's Social Composite), while IQ never reached significance.

Table 16

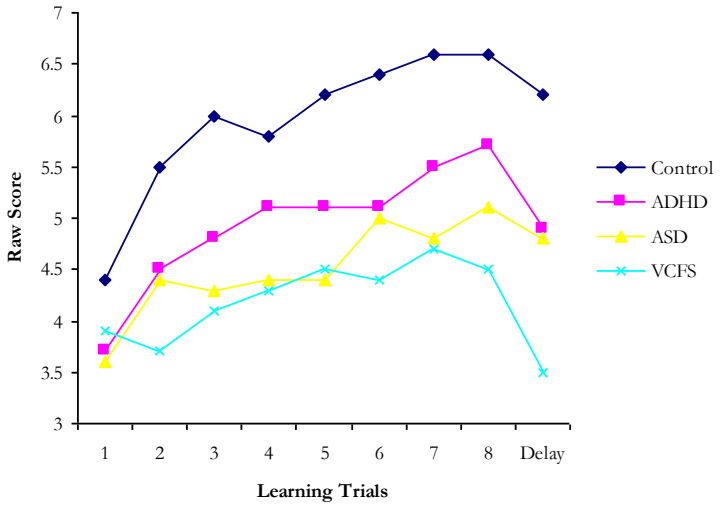
Summary of Regression Models Predicting Adaptive Functioning using TOMAL Subtests, Gender, Age and FSIQ

Models	Criterion Variables														
	Reading			Spelling			Math			CBCL Social			Conners' Social		
TOMAL Subtests	*df*$_2$	*Sig.*	*Adj.* R^2	*df*$_2$	*Sig.*	*Adj.* R^2	*df*$_2$	*Sig.*	*Adj.* R^2	*df*$_2$	*Sig.*	*Adj.* R^2	*df*$_2$	*Sig.*	*Adj.* R^2
SS Immediate + gender, age & FSIQ	158	<.01	.39	159	<.01	.28	164	<.01	.55	72	.05	.08	23	.02	.39
Significant predictors	Story, Facial, IQ			IQ			Word, IQ			Age			Visual, Age		
SS Delayed + gender, age & FSIQ	158	<.01	.38	159	<.01	.27	164	<.01	.54	72	<.01	.11	23	<.01	.25
Significant predictors	IQ			IQ			IQ			Age			Age		

Learning Curve Analyses

A special interest of this study was examining the rate at which individuals belonging to the selected diagnostic groups learn the presented material through several trials. Two of the TOMAL subtests studied here contain repeated trials: the Word and Visual Selective Reminding. The learning curve analysis was performed using repeated-measures ANOVAs.

Visual selective reminding. In this visual span test, as expected, controls remembered an increasing amount of the stimuli presented over the eight trials, with very large within-group effect (adjusted η^2 = .37). The children with ADHD (within-group adjusted η^2 = .27) and ASD (within-group adjusted η^2 = .14) also showed significant improvement over repeated exposure to the stimuli, but stayed consistently below controls, showed more variability, and plateaued earlier. The VCFS group, however, showed no significant increase in material remembered from the first to the last learning trial in visual memory. This is partly due to the smaller sample size, but at the same time it should be noted that the repeated measures ANOVA is a powerful design and is likely to detect sizeable effects even in small samples. During delayed recall all groups deteriorated, but controls and ASD lost the least amount of information. The visual learning curves are summarized in Figure 10, which displays group RS means across trials.



The saw-tooth pattern in all of the learning curves suggests that the underlying construct (visual span) is volatile, and paradoxically, subsequent exposures to the same stimuli may actually interfere with the consolidation of the memory trace. There are several instances of "slipping," when a trial produces a mean that's lower than the mean of the trial preceding it. Slipping was observed in all groups but ADHD.

Interestingly, there were no significant between-group differences during the first trial, as shown in Table 17. All groups performed similarly after the first exposure to the target stimuli but differently at each trial thereafter. In other words, there is a divergence in between-group performance over time: the rate at which they encode and are able to retrieve visual information differs, even though initially they retain the same amount of information. The underlying learning deficit seems to reveal itself gradually, becoming even more pronounced during delayed recall.

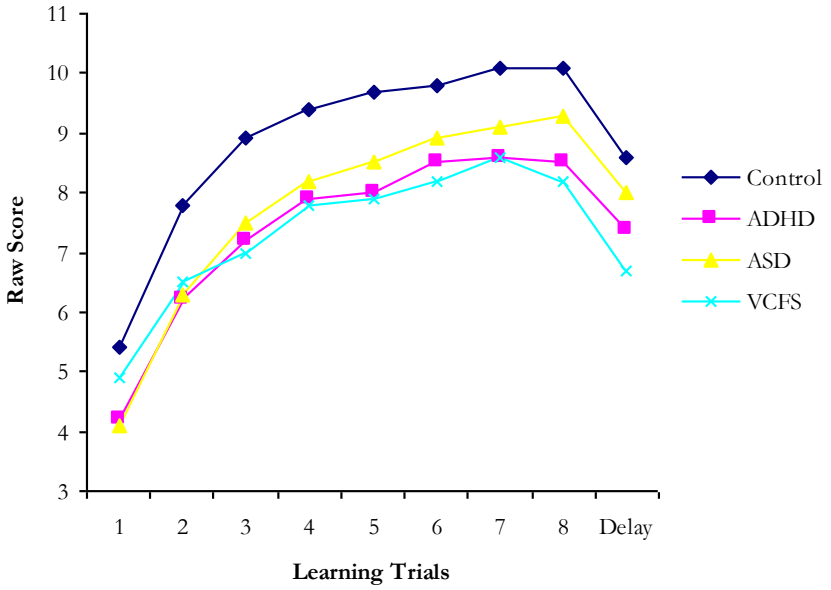
Table 17

Summary of Within- and Between-Group Contrasts during Visual Selective Reminding Trials

		Visual Selective Reminding Trials									Within-Group contrasts			
		1	2	3	4	5	6	7	8	*Delay*	*F*	*ε**	*p*	η^2
Control	*M*	4.4	5.5	6.0	5.8	6.2	6.4	6.6	6.6	6.2	18.9	.68	<.001	.34
n = 37	*SD*	1.5	1.7	1.8	1.9	1.8	1.6	1.4	1.5	1.5				
ADHD	*M*	3.7	4.5	4.8	5.1	5.1	5.1	5.5	5.7	4.9	18.2	.64	<.001	.23
n = 61	*SD*	1.7	1.7	1.8	1.9	2.0	2.0	2.0	1.9	2.3				
ASD	*M*	3.6	5.4	4.3	4.4	4.4	5.0	4.8	5.1	4.8	4.23	.75	<.001	.13
n = 30	*SD*	1.6	5.5	2.0	2.1	2.0	2.0	2.2	2.3	2.0				
VCFS	*M*	3.9	3.7	4.1	4.3	4.5	4.4	4.7	4.5	3.5	1.84	.68	.08	.12
n = 16	*SD*	1.6	1.8	1.8	2.2	2.4	2.7	2.2	2.1	2.1				
Between-Group Contrasts	*F*	1.91	4.53	6.38	3.62	5.87	5.41	5.73	6.03	7.20				
	p	.13	.01	.00	.02	.00	.00	.00	.00	.00				
	η^2	.04	.09	.12	.08	.11	.10	.11	.11	.13				

* The arithmetic average of the Greenhouse-Geisser and the Huynh-Feldt sphericity estimate

Word selective reminding. During this auditory verbal learning task, all groups made significant gains over repeated trials. Within-group adjusted η^2 for trials one through eight ranged between .53 (VCFS) and .73 (controls). In other words, all learning curves were steep and closely followed each other, as shown in Figure 11, which displays group RS means across trials.



During delayed recall all groups deteriorated and lost information at approximately the same rate. The mirror image of the trend in the visual learning trials was observed here: the groups were significantly different throughout the first eight learning trials but not during delayed recall. The differences in recall were the most pronounced after the first trial (adjusted η^2 = .09, medium effect), partly driven by the small within-group variability, fluctuated throughout the remaining trials (adjusted η^2 ranged between .05 [small effect] and .07, [medium effect]), and disappeared during delayed recall (p value = .15, estimated adjusted η^2 = .03 [very small]). It must be noted that this convergence at delayed recall is partially driven by the substantially and uniformly larger within-group variability compared to the first trial.

Consolidation seems to affect individuals within the same group differently, making it more difficult to draw general conclusions. Moreover, the learning curves show a steady, almost uniform increase without the saw-tooth patterns seen in visual memory. This suggests that the underlying construct in auditory verbal learning trials is more stable, and its acquisition follows a predictable pattern in both neurotypicals and children with developmental disorders. The details of the analyses are shown in Table 18.

Table 18

Summary of Within- and Between-Group Contrasts during Word Selective Reminding Trials

		Word Selective Reminding Trials									Within-Group contrasts			
		1	2	3	4	5	6	7	8	*Delay*	*F*	*ε**	*p*	η^2
Control	*M*	5.3	7.7	8.8	9.3	9.6	9.7	10.0	10.0	8.5	66.1	.57	<.01	.65
n = 38	*SD*	1.5	2.0	2.4	2.1	2.3	2.1	2.0	2.0	2.7				
ADH	*M*	4.2	6.5	7.2	7.9	8.0	8.5	8.6	8.5	7.4	87.2	.66	<.001	.52
D	*SD*	1.6	2.3	2.6	2.4	2.7	2.4	2.7	2.7	3.5				
n = 82														
ASD	*M*	4.1	6.3	7.5	8.2	8.5	8.9	9.1	9.3	8.0	55.1	.69	<.001	.58
n = 41	*SD*	1.7	2.2	2.4	2.6	1.9	2.0	2.0	1.9	2.6				
VCFS	*M*	4.9	6.5	7.0	7.8	7.9	8.2	8.6	8.2	6.7	10.7	.38	<.001	.40
n = 17	*SD*	1.4	1.9	2.0	2.8	2.6	2.7	2.4	2.7	3.9				
Between	*F*	5.36	3.56	4.16	3.13	4.16	3.01	3.16	4.38	1.82				
-Group	*p*	.00	.02	.01	.03	.01	.03	.03	.01	.15				
Contrasts	η^2	.09	.06	.07	.05	.07	.05	.05	.07	.03				

* The arithmetic average of the Greenhouse-Geisser and the Huynh-Feldt sphericity estimate

Discussion

The results presented above converge into one general observation: different developmental disorders, memory domains, times at which learning is sampled and measures of adaptive functioning have a complex relationship. A few interesting patterns with clinical relevance have emerged. However, in many areas error variance due to the inherent variability in neurodevelopmental disorders is too large for a clear pattern to be detected.

The first hypothesis, that measures of memory consolidation would contribute additional information to the neurocognitive profiles of the studied diagnostic groups beyond a single-time sampling of a given memory domain, was supported. Also, the study concurred with several earlier reports, thus contributing to the existing knowledge base on cognitive profiling by providing independent confirmation of old result using different samples, instruments, and data analyses. There were also a few puzzling disagreements with the existing literature, suggesting a need for further studies. The simultaneous comparison of multiple clinical samples, memory domains, and learning trials allowed for unique opportunities to evaluate the differential diagnostic efficiency of the TOMAL and CBCL as well as other conceptually similar instruments.

Several noteworthy findings emerged. First, it appears that facial memory and visual span are general areas of weakness in developmental disorders, although this is much more evident during immediate recall. This finding is consistent with meta-analytical reviews that reported a specific weakness in the visual-spatial domain in children with ADHD (Martinussen et al., 2005). However, other studies found no impairment in children and adults with ADHD (Crawford et al., 2007; Kaplan et al., 1998; Mealer et al., 1996). In ASD, this finding contradicts previous reports of normative performance in the visual-spatial domain (Ambery et al., 2006; Renner, Klinger, & Klinger, 2000; Williams et al., 2006), but agrees with others (Minshew & Goldstein,

2001; Williams et al., 2005). In VCFS, this finding is consistent with previous reports (Lajiness-O'Neill et al., 2005).

After a delay, the performance gap narrowed considerably. In fact, pronounced improvement in these domains from immediate to delayed recall appears to be a potential diagnostic marker of ASD and VCFS based on the present data. Although this finding was replicated using alternative data analytical techniques, it contradicts previous reports by Burden and Mitchell (2005) and Johnson et al. (2001) who showed that initially, the ADHD group performed similarly to controls, but after a delay their performance deteriorated and had a steeper forgetting curve than controls. Similarly, the existing ASD literature on temporal processing of visual information suggests that this type of stimulus decays over time – the opposite of what was observed in the present study.

Second, auditory verbal learning cannot distinguish the studied diagnostic groups very well. Moreover, after a delay, even neurotypicals and all the clinical groups perform similarly on word memory, and all perform at the same level as the normative sample. Therefore, list learning tasks have little differential diagnostic potential in these populations, especially during delayed recall trials. One exception is the ASD group, which reliably improved its performance from immediate to delayed recall. All of these findings are consistent with previous reports (Ambery et al., 2006; Burden & Mitchell, 2005; Cutting, Koth, Mahone, & Denckla, 2003; Leitner et al., 2007; Minshew & Goldstein, 1993; 2001; Øie et al., 1999; Seidman et al., 1998; Toichi & Kamio, 2002; Williams et al., 2005; 2006).

Third, learning curve analyses revealed a couple of interesting trends. On visual trials, initially all groups performed at the same level but diverged over subsequent trials. After a delay, their performance was once again indistinguishable from one another. This observation argues for the spacing out of the visual material to optimize encoding – not just as a remedial

technique, but a mainstream didactic strategy, as neurotypicals are not immune to the disruption caused by rapid repeated exposure to the same visual stimuli.

This is a somewhat surprising result and may have important implications to diagnosing and managing these disorders. Diagnostically, visual memory performance sampled later (either during an n^{th} trial of identical exposures to the same stimuli or a delayed recall of a single learning trial presented earlier) has a much better discriminative power. Conversely, if a visual learning task has only one trial, it may mask significant between-group differences – or bias probability estimates of an individual belonging to one clinical group vs. another. From a treatment perspective, cognitive profiles provide important insight about what kind of treatment modality would be best suited for a given clinical population. Specifically, visual aids and prompting is frequently used in managing these disorders. However, these results suggest that contrary to the dominant views, children with autism or VCFS do not learn well visually – at least they do not encode nearly as well as during rote verbal learning.

On word trials, the opposite trend was observed: the group performance was different after the first trial but converged after a delay. In other words, there is a convergence in between-group performance over time: their rate of acquisition differs during the immediate recall trials, but the performance gap closes after a delay. The underlying learning deficit is apparent initially, but it is no longer detectable during delayed recall.

Again, this is a somewhat surprising result with potential implications to diagnosing and managing these disorders. Diagnostically, auditory verbal learning has the most clinical utility during the first trial. Beyond that, its discriminant power decays rapidly and disappears during delayed recall, when between-group differences become undistinguishable. Put differently, this finding suggests that time alone is a performance enhancer in developmental disorders. In terms of intervention, this offers a simple strategy to give children with these diagnoses extra time with

learning tasks, essentially allowing them to attain and/or demonstrate their full learning potential.

One important confound in fully assessing the parallel processes in auditory (i.e. verbal) and visual (i.e. non-verbal) memory is the lack of a visual analogue for story memory among the four core TOMAL subtests. Language (i.e. verbal mediation in memory performance) is difficult to control for, as eliminating a semantic context shifts the cognitive paradigm into a qualitatively different domain. Therefore, it is nearly impossible to establish that a certain visual memory task is the visual equivalent to a contextual verbal memory task.

Unrelated to memory, academic achievement scores also have differential diagnostic potential, especially math scores, which seem to be especially sensitive markers of the underlying deficits in all developmental disorders. The most noticeable decline was observed in VCFS. However, even the ASD group shows a relative weakness in math. Therefore, math-based tests seem to have the best differential diagnostic potential within the achievement domains.

In terms of adaptive functioning, no phenotypic marker was found with the measures used. All children with developmental disorders had significant elevations on all clinical scales. Similarly, significant social skill deficits were reported by all parents. Somewhat surprisingly, attention deficit was the most endorsed symptom across all diagnostic categories and did not differentiate the clinical groups. Given the publicity of ADHD as a childhood disorder and the salience of attention problems vs. the complexity and subtlety of ASD and VCFS symptoms, coupled with the higher stigma associated with ASD, it is understandable why parent reports on child symptomatology is skewed towards ADHD traits. However, if attention deficit is invariably the most significant deficit reported by the parent regardless of the diagnosis (as suggested by the data presented here), clinicians should interpret elevations on this scale carefully and not

assume that it is the defining area of weakness in the child's functioning, but rather a ubiquitous tendency in parent rating of all psychopathology - acquired or neurodevelopmental.

The second hypothesis was that consolidation indices will perform better at predicting functional outcome than static measures of memory. This hypothesis was refuted by the data. In fact, the opposite seems to be true: static measures are consistently better predictors of adaptive functioning across various domains and instruments. Specifically, immediate recall scores are the most effective predictors. Measures of consolidation do not predict any aspect of adaptive functioning very well. This negative finding may not necessarily mean that measures of consolidation are poorer predictors than static ones. Given the reliable and sometimes robust between-group differences on SSDs and RRs, it is apparent that different clinical groups have different (in both direction and magnitude) consolidation patterns. This between-group heterogeneity cancels out within-group patterns when all data points are collapsed in a single analysis, as was done with the regression models. In other words, as each diagnostic group has a different consolidation pattern, when combined and averaged, all patterns disappear. Therefore, the only conclusion that can be ascertained with confidence is that no universal consolidation pattern exists that would predict functional outcome across the studied diagnostic categories better than static measures of memory. It is quite plausible, however, than within each group consolidation does in fact predict functional outcome. Given the complexity of models used and the limited within-group sample sizes, there was not enough power to perform the regression analyses within each group separately.

Memory-based regression models performed the best when predicting academic achievement. Math skills shared the most variance (26-37%) with memory functioning. Interestingly, story memory was the most influential predictor of the four memory domains. However, mathematical reasoning is analytical, integrative, and sequential in nature, thus similar

to the cognitive demands involved in remembering a narrative. In that sense, they may be recruiting similar underlying neurocognitive resources. Single word reading shared 22% of the variance with memory functioning, story and visual memory being the two significant predictors. This finding is consistent with the expectations: reading is a complex skill that requires both semantic organization (comprehension) and efficient scanning (visual span). Spelling shared the least amount of variance with memory functioning (11-13%) with the same pair of significant predictors as reading. With spelling, they are likely tapping into the larger construct of language processing and graphomotor skills in particular. Another reason for the shrinkage in predictive power may be the unique influence of the motor component, which is not assessed by the four core domains of the TOMAL.

Social skills as measured by the CBCL could not be predicted with any of the models used for the present analyses. Visual memory during immediate recall shared 18% of variance with Conners' Social Index, suggesting that the predictive power of memory on social functioning partly depends on the instrument used to measure the criterion variable. One implication of this finding is that there is a significant amount of chance variability (instrumentation artifacts) involved in regression models used to describe social functioning. This observation calls for caution while interpreting the results: given the imperfect measurements available, statistical models remain prone to random perturbations in the data, thus limiting our ability to draw definite conclusions. The failure of traditional measures of memory to predict social functioning may be due to the fact that social learning is a cognitive domain that is not assessed by most standardized tests that focus on declarative memory and abstract verbal or visual learning.

To further explore the negative findings with social functioning, age, gender, and FSIQ were added as individual predictors to TOMAL scores in a hierarchical regression model. In all

of the models predicting academic achievement, of the newly included predictors, IQ was the only significant one even though it was entered last. Its inclusion nearly doubled the amount of variance the models were able to explain in the criterion variable. A different trend was observed in the models predicting social functioning: the only significant predictor was age, also roughly doubling the amount of variance explained in prior models.

It appears that there is a clear divide between the academic and social skills: the former is driven by cognitive variables, while the latter is not. This finding is remarkable, as IQ tends to be a ubiquitous and influential covariate of virtually all psychological variables. The present results, however, suggest that social functioning is independent of cognitive abilities and follows a developmental trend: as the child gets older, he or she will encounter more social problems even though the exact mechanism cannot be ascertained at this time. Alternatively, this could mean that the same level of impairment is perceived as increasingly severe as expectations rise with each developmental milestone. At this time, the above statement must be restricted to parental reports within clinical populations. No data are available on collateral observers (other parents, teachers, peers, self) or parent reports on normally developing children to assess whether this is a universal trend or driven by underlying neurological deficits, thus unique to childhood disorders.

Also, the present analysis cannot elucidate the exact mechanism behind age as a predictor of social functioning, but some tentative explanations can be generated. First, the results could be artifactual: achievement and IQ scores are much more rigorously age-corrected (norming is done in as narrow as three-month intervals) than scores on parent rating scales (norming is done in 3- or 4-year intervals). Hence, the effect of age is much more rigorously controlled for in the former measures, allowing age to exert a different influence on the regression models. Second, the results may reflect a real developmental trend: as children age,

social norms regulating social interactions are becoming increasingly stringent, thus creating larger discrepancies between parental expectations and the child's actual ability to be effective in social situations that are turning more complex. Such complex relationships between cognitive and adaptive functioning variables have been reported in the literature. There appears to be an intricate web of interactions among age, IQ, ASD severity, and type of adaptive functioning. The picture is further complicated by instrumentation issues: outcome often varies with the test used to measure it (Klin et al., 2007; Perry et al., 2009; Nordin & Gillberg, 1998; Schatz & Hamdan-Allen, 1995).

The strengths of the results described here include a large clinical sample, comprehensive and valid statistical analyses, and the use of well-established, standardized measurement instruments. The simultaneous comparison of several diagnostic groups using the same measures allows for a unique opportunity to detect patterns in cognitive functioning that can enhance both the differential diagnosis and the treatment planning of children with developmental disorder.

The present study also has several weaknesses. The groups had unequal sample sizes, which on occasion biased the results of between-group contrasts. The samples were quite heterogeneous, which improves the generalizability of the findings but works against internal validity. The most significant limitations, however, stem from the sources of data used. First, there were no data on the social functioning of neurotypicals, which might have altered the overall trends. Second, there were no collateral observers besides parents for the clinical groups, which could have provided an independent source of data to cross-validate the models. Third, there were no direct observation data by experts, which could have been an invaluable alternative to objectively verify how the child actually functions in the social environment, independent of parent perception, which might be biased.

The present study produced several results with potential research and clinical interest. 1) Non-verbal memory (facial recognition and visual span) seems to be a relative weakness in ADHD, ASD and VCFS. 2) Differential consolidation patterns operate across diagnostic categories and memory domains that could enhance the existing neurocognitive profiles associated with the studied disorders, and implicitly the diagnostic accuracy. 3) Learning curve analyses revealed that, contrary to dominant views, children with ASD do not learn well visually, but have a relatively well-preserved auditory verbal memory. 4) Among the commonly assessed domains of academic achievement, math scores are the most vulnerable to the cognitive impairments associated with neurodevelopmental disorders. 5) On structured rating scales, parents tend to report attention deficit as the dominant symptom regardless of the actual diagnosis of the child. 6) Current ways of measuring memory functioning predicts academic achievement the best (especially math), but performs poorly at predicting social outcome. 7) Of the measured variables, age is the best predictor of social impairment: the older the child, the higher the parent-reported level of functional deficit.

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Appendix
IRB Approval Letter

EASTERN MICHIGAN UNIVERSITY

Education First

March 26, 2009

Laszlo Erdodi
Department of Psychology

Dear Laszlo Erdodi:

The Human Subjects Institutional Review Board (IRB) of Eastern Michigan University has granted approval to your proposal, "Memory Consolidation In Development Disorders."

After careful review of your completion application, the IRB determined that the rights and welfare of the individual subjects involved in this research are carefully guarded. Additionally, the methods used to obtain informed consent are appropriate, and the individuals participating in your study are not at risk.

You are reminded of your obligation to advise the IRB of any change in the protocol that might alter your research in any manner that differs from that upon which this approval is based. Approval of this project applies for one year from the date of this letter. If your data collection continues beyond the one-year period, you must apply for a renewal.

On behalf of the Human Subjects Committee, I wish you success in conducting your research.

Sincerely,

Deb de Laski-Smith

Deb de Laski-Smith, Ph.D.
Interim Dean
Graduate School
Administrative Co-Chair
University Human Subjects Review Committee

Note: If project continues beyond the length of **one** year, please submit a continuation request form by **3/26/09**.

Reference # 0902016

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