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The Longitudinal Stability of Memory in Males with Autism Spectrum Disorder

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ABSTRACT

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Previous research has demonstrated mixed evidence on impaired memory functioning in autism spectrum disorder (ASD), with the only consensus that there appears to be much heterogeneity. In addition, no research to date has examined the stability of memory in ASD. This study examined the stability of memory function in ASD compared to typically developing age-matched controls. Participants were administered the Test of Memory and Learning (TOMAL) twice, three years apart, in an established longitudinal NIH-supported investigation of ASD. Based on available research contrasting memory development in healthy individuals versus those with ASD, it was hypothesized that memory performance in the control group would be stable across time and that, compared to the control group, the autism group would demonstrate less stable memory function as measured by the TOMAL. Repeated Measures ANOVA and Reliable Change Index calculations of TOMAL Index and Subtest scores largely supported these hypotheses. The control group remained stable across time on all TOMAL indices and the ASD group showed improvement on the Composite Memory Index, Verbal Memory Index, and Delayed Memory Index but not the Non Verbal Memory Index. Clinical and research implications are discussed.

Keywords: memory, autism spectrum disorders, Test of Memory and Learning

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The Longitudinal Stability of Memory in Males with Autism Spectrum Disorder

Autism, a Greek term that means "living in self" was coined by a Swiss psychiatrist, Eugen Bleuler in 1911 to describe self-absorption and reduced social relatedness. Leo Kanner (1943), in his seminal piece, "Autistic Disturbances of Affective Contact," used this term to describe the behavior of 11 children with an "inability to relate to themselves in the ordinary way to people and situations from the beginning of life [and] acting if almost as if hypnotized" (p. 242). Kanner's "infantile autism" was similar to schizophrenia in regards to obsessiveness, echolalia and stereotyped behavior; however, autistic symptoms were noted in infancy, thereby qualifying it as its own syndrome since it was not a period of normal function and then regression as can be observed in schizophrenia. During this same era of mid-twentieth century, Hans Asperger independently described a similar condition which he labeled "autistic psychopathy" in which individuals had severe deficits in social integration and odd eye gaze but intact speech (Asperger, 1944).

Despite the number of studies that followed Kanner and Asperger's influential works, autism was not included in the first edition publication of the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 1952). Instead, it debuted in DSM-II as schizophrenia, childhood type and was described as the following:

> "This category is for cases in which schizophrenic symptoms appear before puberty. The condition may be manifested by autistic, atypical and withdrawn behavior; failure to develop identity separate from the mother's; and general unevenness, gross immaturity and inadequacy of development. These developmental defects may result in mental

> retardation, which should also be diagnosed." (American Psychiatric Association, 1968, p. 35)

It was later relabeled as "infantile autism" in the DSM-III (American Psychiatric Association, 1980) and included the following criteria:

> "(1) social delay or deviance that was not just a function of mental retardation, (2) communication problems that were also not a function of mental retardation, (3) unusual behaviors like stereotyped movements/mannerisms and (4) onset prior to 30 months of age." (p. 132)

The label changed to "autistic disorder" in the DSM-III text revision (American Psychiatric Association, 1987) due to controversy surrounding the use of the infantile descriptor. "Childhood autism" was used in the tenth release of the International Classification of Diseases (ICD-10) (World Health Organization, 1993). A year later, the DSM-IV (American Psychiatric Association, 1994) changed it to "autistic disorder" and classified it as a pervasive developmental disorder with associated subtypes. These subtypes included: Autism Disorder, Asperger's Disorder, Rett's Disorder, Childhood Disintegrative Disorder, and Pervasive Developmental Disorder Not Otherwise Specified (PDD-NOS). Yet another major diagnostic shift regarding pervasive developmental disorders is expected to occur in the next edition, DSM-V. The term "Autism spectrum disorders" (ASD) will be used to identify those previously diagnosed with autistic disorder (autism), Asperger's disorder, childhood disintegrative disorder, and pervasive developmental disorder not otherwise specified; these disorders will be subsumed under the ASD label. With regards to the DSM-V, an individual will have to meet all three criteria listed below to qualify for diagnosis of autism spectrum disorder (American Psychiatric Association, 2012)—see website:

1. Clinically significant, persistent deficits in social communication and interactions, as manifest by all of the following:

    a. Marked deficits in nonverbal and verbal communication used for social interaction

    b. Lack of social reciprocity

    c. Failure to develop and maintain peer relationships appropriate to developmental level

2. Restricted, repetitive patterns of behavior, interests, and activities, as manifested by at least TWO of the following:

    a. Stereotyped motor or verbal behaviors, or unusual sensory behaviors

    b. Excessive adherence to routines and ritualized patterns of behavior

    c. Restricted, fixated interests

    d. Hyper- or hypo-reactivity to sensory input

3. Symptoms must be present in early childhood (but may not become fully manifest until social demands exceed limited capacities)

Although these criteria are focused on deficits in communication and behavior, numerous cognitive impairments are associated with autism (Geschwind, 2009; Giedd & Rapoport, 2010). Intellectual impairments are the most commonly reported (Spencer et al., 2006), but some form of memory impairment also seems common to the disorder (Boucher, Mayes, & Bigham, 2012). For example, early research demonstrated that children diagnosed with autism perform at lower levels than age-matched controls on free recall of information (Boucher & Warrington, 1976)

including immediate recall of word lists (Boucher, 1981) and recognition memory (Boucher & Warrington, 1976). More recent studies indicate deficits in episodic memory in autism (Southwick et al., 2012) and difficulty remembering when words have been switched in a sequence (Poirier, Martin, Gaigg, & Bowler, 2011).

Research examining memory for emotionally salient material has been mixed, suggesting that if emotion is experimentally modulated to increase arousal, which may positively influence attention, subjects with autism perform more similarly to individuals with typical development (Maras, Gaigg, & Bowler, 2012). Maras and colleagues conducted two experiments to assess memory for emotionally salient material. The first examined 19 individuals (males and females) diagnosed with ASD (mean age 35.2) and 19 typically developing individuals (males and females; mean age 37.1) using two versions ("neutral" and "emotional") of a 12-image, narrated slide show. The second examined 24 individuals (males and females) diagnosed with ASD (mean age 40) and 24 typically developing individuals (males and females; mean age 43.3) using two versions ("neutral" and "emotional") of a short scene from a film. They found that "observations from two experiments showed that both individuals with and without a diagnosis of ASD demonstrate enhanced memory for, and diminished forgetting rates of, emotionally salient compared with neutral events" (p. 7). Nonetheless, Maras and Bowler (2012) report overall reduced memory performance in autism, especially remembering naturalistic events in the absence of cues. Likewise, even after matching on IQ, Southwick et al. demonstrated a generalized reduction of memory ability in autism, based on performance on the Test of Memory and Learning (Reynolds & Bigler, 1994).

Stability of Memory Impairment in ASD

Although Kanner (1943) did not specifically assess memory function, he noted the variability of cognitive performance in his sample. Indeed, cognitive heterogeneity is a characteristic of ASD, with some studies indicating that upwards of 70% of a general population sample of ASD subjects will have some level of intellectual impairment that generally remains stable over time (Mandelbaum et al., 2006; Silver & Rapin, 2012). Yang et al. (2011; 2010) have demonstrated considerable variability in intellectual performance over time in children with developmental disabilities, including ASD. Based on a review of 23 studies, Begovac et al. (2009) found that in general intellectual ability in ASD was most often reported to be in the borderline to mild range of intellectual impairment. In regards to stability, the majority of studies have found no longitudinal changes in IQ metrics. Although these studies suggest that cognitive ability remains relatively stable in ASD, there are no studies that have specifically examined the stability of global memory functioning in ASD.

Importance of Memory in Understanding the Cognitive Deficit in Autism

Some form of memory impairment seems common to autism (Boucher et al., 2012). Williams, Goldstein, and Minshew (2006b) stated that "memory has been characterized as both the cardinal cognitive domain largely responsible for the clinical manifestations of the disorder or as secondary to a more generalized cognitive deficit that transcends memory, such as executive dysfunction" (p. 21). Gaigg, Gardiner, and Bowler (2008) reported that "autism spectrum disorders (ASDs) are characterized by a relatively specific pattern of typical and atypical memory functioning" (p. 983). "Typical" memory functioning was defined as that which is similar to typically developing controls and "atypical" memory functioning as that which demonstrated diminished performance compared to healthy peers. They stated that

individuals with ASD tend to show "typical" performance on memory tasks involving recognition, priming, and cued recall whereas free recall tasks tend to yield "atypical" performance, although there is substantial contrary evidence. To assess memory functioning, Gaigg and colleagues tested 20 ASD individuals and 20 typically developing controls (males and females in each group; age range unreported) using lists of 16 words from 5 categories presented on flashcards. They found that ASD individuals demonstrated reduced performance on recall tasks compared to controls when categorical information was available to help them with recall, indicating that they rely on relational memory processes to a lesser degree than typically developing individuals.

Williams et al. (2006b) noted that although memory research in autism has been very inconsistent, one of the most common characteristics of memory in this population involves poor organizational strategies or context to support memory, more specifically difficulty with complex information processing abilities. They administered a memory battery normed for children to a group of high-functioning children with autism and compared their scores against matched controls and found that children in the autism group performed poorer on complex visual and verbal information as well as spatial working memory. The researchers went a step further and analyzed the performance of each group using principal components analysis, which indicated that memory abilities are organized differently for children with autism as compared to healthy children.

With regard to specific memory processes, one study focused on self-referenced memory in children diagnosed with autism and concluded that they did not show the standard self-referencing memory effect of enhanced processing of self-relevant information (Henderson et al., 2009). Another study examined metacognition (cognitive evaluation of one's own mental

processes) in autism (Wilkinson, Best, Minshew, & Strauss, 2010). By comparing high-functioning children and adults diagnosed with autism to typically developing children, they showed that those in the autism group (children specifically) were less accurate with memory awareness and were less reliable about differentiating between their confidence ratings.

A number of theories have been presented to account for these abnormalities in memory functioning and highlight other aspects of cognition that can lead to memory impairments including amnesia theory and executive function deficits. Amnesia theory was one of the first neurobehavioral models of autism that suggested memory was the underlying basis for the social, behavioral and language anomalies seen in this population. Boucher and Warrington (1976) compared children diagnosed with autism with age-matched normal children and an independent group of children matched on language and nonverbal reasoning ability on tasks. Each group was administered tasks that examined memory for written words, spoken words, and pictures. The autism group demonstrated impairment with free recall, while recognition and cued recall ability was spared. The authors suggested that the pattern of memory performance of the sample of children used in this study resembled memory performance patterns commonly found in amnestic adults.

The presence of an amnestic process was only partially supported by subsequent investigations. Impairments with immediate recall of word lists (Boucher, 1981), free recall, and recognition (Boucher & Warrington, 1976) were limited to low functioning individuals diagnosed with autism. Later studies failed to find this effect in samples comprised of autistic individuals of more average intelligence (Bennetto, Pennington, & Rogers, 1996; Minshew & Goldstein, 1993; Minshew & Goldstein, 2001; Minshew, Goldstein, Muenz, & Payton, 1992;). High functioning individuals with autism have been found to be proficient in the following areas:

immediate/delayed recognition of visual information (Ameli, Courchesne, Lincoln, Kaufman, & Grillon, 1988), long-term recognition and cued recall (Bennetto et al., 1996), and delayed match to sample tasks (Barth, Fein, & Waterhouse, 1995). Some individuals with autism have also been found to have superior list memorization ability, a task most typically impaired by amnesia (Mottron, Belleville, Stip, & Morasse, 1998; Pring & Hermelin, 2002; Thioux, Stark, Klaiman, & Schultz, 2006).

Executive function (EF) is an umbrella term for a set of behaviors that include inhibition, working memory, cognitive flexibility, set-shifting, initiation, generativity and self-monitoring (Jurado & Rosselli, 2007). EF contributes to memory task performance via selection of a recall strategy. For example, on delayed free recall of a list learning task, a typically developing individual with no history of traumatic brain injury may recite previously learned items by regrouping them into semantic categories. However, patients with prefrontal lesions commonly fail to incorporate such a retrieval strategy and have difficulty in retrieving memorized information without an external aid.

Research regarding the ability of ASD individuals to spontaneously generate novel behaviors is also mixed. Some have reported impairments in this area (Minshew et al., 1992) whereas others have not (Scott & Baron-Cohen, 1996). The same goes for self-monitoring tasks, where ASD impairments have only been reported on a post hoc basis. Despite over sixty years of research, no agreement has been reached on the role of memory functioning in ASD because the examination of memory as an underlying cognitive deficit in autism has yielded mixed results (Williams, Goldstein & Minshew, 2006a). This phenomenon may be attributed to a number of factors: (1) a high degree of variability within the autism population, such as the

difference between high and low functioning individuals and (2) the use of different memory measures and varied format of tasks (Ozonoff & Strayer, 2001).

Longitudinal Memory in Healthy Children

The brains of children age 3 to 15 years undergo a period characterized by dynamic growth and tissue loss. Developmental differences are also known to exist in children and adolescents of varying age groups, for example accelerated growth of frontal networks has been documented for young children between 3 to 6 years of age, whereas substantial parietal changes have been observed in pre-adolescents and adolescents between 11 to 15 years of age (Thompson et al., 2000). Anatomical studies suggest that white matter increases linearly throughout childhood while cortical and subcortical grey matter increases during pre-adolescence and then diminishes post-adolescence. Significant changes in cortical thickness are known to occur in children and adolescents 7 to 16 years of age and neurocognitive abilities develop in concert with these changes (Shaw et al., 2006).

Memory in healthy children develops in a linear and predicable manner and is reported to be stable. It is important to note, however, that a portion of what appears to be "stability" in developmental studies actually relates to how, within normative studies, standard scores are adjusted for age and inherent variability within the control sample; the "normative" data somewhat masks variability. Age-corrected standard scores, while not necessarily "stable," generally have a limited amount of variability of scores surrounding them, which makes it possible to predict future academic and neurocognitive functioning from current ability level. Bull, Espy, and Wiebe (2008) conducted a longitudinal examination of short-term memory, working memory, and executive functioning in preschool children (mean age = 4 years, 6 months) and were able to predict academic achievement at 7 years of age. Visual-spatial short-

term memory span was shown to specifically predict mathematical ability at age 7, while short-term and working memory predicted mathematical achievement at every time point. Children with better digit span and executive function skills also had a head start in math and reading, abilities which were maintained over the first three years of primary school.

Although child and adolescent brains develop rapidly, neurocognitive abilities tend to be stable into adulthood. Townes and colleagues (2008) conducted a longitudinal study where they annually assessed the neurocognitive abilities in 503 children for 8 consecutive years. The ages of the children at the beginning ranged from 8 to 11.9 years; at the end of the studies these children, and now adolescents, ranged in age from 16 to 19.9 years of age. Exploratory factor analyses suggested that neurocognitive structures are expressed and predictable by adolescence. Longitudinal correlations between ages 5 to 12 for working memory were reported to be .37, which, considering the substantial changes in brain development that are known to occur in childhood, is less variable than might be expected (Polderman et al., 2007). Although raw scores may change between these ages, tests are normed on typically developing controls so it is the percentile that is stable, not the raw score. In addition, this correlation accounts for only approximately 10% of the variance, which is relatively substantial considering the number of neurocognitive changes occurring during this time of development, many of which have yet to be fully explained in the literature. The heritability of cognitive ability appears to be relatively stable throughout adulthood (Vogler, 2006).

Memory Impairment in ASD

Compared to typically developing children, individuals diagnosed with autism tend to have increased variability of memory function throughout the course of development. Given the heterogeneity of this group it is often difficult to predict future academic achievement from

current memory functioning. Although memory in typically developing controls can also vary, there is much less variance than their ASD counterparts. As previously discussed, research has shown that memory in ASD individuals is highly variable, develops in an uneven manner, and with deficits in various aspects of memory.

For example, Southwick et al. (2011) examined memory functioning in male children and adolescents (ages 5-19) with autism and compared them to typically developing controls. They stipulated that nonverbal intellectual abilities be at 85 or greater but allowed verbal intellectual abilities to vary, which it did. The autism group mean IQ was almost a standard deviation below and much more variable than that of the control sample. These researchers found that the two groups differed in performance on tests measuring many aspects of memory including immediate and delayed, verbal and nonverbal, sequential recall, attention and concentration, associative recall, free recall, and multiple-trial learning memory. These results supported the conclusion that encoding and organization of information are the factors that limit memory performance in autism, as opposed to storage and retrieval.

Another study examined memory and learning in children with 22q11.2 Deletion Syndrome (DS; velocardiofacial syndrome), one of the most common causes of mild mental retardation and learning disability (Lajiness-O'Neill et al., 2005). They administered a measure of memory to children with 22q11.2 DS, their siblings, children with low intellectual ability, and children with autism. Children with 22q11.2 DS performed similarly to children with autism on the Composite Memory Index, Facial Memory subtest, and the Delayed Recall subtest, with an overall pattern of verbal better than nonverbal memory. The researchers concluded that deficits in facial memory in children with autism as well as children with 22q11.2 may represent ventral

temporal pathway disruptions, such as between the parahippocampal/hippocampal regions and the fusiform gyrus.

The Importance of Global Assessment Metrics in Measuring Memory: The Test of Memory and Learning (TOMAL)

The Test of Memory and Learning (TOMAL; Reynolds & Bigler, 1994) is highly useful for measuring longitudinal stability of memory in autism due to its robust child norms and structure of memory composition. Specifically, the TOMAL dichotomizes memory into verbal and nonverbal domains, and throughout the literature autism is often associated with intact nonverbal ability and dysfunctional verbal ability. In addition, it has been successfully used in previous studies examining memory in participants with ASD (Southwick et al., 2011; Lajiness-O'Neill et al., 2005).

Given what is known about cognitive functioning, it is not possible to measure "pure memory" without tapping into other aspects of cognitive functioning (e.g., executive function). This is important to note since the majority of studies examining memory in autism have utilized a singular memory task, which may not be effective or particularly informative about clinical implications of memory impairment or the everyday functioning of the individual with ASD (Moritz, Ferahli, & Naber, 2004). One main advantage of omnibus memory assessment batteries such as the TOMAL is that they provide indices of multiple types of memory, not just a singular measure and may therefore be a more appropriate assessment instrument.

Stability of Cognitive Ability in Childhood

There are a number of longitudinal studies demonstrating the relative low variability of general cognitive ability relative to peers (age-corrected), such as intellectual functioning, in children (van Soelen et al., 2011; Gow et al., 2011; Davis, Haworth, & Plomin, 2009). However,

there are no true longitudinal studies that have examined the stability of memory function over childhood development. Almost all studies in this area are cross-sectional by design and have assessed memory at certain set-points in child development, as opposed to longitudinal assessments (Gaigg et al., 2008; Henderson et al., 2009; Sinzig, Morsch, Bruning, Schmidt, & Lehmkuhl, 2008; Wilkinson et al., 2010; Williams et al., 2006b).

A number of genetic studies have assessed and demonstrated the stability of working memory (Polderman et al., 2007) and vulnerability of memory function in emerging neuropsychiatric conditions (Maziade et al., 2011); however, no study has assessed an omnibus measure of memory stability using a battery of memory tests. As previously stated, given the likely overlap of cognitive functioning that can contribute to memory impairment, an omnibus measure of memory provides the advantage of assessing several aspects of cognition at once. Often, the only longitudinal studies can be inferred by data from control groups used to study conditions such as prematurity (Rose, Feldman, & Jankowski, 2005), epilepsy (Gonzalez, Mahdavi, Anderson, & Harvey, 2012), head injury (Crowther et al., 2011), schizophrenia (Ross, Wagner, Heinlein, & Zerbe, 2008), or from disorders such as cancer and children receiving radiation therapy (Mabbott et al., 2011; Spiegler, Bouffet, Greenberg, Rutka, & Mabbott, 2004). For example, Spiegler and colleagues tracked 25 children with posterior fossa tumors who received radiation therapy; they did not observe a decline in verbal memory, although there were other cognitive areas that did decline (i.e., visual memory, verbal fluency, executive functioning).

One important consideration in assessing memory longitudinally is the question of stability of performance across administrations. Knowing whether changes over time represent the result of practice effects or true changes in performance can help avoid erroneous

conclusions regarding a participant's changes in cognitive functioning. Calamia, Markon, and Tranel (2012) conducted a meta-analysis of practice effects in neuropsychological assessment. While studies utilizing the TOMAL were not specifically examined, the researchers included a number of other memory measures and their findings apply to any neuropsychological test administered more than once. The researchers reviewed nearly 1600 individual effect sizes for changes in performance on a number of neuropsychological measures. They placed no limit on the test-retest interval, with means for individual measures ranging from 0.53 to 4.54 years. Results indicated that "the overall practice effect across tests was nearly a quarter of a standard deviation" (p. 560). However, this varied greatly across the different tests and high within-domain variability cautions against overgeneralizing with these results.

With regard to memory specifically, effect size estimates for visual memory measures were somewhat higher than most of the other domains. Calamia et al. (2012) found practice effects for some tests up to five years following initial testing, with a minimum of two to three years needed to eliminate score gains. Additionally, smaller effect sizes were seen in clinical samples, which could be problematic when using a healthy comparison group with larger practice effects (i.e., overcorrection). The authors cautioned researchers to be aware of practice effects when performing studies to avoid misinterpretation of results.

Present Study

To date, no studies have yet examined the longitudinal stability of memory in individuals diagnosed with autism using a battery of memory tests. The present study uses the TOMAL to assess memory at baseline (Time 1) in a group of ASD males compared to age- and education matched males with typical development and no diagnosable psychiatric disorder. After the TOMAL was administered at Time 1, a revised version was released (Reynolds & Voress, 2007).

In order to maintain consistency between administrations, the original TOMAL was again administered at Time 2 instead of the revised version.

This study proposed one main hypothesis separated into two parts. First, since research suggests that in healthy children cognitive functions, with memory assumed as a subset, is relatively stable from mid-childhood on. Considering the dramatic changes in childhood brain development and maturation, it was predicted that memory in the control group would remain stable (age-corrected) over approximately a three year time period, with no expected differences in index scores or on individual subtests. Second, in contrast to the control group, research has demonstrated mixed evidence of impaired memory functioning in ASD, with the only consensus that it appears to be variable. Thus, it was predicted that memory performance in the ASD group would be variable over time.

Method

Participants

This study included 50 male participants who had been rigorously diagnosed for ASD using DSM-IV-TR criteria and had a Full Scale IQ > 65, as measured by the Wechsler Abbreviated Scale of Intelligence (WASI), Wechsler Adult Intelligence Scale - 3rd ed. (WAIS-III), or Wechsler Intelligence Scale for Children - 3rd ed. (WISC-III). These individuals were compared to 92 age-matched male controls. All participants were recruited from community resources including parent support groups, youth groups and clinic social-skills groups.

This study was approved by the Brigham Young University and the University of Utah Institutional Review Boards. Procedures were fully explained to participants and/or their legal guardians. Written and informed consent was obtained by participants and respective

parents/guardians prior to experimentation. Additional details concerning the sample from which these data were obtained may be found in Southwick et al. (2010).

For individuals in the ASD group, diagnosis was made on the basis of the Autism Diagnostic Observation Schedule (Lord, Rutter, DiLavore, & Risi, 1999) and confirmed by independent expert clinical evaluation by a board-certified child psychiatrist. Participants were excluded if they were found to have associated neurologic, genetic, infectious, or metabolic disorders (including fragile X syndrome), tuberous sclerosis, or cytomegalovirus. These exclusions were based upon a physical examination, neurologic history, and chromosomal analysis performed by a qualified physician.

The control group was comprised of neuropsychiatrically normal and medically healthy community volunteers. Potential applicants were screened via a telephone interview. Exclusionary criteria included a history of birth or any developmental abnormality, traumatic brain injury, learning or language disability, history of or current neuropsychiatric disorders, alcoholism, or family history of first-degree relatives diagnosed with autism.

Study Design

This study employed a quasi-experimental design, meaning that the investigator had little or no control over the assignment of treatments as it was impossible to assign children to the ASD or control groups. Thus, the quasi-independent variables were the two groups. Performance on the TOMAL was the dependent variable, as each group was observed for changes on the indices and subtests over time. The largest nuisance, or extraneous, variable was the enrollment of the ASD participants in outpatient social-skills training (discussed in depth in the following section). Gender was a controlled variable, as all participants were male.

Instruments

Test of Memory and Learning (TOMAL). The Test of Memory and Learning (TOMAL; Reynolds & Bigler, 1994) is a standardized comprehensive memory battery normed for children 5 to 19 years of age. The core battery is comprised of 10 subtests (5 verbal, 5 nonverbal) that are organized into a Verbal (VMI) or Nonverbal Memory Index (NVMI). These scales are combined to form the Composite Memory Index (CMI). A Delayed Recall Index (DRI) is also available and is based on stimuli recall of the first four subtests. Relative subtest performances can be directly compared via the conversion of raw to scaled scores. Refer to Table 1 for a breakdown of TOMAL Indices and subtest composition. This investigation focused exclusively on the Index scores, but since each Index is made up of subtests, for reference purposes, all subtest data are included in the Appendix. All scores are reported as age-corrected standard scores. For those subjects 20 and older, the 19 year old normative values were used for calculating standard scores.

The VMI consists of five core subtests: Memory for Stories (MFS), a task in which the examinee recalls details of three short stories read aloud by the examiner; Word Selective Reminding (WSR), a verbal free recall task where the examinee learns a wordlist and repeats it and is reminded of words they left out in each case; Object Recall (OR), a task in which the examiner presents a series of named pictures which the examinee must verbally recall; Digits Forward (DF), rote recall of a sequence of numbers; and Paired Recall (PR), a verbal paired associates learning task. Three supplementary subtests are also presented that can be substituted for a core subtest when it is not given. These include: Letters Forward (LF), a rote recall of a sequence of letters; Digits Backward (DF), reversed recall of a sequence of numbers; and Letters Backward (LB), a similar subtest that uses letters.

NVMI includes: Facial Memory (FM), a subtest in which the examinee must recognize and identify black and white photos of faces from a set of distracters; Visual Selective Reminding (VSR), dots presented on a card; Abstract Visual Memory (AVM), a test of immediate recall of meaningless figures; Visual Sequential Memory (VSR), sequential recall of a series of meaningless geometric figures; and Memory for Location (MFL), spatial recall of large dots. Manual Imitation (MI) is supplemental subtest that requires the examinee to reproduce sequential hand movements presented by the examiner.

Table 1

Test of Memory and Learning (TOMAL) Indices and Subtest Composition

VMI	NVMI	DRI
Memory for Stories	Facial Memory	Memory for Stories
Word Selective Reminding	Visual Selective Reminding	Facial Memory
Object Recall	Abstract Visual Memory	Word Selective Reminding
Digits Forward	Visual Sequential Memory	Visual Selective Reminding
Paired Recall	Memory for Location	
Letters Forward*	Manual Imitation*	
Digits Backward*		
Letters Backward*		

Note. VMI = Verbal Memory Index; NVMI = Nonverbal Memory Index; DRI = Delayed Recall Index.
* Optional subtests.

The TOMAL was normed on a normative sample of 1,342 children matched to 1990 United States of America Bureau of Census data. The TOMAL manual reports median internal consistency of subtests (across age) ranging from .74 to .98. Median coefficient alpha for the VMI (.96), NVMI, CMI and DRI (.95, .97 and .85, respectively) are also quite high. Test-retest reliability was examined using a sample of 35 children (8-11 years old) assessed 4 and 9 weeks

apart. Pearson product-moment correlation coefficients of subtests and indices (.71-.92) suggest a high degree of stability over time.

Wechsler Adult Intelligence Scale – 3rd Edition (WAIS-III). This is a measure of general intellectual function in older adolescents and adults aged 16 to 89 years. The WAIS-III manual reports test-retest reliability ranging from .92 to .97 and internal consistency ranging from .88 to .97. It is comprised of 14 subtests (2 optional) within four factor-based indices (Verbal Comprehension, Working Memory, Perceptual Organization and Processing Speed), which yield a Full Scale (FSIQ), a Verbal (VIQ) score and a Performance Scale (PIQ) score.

Wechsler Intelligence Scale for Children – 3rd Edition (WISC-III). The WISC-III is a measure of general intellectual function used in children and adolescents aged 6 to 16 years. It is divided into two scales, a Verbal and a Performance Scale. Internal consistency ranges from .80 to .97 and reliability ranges from .74 to .95. Correlations with the WAIS-III are high (.88, .78, and .88 for VIQ, PIQ and FSIQ, respectively). The two measures appear to yield comparable IQs and measure similar constructs.

Wechsler Abbreviated Scale of Intelligence (WASI). This instrument was developed as an IQ screening measure that assesses constructs similar to the WAIS-III and WISC-III. The manual reports concurrent validity ranging from .72 to .92.

Autism Diagnostic Observation Schedule (ADOS). The ADOS is a semi-structured diagnostic interview to assess behaviors related to autism or Autistic Spectrum Disorders based on DSM-IV and ICD-10 criteria. This measure demonstrates adequate inter-rater and test-retest reliability as well as internal validity and is considered the "gold standard" for ASD assessment (Lord et al., 1999).

Procedure

The ADOS was administered to all participants as a part of the initial diagnostic examination process (see Southwick et al., 2011). Measures of intelligence and the TOMAL were administered by doctoral-level graduate students trained in test administration. TOMAL administration was performed at Time 1 and again at Time 2, using the original TOMAL, without any modification except adjustments for differences in age.

Data Analysis

Assessments were collected and analyzed in the following ways for each of the hypotheses using primarily SPSS 17.0 for Windows, including various analysis of variance and correlational techniques. Reliable change indices (RCI; Jacobson & Truax, 1991) were also calculated for each TOMAL Index and scaled subtest score. RCI measures change by subtracting Time 1 score from Time 2. The resulting number is divided by the standard error of difference (S_{diff}), which is derived from the standard error of the measurement (S_E) using the following formula:

$$S_{diff} = \sqrt{2(S_E)^2}$$

Below is the formula used to calculate reliable change (Jacobson & Truax, 1991):

$$RCI = \frac{X_2 - X_1}{S_{diff}}$$

Scores greater than 1.96 ($p < .05$) are considered to have improved to a clinically significant degree. Those less than -1.96 ($p < .05$) are considered to have deteriorated to a clinically significant degree. The proportion of cases that surpassed the aforementioned cutoff score were examined for each group.

Tables and figures were created with SigmaPlot 11.0 for Windows. Time 1 refers to the initial assessment and Time 2, the follow-up assessment.

Due to the attrition rate in the TDC group, which reduced statistical power, although subtest data will be reported in the appendix, detailed subtest analyses will not be the focus of this study. Instead, this study will focus on the overall memory performance index scores on the TOMAL.

Correlational Analyses

Bivariate correlational analyses were conducted to assess the relationship between IQ and TOMAL variables, including subtest and index scores, at Time 1 and Time 2. Given the substantial number of correlational analyses, a modified Bonferroni test proposed by Larzelere and Mulaik (1977) was then used to attenuate family-wise error. Variables of interest were first examined via a two-tailed bivariate correlational analysis. The resulting correlations were arranged by increasing numerical order by p values. The total number of contrasts (*k)* and contrast order number (i) are entered into the equation below to calculate an adjusted critical value (α') for each correlational comparison.

$$\alpha' = \frac{.05}{(k - i + 1)}$$

The original bivariate correlations are reexamined for significance by comparing the corresponding coefficient *p* value with the adjusted critical value (α'). This procedure is continued until the *p* value of a given contrast exceeds α'. At that point the correlation is declared to be nonsignificant (as well as any correlations beyond that point) and testing is discontinued.

Because of attrition in the TDC group, diminished power for examining correlational relationships was present and while both index and individual TOMAL subtest scores were examined in correlational matrices, only the index scores will be fully examined. Subtest TOMAL memory correlation matrices may be found in the Appendix section.

Results

Descriptive Analyses

Table 2 shows a total of 142 participants that were tested at Time 1 [50 typically developing controls (TDC)] with a 40% retention rate was achieved at Time 2. Ninety-two participants diagnosed with an autism spectrum disorder (ASD) were examined at Time 1 with a 55.43% retention rate at Time 2.

At Time 1, the TDC group had a mean age of 15.39 years (SD = 6.17); ASD group mean age was 14.67 years (SD = 8.12). Non-significant age differences were noted at Time 1, $F(1, 141) = .294$, $p = .589$. Significant differences were observed for Verbal and Non Verbal IQ indices (tested at Time 1), where both intelligence indices were higher for the TDC group, $F(2, 135) = 17.57$, $p = .000$. Refer to Table 3 for specific values and Figures 1 and 2 for frequency distributions. As can be viewed in the figures, the lowest memory scores occurred in the youngest participants. However, because of the limited sample size of older participants further analyses on age by TOMAL performance could not be completed in this study.

IQ and TOMAL Index Correlations

When both IQ indices (verbal and non-verbal) were correlated with TOMAL indices and age at Time 1 and Time 2, significant correlations were found between all TOMAL indices and both VIQ and NVIQ for the TDC group. For the ASD group, all TOMAL indices were significantly correlated with VIQ and all but two TOMAL indices (Time 1 DRI and Time 2

VMI) were significantly correlated with NVIQ. See Tables 4 and 5. Note that a modified Bonferroni test was used to attenuate family-wise error.

Table 2

Age Characteristics of TDC and ASD Groups

	Age at Time 1			N	
Group	Mean	SD	Range	Time 1	Time 2
TDC	15.39	6.17	21.08	50	20
ASD	14.67	8.12	40.17	92	50

Note. TDC = Typically Developing Controls; ASD = Autism Spectrum Disorder.

Table 3

IQ Descriptives at Time 1

	VIQ		NVIQ	
	TDC	ASD	TDC	ASD
Mean	115.55	104.09	119.50	103.60
SD	16.15	21.87	16.18	19.50
Min.	90.00	66.00	88.00	70.00
Max.	151.00	145.00	152.00	145.00
Range	61.00	79.00	64.00	75.00
Skew	0.41	0.07	-0.19	0.13
Kurtosis	-0.24	-0.96	0.16	-0.93

Note. TDC = Typically Developing Controls; ASD = Autism Spectrum Disorder.

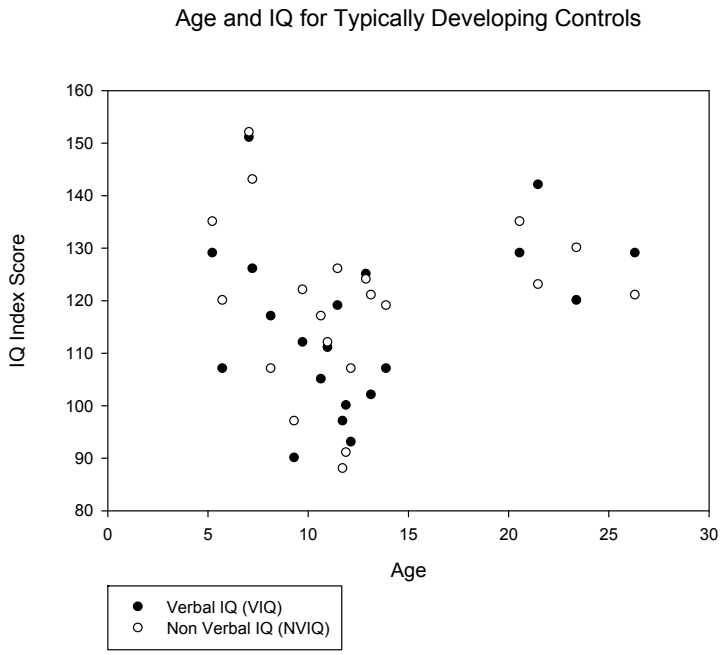

Figure 1. Age and IQ for typically developing controls.

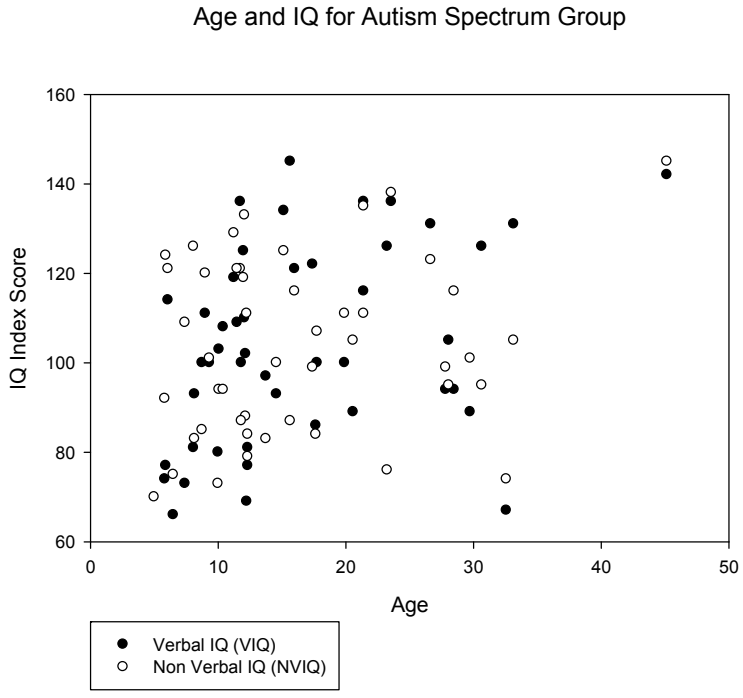

Figure 2. Age and IQ for autism spectrum disorder group.

Table 4

Correlations between IQ and Time 1 and 2 TOMAL Indices for TDC

	AGE-1	AGE-2	VIQ	NVIQ	CMI-1	VMI-1	NVMI-1	DRI-1	CMI-2	VMI-2	NVMI-2	DRI-2
AGE-1	-	.99*	.26*	.07	-.14	-.02	-.23	.09	-.30*	-.14	-.40**	-.09
AGE-2		-	.26*	.07	-.14	-.02	-.23	.08	-.29*	-.14	-.38**	-.08
VIQ			-	.59*	.60*	.64*	.49**	.57**	.54**	.56**	.46**	.53**
NVIQ				-	.62*	.52*	.61**	.44**	.55**	.45**	.57**	.42**
CMI-1					-	.94**	.94**	.81**	.86**	.80**	.82**	.71**
VMI-1						-	.77**	.80**	.84**	.82**	.76**	.73**
NVMI-1							-	.71**	.78**	.68**	.78**	.60**
DRI-1								-	.67**	.65**	.60**	.65**
CMI-2									-	.94**	.95**	.87**
VMI-2										-	.77**	.87**
NVMI-2											-	.75**
DRI-2												-

Note. AGE-1 = Chronological age at Time 1; AGE-2 = Chronological age at Time 2; VIQ = Verbal IQ; NVIQ = Nonverbal IQ; CMI-1 = Time 1 Composite Memory Index; VMI-1 = Time 1 Verbal Memory Index; NVMI-1 = Time 1 Non Verbal Memory Index; DRI-1 = Time 1 Delayed Recall Index; CMI-2 = Time 2 Composite Memory Index; VMI-2 = Time 2 Verbal Memory Index; NVMI-2 = Time 2 Non Verbal Memory Index; DRI-2 = Time 2 Delayed Recall Index.

*Significant at $p < 0.05$ with modified Bonferroni correction.

** Significant at $p < 0.00$ with modified Bonferroni correction.

Table 5

Correlations between IQ and Time 1 and 2 TOMAL Indices for ASD

	AGE-1	AGE-2	VIQ	NVIQ	CMI-1	VMI-1	NVMI-1	DRI-1	CMI-2	VMI-2	NVMI-2	DRI-2
AGE-1	-	.99**	.36*	.19	-.06	.12	-.21	.27	-.28	-.06	-.40*	-.01
AGE-2		-	.37*	.20	-.06	.12	-.20	.26	-.25	-.06	-.37*	-.01
VIQ			-	.49**	.54**	.62**	.39**	.53**	.45**	.50**	.34*	.53**
NVIQ				-	.50**	.38**	.52**	.27	.35*	.28	.38*	.31*
CMI-1					-	.93**	.92**	.73**	.82**	.76**	.76**	.67**
VMI-1						-	.71**	.77**	.79**	.78**	.69**	.72**
NVMI-1							-	.58**	.73**	.63**	.73**	.53**
DRI-1								-	.54**	.56**	.45**	.55**
CMI-2									-	.93**	.93**	.87**
VMI-2										-	.73**	.86**
NVMI-2											-	.73**
DRI-2												-

Note. AGE-1 = Chronological age at Time 1; AGE-2 = Chronological age at Time 2; VIQ = Verbal IQ; NVIQ = Nonverbal IQ; CMI-1 = Time 1 Composite Memory Index; VMI-1 = Time 1 Verbal Memory Index; NVMI-1 = Time 1 Non Verbal Memory Index; DRI-1 = Time 1 Delayed Recall Index; CMI-2 = Time 2 Composite Memory Index; VMI-2 = Time 2 Verbal Memory Index; NVMI-2 = Time 2 Non Verbal Memory Index; DRI-2 = Time 2 Delayed Recall Index.

*Significant at $p < 0.05$ with modified Bonferroni correction.

** Significant at $p < 0.00$ with modified Bonferroni correction.

TOMAL Index Scores

Scores from the four TOMAL indices at both time points — Composite Memory (CMI), Verbal Memory (VMI), Non Verbal Memory (NVMI) and Delayed Recall (DRI) — were subjected to a repeated measures analysis of variance with supplementary polynomial contrasts to examine index score stability across time (Tables 6 and 7). Main effects were noted for group (TDC versus ASD); $F(1, 58) = 35.25$, $p = .000$. The TDC group demonstrated higher scores than the ASD group on all indices; $M_{diff} = 35.2$, $SE = 2.43$, $p = .000$.

Main effects were also noted when comparing Time 1 and Time 2 administration of each index; $F(7, 52) = 4.29$, $p = .001$. For the control group, polynomial contrasts revealed non-significant differences for all four indices; CMI ($M_{diff} = -3.58$, $SE = 2.01$, $p = .092$), VMI ($M_{diff} = -2.21$, $SE = 2.26$, $p = .341$), NVMI ($M_{diff} = -4.79$, $SE = 2.76$, $p = .100$), and DRI ($M_{diff} = -3.53$, $SE = 1.75$, $p = .069$). The ASD group demonstrated significant differences for CMI ($M_{diff} = -5.78$, $SE = 1.68$, $p = .001$), VMI ($M_{diff} = -7.37$, $SE = 1.84$, $p = .000$) and DRI ($M_{diff} = -8.08$, $SE = 1.91$, $p = .000$). Non-significant differences were noted for NVMI ($M_{diff} = -3.03$, $SE = 2.11$, $p = .185$). No significant interactions were found $F(7, 52) = 4.56$, $p = .463$. See Figure 3.

Subtest score findings are reported in Appendix Tables 9-20 and Figures 4-7.

Table 6

TOMAL Index Scores across Time for TDC Group

	TDC					
	TOMAL 1			TOMAL 2		
	Mean	*SD*	*Range*	*Mean*	*SD*	*Range*
CMI	107.84	11.16	37	111.42	10.29	32
VMI	105.42	12.67	48	107.63	11.47	39
NVMI	109.16	12.58	46	113.95	11.31	42
DRI	103.40	8.41	31	106.75	7.25	28

Note. CMI = Composite Memory Index; VMI = Verbal Memory Index; NVMI = Non Verbal Memory Index; DRI = Delayed Recall Index.
*Significant at $p = .000$.

Table 7

TOMAL Index Scores across Time for ASD Group

	ASD					
	TOMAL 1			TOMAL 2		
	Mean	*SD*	*Range*	*Mean*	*SD*	*Range*
CMI	82.86	17.18	66	88.49	18.34*	69
VMI	81.62	17.43	71	88.51	18.85*	68
NVMI	85.83	17.40	66	88.81	18.88	78
DRI	87.28	13.16	55	94.49	13.13*	52

Note. CMI = Composite Memory Index; VMI = Verbal Memory Index; NVMI = Non Verbal Memory Index; DRI = Delayed Recall Index.
*Significant at $p = .000$.

RCI and Index Scores

Reliable Change scores were calculated for each of the TOMAL indices (see Table 8). These scores tended to support repeated measures ANOVA results that indicated Composite (CMI), Verbal (VMI) and Delayed Recall Index (DRI) improvement across time (Figure 3). On the CMI, 36.84% of the typically developing controls and 37.21% of individuals diagnosed on the autism spectrum showed reliable improvement by surpassing the 1.96 cutoff point. However, 15.70% of TDC individuals demonstrated reliable decline, whereas the ASD group did not.

For the VMI, 26.32% of TDCs reliably improved versus 40.23% of ASD participants. NVMI improvements were similar for each group; TDC (31.58%) and ASD (32.56%) which is consistent with previously reported non-significant ANOVA differences. The DRI had the most compelling RCI results; no TDC scores reliably changed, in contrast to the 27.66% of ASD participants that improved across time.

Table 8

Reliable Change Index Improvement and Deterioration Effects for TOMAL Index Scores

	Group			
	TDC		ASD	
Index	RCI (*n*)	RCD (*n*)	RCI (*n*)	RCD (*n*)
CMI	36.84 (*19*)	15.70 (19)	37.21 (*43*)	0.00 (43)
VMI	26.32 (19)	15.79 (19)	40.23 (47)	17.02 (47)
NVMI	31.58 (19)	5.26 (19)	32.56 (43)	16.28 (43)
DRI	0.00 (20)	0.00 (20)	27.66 (47)	0.00 (47)

Note. Numbers indicate percentage of cases surpassing 1.96 on RC. RCI = Reliable Change Index-Improvement; RCD = Reliable Change Index-Deterioration. CMI = Composite Memory Index; VMI = Verbal Memory Index; NVMI = Nonverbal Memory Index; DRI = Delayed Recall Index.

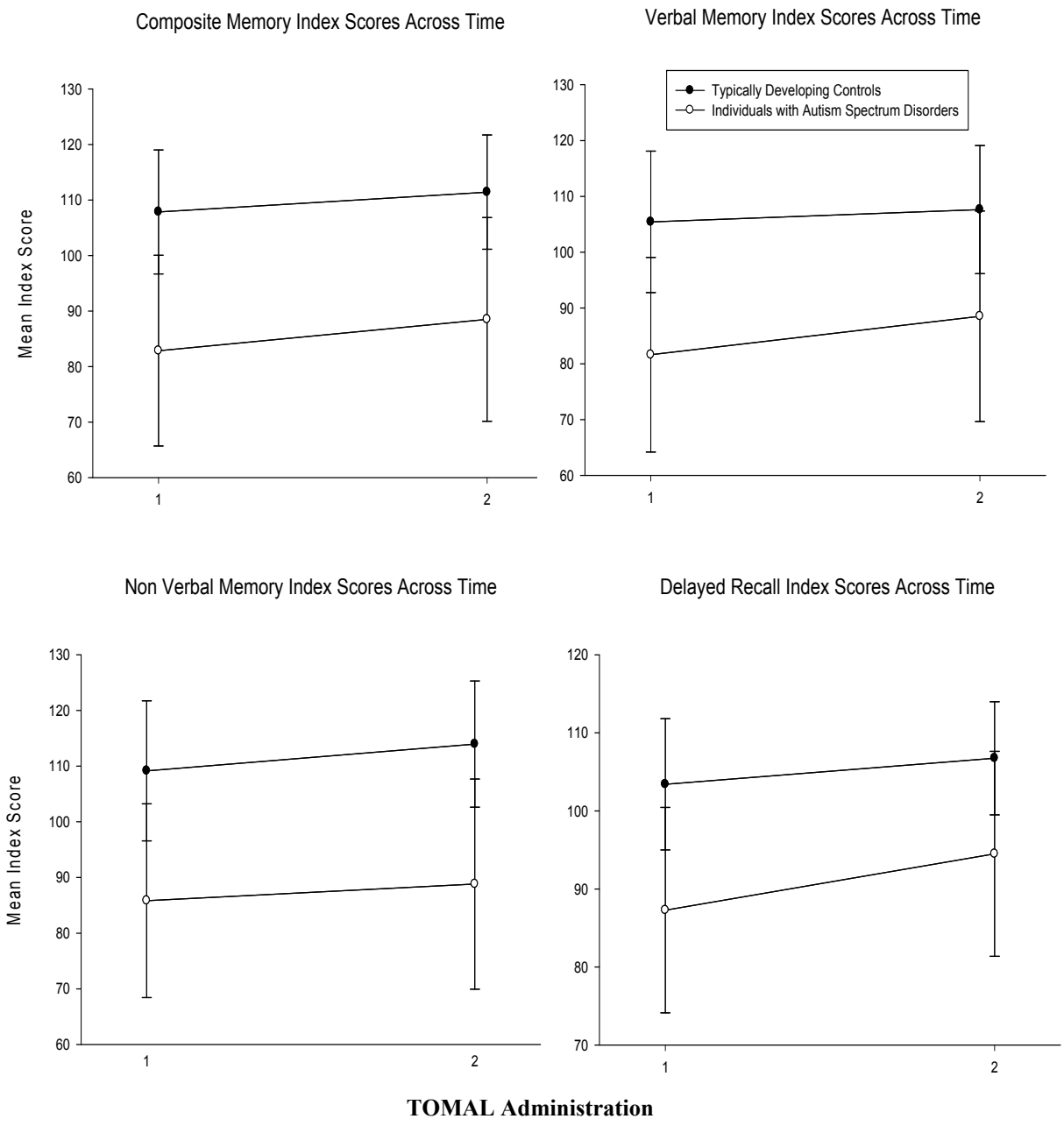

Figure 3. Index scores across time for TDC and ASD groups. Significant main effects across administration are depicted, $F(7, 52) = 4.29$, $p = .001$. Memory remained stable for TDC subjects on all four indices while the ASD group showed improvements on CMI, VMI, and DRI. No significant interactions were found.

Discussion

The current study examined one main hypothesis with two parts. First, since research suggests that memory in healthy children is quite stable over the transition of older childhood and adolescence and considering the dramatic changes in childhood brain development and the emotional and biological issues of the emergence of puberty and maturation, it was predicted that memory in the control group would remain stable over the three-year time period, with no essentially no differences in index scores or on individual subtests. Second, in contrast to the control group, research has demonstrated mixed evidence on impaired memory functioning in autism, with the only consensus that it appears to be variable. Because variability in memory performance may adversely influence development of memory ability, it was predicted that memory performance in the ASD group would be more variable over time and likely less stable than observed in controls.

Both groups (individuals diagnosed with autism and healthy controls) were compared with regard to age and intelligence and other demographic variables. Results indicated that the groups were similar in age, but those in the TDC group tended to have higher scores on measures of intellectual functioning. Despite the differences in overall IQ, Dennis et al. (2009) have argued that in disorders where intellectual disability may be a clinical feature of the disorder, such as autism (Feero, Guttmacher, Mefford, Batshaw, & Hoffman, 2012), caution should be used in over controlling for differences in IQ. Given this research, in the current investigation no further statistical controls for IQ were implemented.

Bivariate correlational analyses were first conducted to assess the relationship between IQ, age, and TOMAL variables at Time 1 and Time 2 for each group, comparing just the overall index scores. A modified Bonferroni test was used to attenuate family-wise error since there were a significant number of comparisons completed. Significant correlations were found between all TOMAL indices and both VIQ and NVIQ for the TDC group. For the ASD group, all TOMAL indices were significantly correlated with VIQ and all but two TOMAL indices (Time 1 DRI and Time 2 VMI) were significantly correlated with NVIQ. This demonstrates very little difference between the two groups with regard to memory and IQ correlations. Future research is recommended and discussed in detail below.

A repeated measures analysis of variance was then used to examine TOMAL index and subtest performance comparing Time 1 with Time 2. The control group demonstrated significantly higher scores on all TOMAL index scores (Composite Memory Index, Verbal Memory Index, Non-Verbal Memory Index, and Delayed Recall Index) than the ASD group, as well as non-significant differences over time on all indices. This is consistent with research suggesting memory is generally stable in children over time (Bull et al., 2008; Townes et al., 2008).

When Time 1 and Time 2 TOMAL differences were examined using the Reliable Change Index (RCI). As outlined above, the RCI addresses clinical meaning change by examining difference scores with the standard error of the measure. The metric cut-off for significance is ± 1.96 for a p value of .05. Any scores that fall above or below this cut-off represent reliable change or reliable deterioration, respectively, that is beyond practice effect. Any fluctuations of scores within this critical value are not considered

clinically meaningful. In this study, changes from Time 1 to Time 2 TOMAL scores demonstrated minimal differences. On the Composite Memory Index, 36.84% of the control group showed reliable improvement and 15.70% demonstrated reliable decline. On the Verbal Memory Index, 26.32% of this group demonstrated reliable improvement with 15.79% showing reliable decline. On the Non-Verbal Memory Index, 31.58% showed reliable improvement while 5.26% demonstrated reliable decline. Finally, on the Delayed Recall Index there was no reliable improvement or reliable decline in the control group.

Cross-sectional studies of childhood memory performance generally shows that memory in typically developing children develops in a linear and predictable manner and, in the absence of brain insults, neurocognitive abilities tend to remain relatively stable throughout childhood into adulthood. For example, in one study, researchers were able to predict academic achievement at 7 years of age based on performance on measures of cognitive functioning 3 years earlier (Bull et al., 2008). In another study by Townes and colleagues (2008) that involved testing over 8 consecutive years, results indicated that neurocognitive abilities appear stable by adolescence. However, these studies did not employ an omnibus measure of memory like the TOMAL. The present study adds additional evidence to support this concept, with repeated analyses and RCI data showing that memory at Time 1 was predictive of Time 2, with little absolute group differences between the two time points. Note that RCI data is reported as percentages for improved and declined for each group; in order to further interpret this data, the standardized measurement instrument (TOMAL) would've had to have norms established for populations with ASD as well as without, which it does not.

The second part of the hypothesis posited that participants in the ASD group would demonstrate variable memory across time. In support of greater Time 1 versus Time 2 differences were the results from the Reliable Change Index analyses. A larger percentage of participants in the ASD group showed a reliable improvement of scores on all four indices than participants in the control group, most significantly on the Verbal Memory Index. Although the percentage of improvement on the CMI and NVMI were only marginally greater than the control group, 40.23% of the ASD group showed improvement on the VMI (as compared to 26.23% of the TDC group) and 27.66% of the ASD group showed improvement on the DRI (as compared to 0% of the TDC group).

Results from the repeated measures analyses for the ASD group also indicated less stability of memory than the TDC group. Across administrations, the ASD group demonstrated improvements on the Composite Memory Index, Verbal Memory Index, and Delayed Recall Index. They did not improve on the Non-Verbal Memory Index. This suggests that, in addition to some variability of memory, individuals diagnosed with autism exhibited some improvements in verbal memory and delayed recall over time while non-verbal memory remains impaired. Problems with persistent reduced non-verbal memory have been reported by others. This is consistent with previous research including that of Minshew & Goldstein (2001) showing that individuals with autism performed worse on tasks of visual memory than controls, and with results from Williams et al. (2005) who found that those individuals showed deficits in spatial working memory, but not verbal working memory. However, to provide further evidence that research on memory in autism has greater variability than in typical developing individuals, some previous research has demonstrated lower verbal and delayed memory,

such as Kuschner, Bodner, and Minshew (2009) who found no difference between individuals with autism on controls on a task of visual memory and Williams et al. (2006b), who found that individuals in the autism group performed significantly worse some aspects of verbal memory.

Returning to the results of the current study, not only did the ASD subjects exhibit improvements on three of four indices, the standard deviations within the ASD group were considerably more substantial, ranging from 13.13 (DRI, Time 2) to 18.88 (NVMI, Time 2). By comparison, the standard deviations within the TDC group ranged from 7.25 (DRI, Time 2) to 12.67 (VMI, Time 1). Note that the highest standard deviation in the TDC group is lower than the lowest standard deviation within the ASD group. This demonstrates the high level of variability of scores within the ASD group and offers further support to the hypothesis of memory instability over time in autism.

Correlational data for the ASD group was somewhat contradictory to the previous findings. In contrast to the TDC group, the ASD group demonstrated a much higher number of correlations; 44 between IQ and TOMAL Index scores (as compared to 15 significant IQ and TOMAL Index correlations). This appears to suggest better stability of scores for the ASD group and warrants further investigation.

This study highlights the importance of examining longitudinal memory in autism spectrum disorders, and fills a conspicuous gap in current literature, as there is no research to date that has examined the stability of memory ability in such individuals over time. Although previous research has suggested that memory deficits may be central to the disorder (Boucher et al., 2012), the specific type and course of such deficits has not be examined longitudinally. The etiology and underlying neuropathology of autism is

not yet clearly understood so it is not surprising that we do not yet fully understand the deficits that characterize the disorder.

However, speculation has been that in individuals diagnosed with ASD, abnormalities in certain brain structures may be more common than they are in healthy controls. Current theories of memory deficits have focused on the prefrontal cortex and the hippocampus (Boucher et al., 2012), although the amydala, cerebellum, and fusiform gyrus have also been implicated. Many of these structures are involved in some way in facial processing as well as facial memory, a specific type of non-verbal memory impairment common in ASD. It is not surprising then, that individuals diagnosed with ASD showed variable memory improvements, with non-verbal falling behind verbal memory. In this study, the TOMAL includes a non-verbal subtest of Facial Memory. ASD participants performed worse on this subtest than TDC participants (mean test score of 7.30 vs. 10.75 at Time 1), and did not improve on this measure over time (see Appendix A). In addition, their scores at Time 2 yielded the lowest standard deviations on any non-verbal subtest, suggesting stability over time.

One question raised has to do with the source of verbal memory improvement in individuals diagnosed with ASD found in this study. Although empirical answers to this question are beyond the scope of this study, hypotheses can be made based on certain study characteristics and the results obtained. It may be that social skills training may have positively influenced the development of memory in autism. Probably the majority, if not all, of the ASD participants in this study also participated in on-going social skills training (either privately under the direction of a therapist or through special education programs), and all were likely receiving some form of treatment. Controls did not

receive any special education services although all attended school. Differences in history related to therapeutic interventions between Time 1 and Time 2 for participants may have affected the Time 2 score discrepancies between the ASD group and the control group.

For some children with ASD, a key goal in the social skills training would have been to improve verbal communication, which is a diagnosed key element with autism spectrum disorders. The ASD participants demonstrated improvement primarily on the TOMAL verbal memory tests between administrations, and it could be a reflection of overall treatment. It is possible that their participation in social skills training (verbal communication in particular) directly affected their verbal memory functioning. However, since social skills training was not quantified in this study, the effects of such training cannot be directly assessed at this time.

Many of the ASD subjects were treated with psychoactive medications. Some were on psychostimulant medications, which have been known to improve working memory (Wong & Stevens, 2011; Pietrzak, Mollica, Maruff, & Snyder, 2006), but since dose, time of administration, or other aspects of medication were not systemically controlled for in this study, issues of medication effects could not be further explored.

Lastly, as noted in Figures 1 and 2, the lowest memory scores occurred in the youngest participants. However, because of the limited sample size of older participants further analyses on age by TOMAL performance could not be completed in this study.

Limitations

The current study has a number of limitations that should be taken into consideration when interpreting the results. First, there is restricted generalizability across

constructs. Due to sample characteristics, the results are limited to males diagnosed with autism who have a FSIQ greater than 65. Since there are potential sex differences in memory (Munro et al., 2012), the fact that females were not included limits the generalizability of the current findings. Furthermore, a large segment of the clinical autism population has some level of intellectual impairment, further limiting the generalizability of the current findings.

It should also be noted that there may be social-cultural influences on the ASD sample used in this study due to unique characteristics of the religious make-up of the sample. Most participants were members of the Church of Jesus Christ of Latter-Day Saints, an organization that fosters social development and interaction, emphasizing positive family values, socialization and group inclusion with a variety of community resources, potentially not available to the general public. While these values are likely an advantage to the general wellbeing of individuals with ASD, it could represent a unique limitation to this study, making the findings region specific, since in addition to community and social resources and availability to social skills training, ASD participants may have had disproportionately greater access to these experiences than ASD participants in the general population.

There was substantial attrition between time 1 and 2; in cohort studies (such as the current one), high attrition is a serious potential threat to validity. Although demographic comparisons suggest that this did not adversely influence overall age or IQ between the two time points, part of the control findings may have been that those who remained in the study were selectively more cognitively stable. This may have artificially increased the differences between TDC and ASD groups.

Another limitation involves participant history. Given the longitudinal nature of this study, an event that occurs between Time 1 and 2 exclusively within one group may confound results. In this study, as already mentioned there were no controls over intervening experiences either within the controls or autism subjects. Many ASD individuals participated in on-going social skills training at the University of Utah on an outpatient basis. In addition, as previously stated, one of the key goals in this training was improving communication. It is possible that improvements in verbal memory on the TOMAL from Time 1 to Time 2 in the ASD group were directly related to such training. With regard to history, there is also the possibility that participants may have participated in self-help memory training either directed by a therapist and/or parent outside of this research study which may have increased their familiarity with the measure, although this is unlikely.

Although some improvement in memory function was observed in the ASD subjects, it is not known if this improvement resulted in any better social or functional skill ability. The study did not employ a metric that would permit the examination of social outcome variables with improved memory function. It may be that the improvement in memory is nothing more than practice effect (see Calamia et al., 2012) and has no relationship to something that would be considered as clinical improvement.

Future Research

Continuing longitudinal studies of memory in autism will be very important, as the current study opens the possibility of verbal memory gains in individuals diagnosed with autism, as compared to the general population. The age-limit of the original TOMAL is 19, but the revised TOMAL (Reynolds & Voress, 2007) extends to age 85.

Since the autism population overall is aging, understanding how memory function relates to aging is an important clinical topic. In order to most effectively investigate the course of memory development in these children and adolescents, a number of suggestions can be made. These will be presented systematically with examples.

Future studies should implement larger and more even sample sizes. Expanding the sample size will allow for stratification of such factors as intellectual functioning (Boucher et al., 2012), sex differences (Munro et al., 2012), co-morbid disorders including ADHD (Gargaro, Rinehart, Bradshaw, Tonge, & Sheppard, 2011) and learning disabilities (O'Brien & Pearson, 2004), and age. In the course of analyzing the data, the current study discovered that among ASD participants, the youngest had the lowest memory function scores, which may be a reflection of how developmental delays are expressed early on in ASD with some adaptation with age. A large-scale, well-funded study could recruit a significantly larger sample that would allow researchers to classify participants into subgroups to analyze differences in aspects of memory in each group. Specifically, stratifying age *a priori*, perhaps creating separate subgroups for children/adolescents (ages 5-19) and those 20 and older, would allow researchers to assess effects of age on memory development in ASD. Larger sample sizes would also allow for more detailed examination of memory subtests.

Although the neuropathology of autism has been often studied, especially from a neuroimaging perspective, the neuropathology that underlies ASD remains largely undefined (Schmitz & Rezaie, 2008). Implementing concurrent brain imaging studies as a covariate for data analysis over the course of longitudinal memory studies may help to better define this field, address the possibility of brain changes over the course of the

disorder, and help link brain structure to function in individuals with autism. This may be particularly useful when incorporating social skills training variables and may shed more light on the neuroanatomical sources of verbal memory improvements.

While there have been studies examining memory functioning in adults and even elderly with autism (Goldstein et al., 2008; Guerts & Vissers, 2012; Kuschner et al., 2009), there are no longitudinal studies that follow individuals diagnosed with autism from childhood through adulthood. Future research should continue to examine the stability of memory in autism over time, not only in childhood and adolescence but also into adulthood. It would be interesting to determine whether or not these observed memory gains continue with age. In addition, extended longitudinal studies may help determine whether such gains remain stable in the absence of social skills training. Researchers can make the time demands of longitudinal studies a little easier by combining them with cross-sectional designs; for example five groups (ages 5-8, 13-16, 21-24, 29-23, and 28-31) could be studied over the course of 5-10 years.

Memory in general has been studied in a variety of other clinical disorders. For example, Kibby and Cohen (2008) examined memory in children with co-morbid reading disability (RD) and ADHD using the Children's Memory Scale. Rapeli and colleagues (2009) utilized a longitudinal design to assess changes in memory functioning in patients being treated for opioid dependence. Of course, longitudinal research is commonly used to assess memory functioning over the course of dementia (Xie et al., 2010). Researchers can use these previous studies as models for designing methods of studying memory in autism. Specifically recommended are large sample sizes, stratifying groups into subgroups based on clinically relevant criteria (i.e., level of intellectual functioning, sex,

co-morbid disorders), longitudinal and/or cross-sectional design, brain imaging as covariate, and control of factors such as social skills training or other treatment that occurs between assessments. Such continued research may finally shed more light on the role of memory in autism, and its changes throughout the course of the disorder.

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References

Ameli, R., Courchesne, E., Lincoln, A., Kaufman, A. S., & Grillon, C. (1988). Visual memory processes in high-functioning individuals with autism. *Journal of Autism and Developmental Disorders, 18*(4), 601-615. doi:10.1007/BF02211878

American Psychiatric Association. (1952). *Diagnostic and statistical manual of mental disorders* (1st ed). Washington, DC: Author.

American Psychiatric Association. (1968). *Diagnostic and statistical manual of mental health disorders* (2nd ed). Washington, DC: Author.

American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental health disorders* (3rd ed). Washington, DC: Author.

American Psychiatric Association. (1987). *Diagnostic and statistical manual of mental disorders* (3rd ed., text revision). Washington, DC: Author.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental health disorders* (4th ed). Washington, DC: Author.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental health disorders* (4th ed, text revision). Washington, DC: Author.

American Psychiatric Association. (2012). Proposed revision: APA DSM-5 Development. http://www.dsm5.org/proposedrevisions/pages/proposed revision.aspx?rid=94

Asperger, H. (1944). Die “Autistichen Psychopathen” Kindesalter. [Autistic psychopaths in childhood] . *Archiv fur Psychiatrie und Nervenkrankheiten, 117*, 76-136. doi:10.1177/088307389601100601

Barth, C., Fein, D., & Waterhouse, L. (1995). Delayed match-to-sample performance in autistic-children. *Developmental Neuropsychology, 11*(1), 53-69. doi:10.1080/87565649509540603

Begovac, I., Begovac, B., Majić, G., & Vidović, V. (2009). Longitudinal studies of IQ stability in children with childhood autism - Literature survey. *Psychiatria Danubina, 21*(3), 310-319. Retrieved from http://www.hdbp.org/psychiatria_danubina/pdf/dnb_vol21_no3/dnb_vol21_no3_310.pdf

Bennetto, L., Pennington, B. F., & Rogers, S. J. (1996). Intact and impaired memory functions in autism. *Child Development, 67*(4), 1816-1835. doi:10.2307/1131734

Boucher, J. (1981). Immediate free-recall in early-childhood autism - Another point of behavioral similarity with the amnesic syndrome. *British Journal of Psychology, 72*(2), 211-215. doi:10.1111/j.2044-8295.1981.tb02177.x

Boucher, J., Mayes, A., & Bingham, S. (2012). Memory in autistic spectrum disorder. *Psychological Bulletin, 138*(3), 458-496. doi:10.1037/a0026869

Boucher, J., & Warrington, E. K. (1976). Memory deficits in early infantile autism: Some similarities to the amnesic syndrome. *British Journal of Psychology, 67*(1), 73-87. doi:10.1111/j.2044-8295.1976.tb01499.x

Bull, R., Espy, K. A., & Wiebe, S. A. (2008). Short-term memory, working memory, and executive functioning in preschoolers: Longitudinal predictors of mathematical achievement at age 7 years. *Developmental Neuropsychology, 33*(3), 205-228. doi:10.1080/87565640801982312

Calamia, M. Markon, K, & Tranel, D. (2012). Scoring higher the second time around: Meta-analyses of practice effects in neuropsychological assessment. *The Clinical Neuropsychologist, 26*(4), 543-570. doi:10.1080/13854046.2012.680913

Crowther, J. E., Hanten, G., Li, X., Dennis, M., Chapman, S. B., & Levin, H. S. (2011). Impairments in learning, memory, and metamemory following childhood head injury. *Journal of Head Trauma Rehabilitation, 26*(3), 192-201. doi:10.1097/HTR.0b013e318218dd22

Davis, O. S. P., Haworth, C. M. A., & Plomin, R. (2009). Dramatic increase in heritability of cognitive development from early to middle childhood, *Psychological Science, 20*(10), 1301-1308. doi:10.1111/j.1467-9280.2009.02433.x

Dennis, M., Francis, D. J., Cirino, P. T., Schachar, R., Barnes, M. A., & Fletcher, J. M. (2009). Why IQ is not a covariate in cognitive studies of neurodevelopmental disorders. *Journal of the International Neuropsychological Society, 15*, 331-343. doi:10.1017/S1355617709090481

Feero, W. G., Guttmacher, A. E., Mefford, H. C., Batshaw, M. L., & Hoffman, E. P. (2012). Genomic medicine: Genomics, intellectual disability, and autism. *The New England Journal of Medicine, 366*(8), 733-743. Retrieved from http://www.nejm.org/doi/full/10.1056/NEJMc1204397

Gaigg, S. B., Gardiner, J. M., & Bowler, D. M. (2008). Free recall in autism spectrum disorder: The role of relational and item-specific encoding. *Neuropsychologia, 46*(4), 983–992. doi:10.1016/j.neuropsychologia.2007.11.011

Gargaro, B., Rinehart, N., Bradshaw, J., Tonge, B., & Sheppard, D. (2011). Autism and ADHD: How far have we come in the comorbidity debate? *Neuroscience and Biobehavioral Reviews*, *35*, 1081-1088. Retrieved from http://dx.doi.org/10.1016/j.neubiorev.2010.11.002

Geschwind, D. H. (2009). Advances in autism. *Annual Review of Medicine, 60*, 367-380. doi: 10.1146/annurev.med.60.053107.121225

Giedd, J. N., & Rapoport, J. L. (2010). Structural MRI of pediatric brain development: what have we learned and where are we going? *Neuron, 67*(5), 728-734. doi:10.1016/j.neuron.2010.08.040

Goldstein, G., Allen, D. N., Minshew, N. J., Williams, D. L., Volkmar, F., Klin, A., & Schultz, R. J. (2008). The structure of intelligence in children and adults with high functioning autism. *Neuropsychology, 22*(3), 301-312. doi:10.1037/0894-4105.22.3.301

Gonzalez, L. M., Mahdavi, N., Anderson, V. A., & Harvey, A. S. (2012). Changes in memory function in children and young adults with temporal lobe epilepsy: A follow-up study. *Epilepsy and Behavior, 23*, 213-219. doi:10.1016/j.yebeh.2011.11.017

Gow, A. J., Johnson, W., Pattie, A., Brett, C. E., Roberts, B., Star, J. M., & Deary, I. J. (2011). Stability and change in intelligence from age 11 to ages 70, 79, and 87: The Lothian birth cohorts of 1921 and 1936. *Psychology and Aging, 26*(1), 232-240. doi:10.1037/a0021072

Guerts, H. M. & Vissers, M. E. (2012). Elderly with autism: Executive functions and memory. *Journal of Autism and Developmental Disorders, 42*, 665-675. doi:10.1007/s10803-011-1291-0

Henderson, H. A., Zahka, N. E., Kojkowski, N. M., Inge, A. P., Schwartz, C. B., Hileman, C. M. … Mundy, P. C. (2009). Self-referenced memory, social cognition, and symptom presentation in autism. *The Journal of Child Psychology and Psychiatry, 50*(7), 853-861. doi:10.1111/j.1469-7610.2008.02059.x

Hill, E. L., & Bird, C. A. (2006). Executive processes in Asperger syndrome: Patterns of performance in a multiple case series. *Neuropsychologia, 44*(14), 2822-2835. doi:10.1016/j.neuropsychologia.2006.06.007

Jurado, M. B., & Rosselli, M. (2007). The elusive nature of executive functions: A review of our current understanding. *Neuropsychology Review, 17*(3), 213-233. doi:10.1007/s11065-007-9040-z

Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child, 2*, 217-250. Retrieved from http://mail.neurodiversity.com/library_kanner_1943.pdf

Kibby, M. Y. & Cohen, M. J. (2008). Memory functioning in children with reading disabilities and/or attention-deficit/hyperactivity disorder: A clinical investigation of their working memory and long-term memory functioning. *Child Neuropsychology, 14*(6), 525-546. doi:10.1080/09297040701821752

Kuschner, E. S., Bodner, K. E., & Minshew, N. J. (2009). Local vs. global approaches to reproducing the Rey Osterrieth Complex Figure by children, adolescents, and adults with high-functioning autism. *Autism Research, 2*(6), 348–358. doi:10.1002/aur.101

Lajiness-O'Neill, R. R., Beaulieu, I., Titus, J. B., Asamoah, A., Bigler, E. D., Bawle, E. V., & Pollack, R. (2005). Memory and learning in children with 22q11.2 deletion syndrome: Evidence for ventral and dorsal stream disruption? *Child Neuropsychology, 11*, 55-71. doi:10.1080/09297040590911202

Larzelere, R. E., & Mulaik, S. A. (1977). Single-sample tests for many correlations. *Psychological Bulletin, 84*(3), 557-569. doi:10.1037/0033-2909.84.3.557

Lord, C., Rutter, M., DiLavore, P. C., & Risi, S. (1999). *Autism Diagnostic Observation Scale - WPS (ADOS-WPS)*. Los Angeles, CA: Western Psychological.

Mabbott, D. J., Monsalves, E., Spiegler, B. J., Bartels, U., Janzen, L., Guger, S….Bouffet, E. (2011). Longitudinal evaluation of neurocognitive function after treatment for central nervous system germ cell tumors in childhood. *Cancer, 117*(23), 5402-5411. doi:10.1002/cncr.26127

Mandelbaum, D. E., Stevens, M., Rosenberg, E., Wiznitzer, M., Steinschneider, M., Filipek, P., & Rapin, I. (2006). *Developmental Medicine and Child Neurology, 48*(1), 33-39. doi:10.1017/S0012162206000089

Maras, K. L. & Bowler, D. M. (2012). Context reinstatement effects on eyewitness memory in autism spectrum disorder. *British Journal of Psychology, 103*(3), 330-42. doi:10.1111/j.2044-8295.2011.02077.x

Maras, K. L., Gaigg, S. B., & Bowler D. M. (2012). Memory for emotionally arousing events over time in autism spectrum disorder. *Emotion*. Advance online publication. doi:10.1037/a0026679

Maziade, M., Rouleau, N., Cellard, C., Battaglia, M., Paccalet, T., Moreau, I….Merett, C. (2011). Young offspring at genetic risk of adult psychoses: The form of the

trajectory of IQ or memory may orient to the right dysfunction at the right time. *Plos One, 6*(4), e19153. doi:10.1371/journal.pone.0019153

Minshew, N., & Goldstein, G. (1993). Is autism an amnesic disorder? Evidence from the California Verbal Learning Test. *Neuropsychology, 7*(2), 209-216. doi:10.1037/0894-4105.7.2.209

Minshew, N., & Goldstein. (2001). The pattern of intact and impaired memory functions in autism. *Journal of Child Psychology and Psychiatry, 42*(8), 1095-1101. doi:10.1111/1469-7610.00808

Minshew, N., Goldstein, G., Muenz, L., & Payton, J. (1992). Neuropsychological functioning nonmentally retarded autistic individuals. *Journal of Clinical and Experimental Neuropsychology, 14*(5), 749-761. doi:10.1080/01688639208402860

Moritz, S., Ferahli, S., & Naber, D. (2004). Memory and attention performance in psychiatric patients: Lack of correspondence between clinician-rated and patient-rated functioning with neuropsychological test results. *Journal of the International Neuropsychological Society, 10*(4), 623-633. doi:10.1017/S1355617704104153

Mottron, L., Belleville, S., Stip, E., & Morasse, K. (1998). Atypical memory performance in an autistic savant. *Memory, 6*(6), 593-607. doi:10.1080/741943372

Munro, C. A., Winicki, J. M., Schretlen, D. J., Gower, E. W., Turano, K. A., Munoz, B….West, S. K. (2012). Sex differences in cognition in health elderly individuals. *Neuropsychology, Development, and Cognition. Section B, Aging,*

Neuropsychology and Cognition. Advance online publication. doi:10.1080/13825585.2012.690366

O'Brien, G., & Pearson, J. (2004). Autism and learning disability. *Autism*, *8*(2), 125-140. doi:10.1177/1362361304042718

Ozonoff, S., & Strayer, D. L. (2001). Further evidence of intact working memory in autism. *Journal of Autism and Developmental Disorders, 31*(3), 257-263. doi:10.1023/a:1010794902139

Pietrzak, R. H., Mollica, C. M., Maruff, P., Snyder, P. J. (2006). Cognitive effects of immediate release methylphenidate in children with attention-deficit/hyperactivity disorder. *Neuroscience and Biobehavioral Review, 30*, 1225-1245. doi:10.1016/j.neubiorev.2006.10.002

Polderman, T. J. C., Posthuma, D., De Sonneville, L. M. J., Stins, J. F., Verhulst, F. C., & Boomsma, D. I. (2007). Genetic analyses of the stability of executive functioning during childhood. *Biological Psychology, 76*(1-2), 11-20. doi:10.1016/j.biopsycho.2007.05.002

Poirier, M., Martin J. S., Gaigg, S. B., & Bowler, D. M. (2011). Short-term memory in autism spectrum disorder. *Journal of Abnormal Psychology*, *120*(1), 247-252. doi:10.1037/a0022298

Pring, L., & Hermelin, B. (2002). Numbers and letters: Exploring an autistic savant's unpractised ability. *Neurocase, 8*(4), 330-337. doi:10.1076/neur.8.3.330.16193

Rapeli, P., Fabritius, C., Kalska, H., & Alho, H. (2009). Memory function in opioid-dependent patients treated with methadone or buprenorphine along with benzodiazepine: Longitudinal change in comparison to healthy individuals.

Substance Abuse Treatment, Prevention, and Policy, 4(6), 254-273. doi:10.1186/1747-597X-4-6

Reynolds, C. R., & Bigler, E. D. (1994). *Test of Memory and Learning (TOMAL)*. Austin, TX: Pro-Ed.

Reynolds, C. R., & Voress, J. K. (2007). *Test of Memory and Learning: Second Edition (TOMAL-2)*. Austin, TX: Pro-Ed.

Rose, S. A., Feldman, J. F., & Jankowski, J. J. (2005). Recall memory in the first three years of life: A longitudinal study of preterm and term children. *Developmental Medicine and Child Neurology, 47*, 653-659. doi:10.1111/j.1469-8749.2005.tb01049.x

Ross, R. G., Wagner, B., Heinlein, S., & Zerbe, G. O. (2008). The stability of inhibitory and working memory deficits in children and adolescents who are children of parents with schizophrenia. *Schizophrenia Bulletin, 34*(1), 47-51. doi:10.1093/schbul/sbm104

Schmitz, C. & Rezaie, P. (2008). The neuropathology of autism: Where do we stand? (2008). *Neuropathology and Applied Neurobiology*, *34*, 4-11. doi:10.1111/j.1365-2990.2007.00872.x

Scott, F. J., & Baron-Cohen, S. (1996). Imagining real and unreal things: Evidence of a dissociation in autism. *Journal of Cognitive Neuroscience, 8*(4), 371-382. doi:10.1162/jocn.1996.8.4.371

Shaw, P., Greenstein, D., Lerch, J., Clasen, L., Lenroot, R., Gogtay, N., . . . Giedd, J. (2006). Intellectual ability and cortical development in children and adolescents. *Nature, 440*(7084), 676-679. doi:10.1038/nature04513

Silver, W.G. & Rapin, I. (2012). Neurobiological basis of autism. *Pediatric Clinics of North America, 59*(1), 45-61. doi:10.1016/j.pcl.2011.10.010

Sinzig, J., Morsch, D., Bruning, N., Schmidt, M. H., & Lehmkuhl, G. (2008). Inhibition, flexibility, working memory and planning in autism spectrum disorders with and without comorbid ADHD-symptoms. *Child and Adolescent Psychiatry and Mental Health, 2*(4), 4-16. doi:10.1186/1753-2000-2-4

Southwick, J. S., Bigler, E. D., Froehlich, A., Dubray, M. B., Alexander, A. L., Lange, N., & Lainhart, J. E. (2011). Memory functioning in children and adolescents with autism. *Neuropsychology, 25*(6), 702-710. doi:10.1037/a0024935

Spencer, M. D., Moorhead, T. W., Lymer, G. K. S., Job, D. E., Muir W. J., Hoare P., … Johnstone, E. C. (2006). Structural correlates of intellectual impairment and autistic features in adolescents. *Neuroimage, 33*, 1136-1144. doi:10.1016/j.neuroimage.2006.08.011

Spiegler, B. J., Boufett, E., Greenberg, M. L., Rutka, J. T., & Mabbott, D. J. (2004). Change in neurocognitive functioning after treatment with cranial radiation in childhood. *Journal of Clinical Oncology, 22*(4), 706-713. doi:10.1200/JCO.2004.05.186

Thioux, M., Stark, D. E., Klaiman, C., & Schultz, R. T. (2006). The day of the week when you were born in 700 ms: Calendar computation in an autistic savant. *Journal of Experimental Psychology: Human Perception and Performance, 32*(5), 1155-1168. doi:10.1037/0096-1523.32.5.1155

Thompson, P. M., Giedd, J. N., Woods, R. P., MacDonald, D., Evans, A. C., & Toga, A. W. (2000). Growth patterns in the developing brain detected by using continuum mechanical tensor maps. *Nature, 404*(6774), 190-193. doi:10.1038/35004593

Townes, B. D., Rosenbaum, G., Martin, M. D., Martins, I. P., Luis, H., & Bernardo, M. (2008). A longitudinal factor analytic study of children's neurocognitive abilities. *The International Journal Of Neuroscience, 118*(7), 1009-1023. doi:10.1080/00207450701768895

van Soelen, I. L. C., Brouwer, R. M., van Leeuwen, M., Kahn, R. S., Hulshoff Pol, H. E., & Boomsma, D. I. (2011). Heritability of verbal and performance intelligence in a pediatric longitudinal sample. *Twin Research and Human Genetics, 14*(2), 119-128. doi:10.1375/twin.14.2.119

Vogler, G. P. (2006). Behavior Genetics and Aging. In J. E. Birren & K. W. Schaire (Eds.), *Handbook of the psychology of aging (6th ed.).* (pp. 41-55). Amsterdam Netherlands: Elsevier.

Wilkinson, D. A., Best, C. A., Minshew, N. J., & Strauss, M. S. (2010). Memory awareness in indviduals with autism. *Journal of Autism and Developmental Disorders, 49*(11), 1371-1377. doi:10.1007/s10803-010-0995-x

Williams, D. L., Goldstein, G., & Minshew, N.J. (2005). Verbal and spatial working memory in autism. *Journal of Autism and Developmental Disorders, 35*(6), 747-756. doi:10.1007/s10803-005-0021-x

Williams, D. L., Goldstein, G., & Minshew, N. J. (2006a). Neuropsychologic functioning in children with autism: Further evidence for disordered complex information-

processing. *Child Neuropsychology, 12*(4-5), 279-298. doi:10.1080/09297040600681190

Williams, D. L., Goldstein, G., & Minshew, N. J. (2006b). The profile of memory function in children with autism. *Neuropsychology, 20*(1), 21-29. doi:10.1037/0894-4105.20.1.21

Wong, C. G., & Stevens, M. C. (2011). The effects of stimulant medication on working memory functional connectivity in Attention-Deficit/Hyperactivity Disorder. *Biological Psychiatry, 71*, 458-466. doi:10.1016/j.biopsych.2011.11.011

World Health Organization. (1993). *The ICD–10 classification of mental and behavioral disorders: Diagnostic criteria for research*. Geneva: World Health Organization.

Xie, S. X., Libon, D. J., Wang, X., Massimo, L., Moore, P., Vesely, L.…Grossman, M. (2010). Longitudinal patterns of semantic and episodic memory in frontotemporal lobar degeneration and Alzheimer's disease. *Journal of International Neuropsychological Society, 16*(2), 278-286. doi:10.1017/S1355617709991317

Yang, P., Jong, Y. J., Hsu, H. Y., & Lung, F. W. (2011). Role of assessment tests in the stability of intelligence scoring of pre-school children with uneven/delayed cognitive profile. *Journal of Intellectual Disability Research, 55*(5), 453-461. doi:10.1111/j.1365-2788.2011.01396.x

Yang, P., Lung, F. W., Jong, Y. J., Hsu, H. Y., & Chen, C. C. (2010). Stability and change of cognitive attributes in children with uneven/delayed cognitive development from preschool through childhood. *Research in Developmental Disabilities, 31*(4), 895-902. Retrieved from http://dx.doi.org/10.1016/j.ridd.2010.02.011

Appendix A

Frequency Distributions

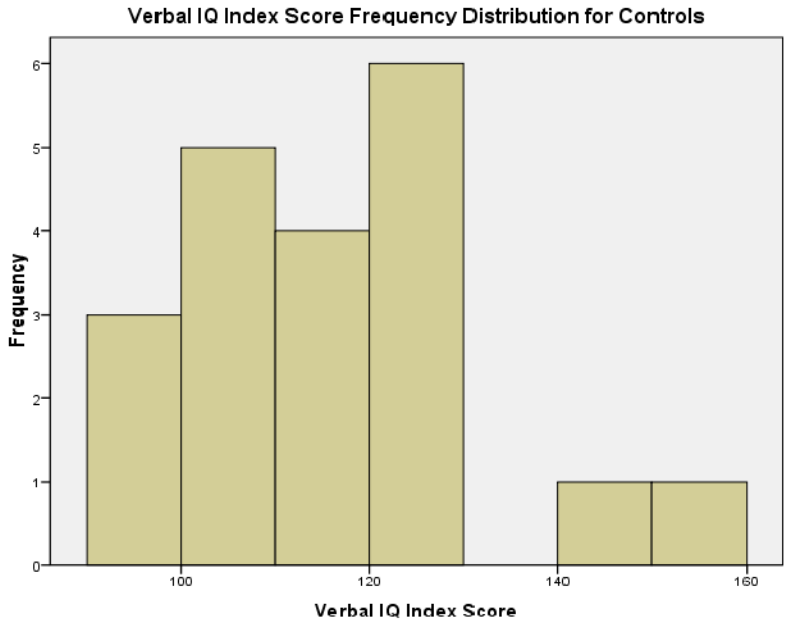

Figure 4. Verbal IQ index score frequency distribution for controls.

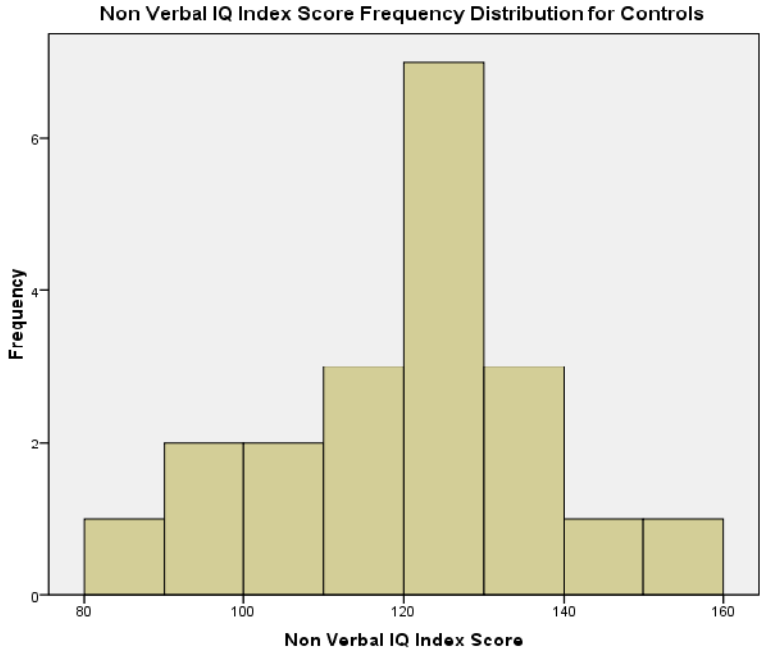

Figure 5. Non-verbal IQ index score frequency distribution for controls.

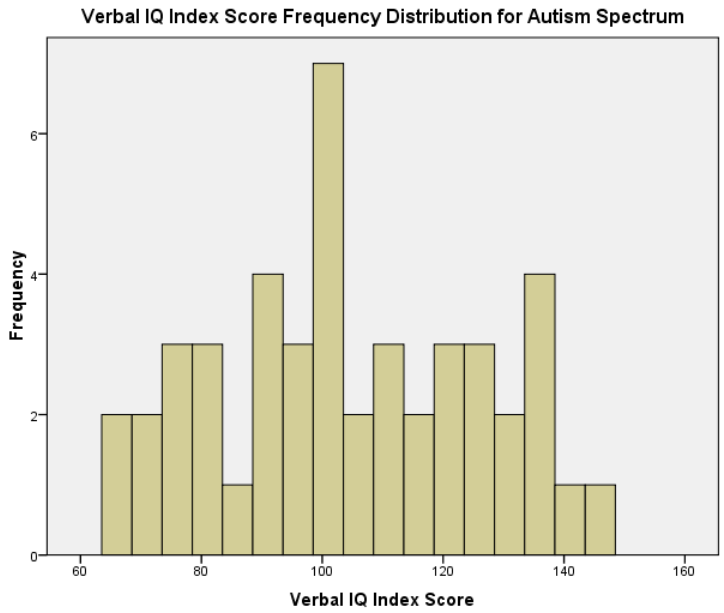

Figure 6. Verbal IQ index score frequency distribution for autism spectrum.

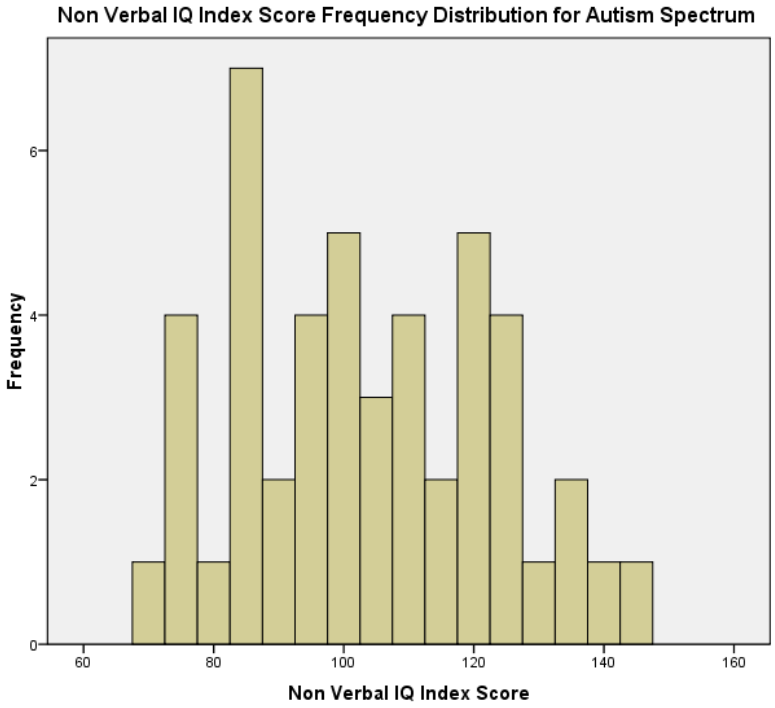

Figure 7. Non-verbal IQ index score frequency distribution for autism spectrum.

TOMAL Subtests

Verbal memory subtest performance by TDC subjects was again stable across time; Memory for Stories, $F(1, 19) = .22$, $p = .643$, Word Selective Reminding, $F(1, 19) = 1.46$, $p = .242$, Object Recall, $F(1, 19) = 2.97$, $p = .101$, Paired Recall, $F(1, 18) = .134$, $p = .719$, and Digits Forward, $F(1, 18) = 1.62$, $p = .145$. The ASD participants demonstrated significant improvement on several subtests. These include Memory for Stories, $F(1, 50) = 11.15$, $p = .002$, Object Recall, $F(1, 47) = 5.53$, $p = .023$, and Digits Forward, $F(1, 47) = 4.62$, $p = .037$. Word Selective Reminding, $F(1, 19) = 1.46$, $p = .242$ and Paired Recall, $F(1, 46) = 2.060$, $p = .158$ were stable over time. Supplemental verbal memory subtests were not analyzed due to small sample sizes.

For TDC participants, performance on most non-verbal subtests did not vary across time. Non-significant differences were noted for Facial Memory, $F(1, 19) = .005$, $p = .944$, Visual Selective Reminding, $F(1, 19) = .039$, $p = .846$, Abstract Visual Memory, $F(1, 18) = 1.235$, $p = .281$, and Visual Sequential Memory, $F(1, 18) = 1.745$, $p = .203$. Controls improved on Memory for Location, $F(1, 18) = 15.59$, $p = .001$.

ASD non-verbal memory subtests also tended to remain stable over time. Non-significant differences were observed on Facial Memory, $F(1, 49) = .978$, $p = .328$, Visual Selective Reminding, $F(1, 49) = .977$, $p = .323$, Abstract Visual Memory, $F(1, 46) = .165$, $p = .687$, and Visual Sequential Memory, $F(1, 43) = .293$, $p = .591$. Time 2 improvements were noted for Memory for Location, $F(1, 43) = 13.010$, $p = .001$.

Non-significant differences were observed for TDC participants between Time 1 and Time 2 administrations of the following delayed memory subtests: Memory for Stories Delayed, $F(1, 19) = 0.025$, $p = .875$; Word Selective Reminding Delayed, $F(1,$

19) = 1.413, p = .249; Visual Selective Reminding Delayed, F(1, 19) = 0.141, p = .711. Significant differences were observed for Facial Memory Delayed, F(1, 19) = 7.257, p = .014. In contrast, ASD participants improved on Memory for Stories Delayed, F(1, 48) = 5.839, p = .020, Facial Memory Delayed, F(1, 47) = 18.148, p = .000, and Word Selective Reminding Delayed, F(1, 47) = 9.765, p = .003. They did not improve on Visual Selective Reminding Delayed, F(1, 48) = 0.458, p = .502.

Table 9

TOMAL Verbal Subtest Scaled Scores across Time

	TDC				ASD			
	TOMAL 1		TOMAL 2		TOMAL 1		TOMAL 2	
Subtest	*Mean*	*SD*	*Mean*	*SD*	*Mean*	*SD*	*Mean*	*SD*
MFS	11.55	3.12	11.85	3.08	7.55	3.35	8.98	4.06*
WSR	11.65	2.54	12.50	2.71	8.04	4.17	9.18	3.83
OR	9.60	2.64	10.65	2.46	6.19	3.61	7.25	3.71*
DF	9.42	3.37	8.42	3.25	6.08	3.49	6.85	3.45*
PR	11.84	2.59	12.05	2.17	8.57	3.51	9.23	3.03

Note. MFS = Memory for Stories; WSR = Word Selective Reminding; OR = Object Recall; DF = Digits Forward; PR = Paired Recall.
*Significant at $p \leq .037$.

Table 10

TOMAL Non-Verbal Subtest Scaled Scores across Time

	TDC				ASD			
	TOMAL 1		TOMAL 2		TOMAL 1		TOMAL 2	
Subtest	*Mean*	*SD*	*Mean*	*SD*	*Mean*	*SD*	*Mean*	*SD*
FM	10.75	2.95	10.70	2.99	7.30	2.11	7.68	2.67
VSR	10.00	2.45	10.15	2.48	6.96	3.54	6.46	3.52
AVM	13.37	2.93	12.74	2.10	9.26	3.08	9.04	3.57
VSM	10.79	3.01	11.79	2.51	7.77	2.33	8.00	3.07
ML	11.79	3.34	14.68	2.89*	8.23	4.99	10.48	4.60*

Note. FM = Facial Memory; VSR = Visual Selective Reminding; AVM = Abstract Visual Memory; VSM = Visual Sequential Memory; ML = Memory for Location.
*Significant at $p = .001$.

Table 11

TOMAL Delayed Recall Subtest Scores across Time

	TDC				ASD			
	TOMAL 1		TOMAL 2		TOMAL 1		TOMAL 2	
Subtest	*Mean*	*SD*	*Mean*	*SD*	*Mean*	*SD*	*Mean*	*SD*
MFSD	11.50	2.93	11.40	2.95	7.18	3.50	8.51	4.54*
FMD	9.65	2.16	11.30	1.46*	7.90	2.68	9.38	1.84*
WSRD	10.60	1.54	11.20	1.15	8.85	2.32	9.92	2.40*
VSRD	10.30	1.46	10.15	1.35	8.33	2.33	8.59	2.20

Note. MFSD = Memory for Stories Delayed; FMD = Facial Memory Delayed; WSRD = Word Selective Reminding Delayed; VSRD = Visual Selective Reminding Delayed.
*Significant at $p \leq .020$.

Subtest Reliable Change Index Results

Table 12

Reliable Change Index Improvement and Deterioration Effects for TOMAL Verbal Subtests

	Group			
	TDC		ASD	
Index	RCI (*n*)	RCD (*n*)	RCI (*n*)	RCD (*n*)
MFS	15.00 (*20*)	10.00 (20)	21.57 (*51*)	5.88 (51)
WSR	5.00 (20)	0.00 (20)	17.65 (51)	13.73 (51)
OR	20.00 (20)	5.00 (20)	12.77 (47)	8.51 (43)
DF	5.26 (19)	36.84 (19)	25.00 (48)	16.67 (48)
PR	15.79 (19)	5.26 (19)	12.77 (47)	6.38 (47)

Note. Numbers indicate percentage of cases surpassing 1.96 on RC. RCI = Reliable Change Index-Improvement; RCD = Reliable Change Index-Deterioration. MFS = Memory for Stories; WSR = Word Selective Reminding; OR = Object Recall; DF = Digits Forward; PR = Paired Recall.

Table 13

Reliable Change Index Improvement and Deterioration Effects for TOMAL Non-Verbal Subtests

	Group			
	TDC		ASD	
Index	RCI (*n*)	RCD (*n*)	RCI (*n*)	RCD (*n*)
FM	20.00 (*20*)	15.00 (20)	12.00 (*50*)	4.00 (50)
VSR	20.00 (20)	15.00 (20)	18.00 (50)	24.00 (50)
AVM	10.53 (19)	15.79 (19)	23.40 (47)	25.53 (47)
VSM	26.32 (19)	15.79 (19)	20.45 (44)	15.91 (44)
ML	52.63 (19)	5.26 (44)	45.45 (47)	11.36 (44)

Note. Numbers indicate percentage of cases surpassing 1.96 on RC. RCI = Reliable Change Index-Improvement; RCD = Reliable Change Index-Deterioration. FM = Facial Memory; VSR = Visual Selective Reminding; AVM = Abstract Visual Memory; VSM = Visual Sequential Memory; ML = Memory for Location.

Table 14

Reliable Change Index Improvement and Deterioration Effects for TOMAL Delayed Subtests

	Group			
	TDC		ASD	
Index	RCI (*n*)	RCD (*n*)	RCI (*n*)	RCD (*n*)
MFSD	10.00 (*20*)	10.00 (20)	18.37 (*49*)	4.08 (49)
FMD	20.00 (*20*)	5.00 (20)	12.50 (*48*)	0.00 (48)
WSRD	5.00 (20)	0.00 (20)	12.50 (48)	0.00 (48)
VSRD	0.00 (20)	0.00 (20)	6.12 (49)	2.04 (49)

Note. Numbers indicate percentage of cases surpassing 1.96 on RC. RCI = Reliable Change Index-Improvement; RCD = Reliable Change Index-Deterioration. MFSD = Memory for Stories Delayed; FMD = Facial Memory Delayed; Word Selective Reminding Delayed; VSRD = Visual Selective Reminding Delayed.

Subtest Correlational Analyses

Table 15

Correlations between IQ and TOMAL Time 1 and 2 Verbal Memory Subtests for TDC

	VIQ	NVIQ	MFS-1	WSR-1	OR-1	DF-1	PR-1	MFS-2	WSR-2	OR-2	DF-2	PR-2
VIQ	-	.78*	.47	.14	.38	.45	.34	.47	.24	.14	.58	.29
NVIQ		-	.42	.26	.24	.41	.34	.51	.30	.05	.66	.17
MFS-1			-	.29	.43	.23	.47	.58	.30	.18	.39	.00
WSR-1				-	.27	.15	.53*	.05	.28	.12	.00	.13
OR-1					-	.17	.28	.19	.46	.43	.01	.02
DF-1						-	.24	.30	.15	.20	.81*	-.20
PR-1							-	.44	.25	.20	.44	.46
MFS-2								-	.40	.24	.40	.30
WSR-2									-	.66	.05	-.13
OR-2										-	.04	-.22
DF-2											-	.23
PR-2												-

Note. VIQ = Verbal IQ; NVIQ = Nonverbal IQ; MFS-1 = Time 1 Memory for Stories; WSR-1 = Time 1 Word Selective Reminding; OR-1 = Time 1 Object Recall; DF-1 = Time 1 Digits Forward; PR-1 = Time 1 Paired Recall; MFS-2 = Time 2 Memory for Stories; WSR-2 = Time 2 Word Selective Reminding; OR-2 = Time 2 Object Recall; DF-2 = Time 2 Digits Forward; PR-2 = Time 2 Paired Recall.
*Significant at $p \leq .001$, with modified Bonferroni correction.

Table 16

Correlations between IQ and TOMAL Time 1 and 2 Verbal Memory Subtests for ASD

	VIQ	NVIQ	MFS-1	WSR-1	OR-1	DF-1	PR-1	MFS-2	WSR-2	OR-2	DF-2	PR-2
VIQ	-	.57*	.56	.46	.47*	.26	.32	.67*	.33	.17	.33	.18
NVIQ		-	.36*	.28	.32	.31	.35*	.43*	.14	.06	.32	.22
MFS-1			-	.49*	.57*	.20	.43*	.67*	.43	.37	.40	.33
WSR-1				-	.60*	.32	.34*	.50*	.42	.50*	.40	.16
OR-1					-	.16	.44*	.53*	.53*	.63*	.37	.36
DF-1						-	.24	.27	.24	.20	.74*	.10
PR-1							-	.48*	.57*	.42	.27	.55*
MFS-2								-	.58*	.47*	.50*	.52*
WSR-2									-	.74*	.25	.50*
OR-2										-	.37	.46*
DF-2											-	.23
PR-2												-

Note. VIQ = Verbal IQ; NVIQ = Nonverbal IQ; MFS-1 = Time 1 Memory for Stories; WSR-1 = Time 1 Word Selective Reminding; OR-1 = Time 1 Object Recall; DF-1 = Time 1 Digits Forward; PR-1 = Time 1 Paired Recall; MFS-2 = Time 2 Memory for Stories; WSR-2 = Time 2 Word Selective Reminding; OR-2 = Time 2 Object Recall; DF-2 = Time 2 Digits Forward; PR-2 = Time 2 Paired Recall.
*Significant at $p \leq .001$, with modified Bonferroni correction.

Table 17

Correlations between IQ and TOMAL Time 1 and 2 Non Verbal Memory Subtests for TDC

	VIQ	NVIQ	FM-1	VSR-1	AVM-1	VSM-1	ML-1	FM-2	VSR-2	AVM-2	VSM-2	ML-2
VIQ	-	.78*	.12	.14	.33	.28	.44	.21	.44	.34	.36	.53
NVIQ		-	.12	.16	.42	.36	.39	.40	.28	.55	.50	.66
FM-1			-	.17	.15	.05	.26	.44	.21	.38	.33	.37
VSR-1				-	.02	.05	.24	.00	.04	-.20	.30	-.33
AVM-1					-	.31	.11	.02	.20	.56	.50	.42
VSM-1						-	.20	.28	.22	.18	.30	.64
ML-1							-	.00	.25	-.11	.09	.48
FM-2								-	.20	.40	.42	.11
VSR-2									-	.10	.07	.25
AVM-2										-	.38	.42
VSM-2											-	.39
ML-2												-

Note. VIQ = Verbal IQ; NVIQ = Nonverbal IQ; FM-1 = Time 1 Facial Memory; VSR-1 = Time 1 Visual Selective Reminding; AVM-1 = Time 1 Abstract Visual Memory; VSM-1 = Time 1 Visual Sequential Memory; ML-1 = Time 1 Memory for Location; FM-2 = Time 2 Facial Memory; VSR-2 = Time 2 Visual Selective Reminding; AVM-2 = Time 2 Abstract Visual Memory; VSM-2 = Time 2 Visual Sequential Memory; ML-2 = Time 2 Memory for Location.

*Significant at $p \leq .001$, with modified Bonferroni correction.

Table 18

Correlations between IQ and TOMAL Time 1 and 2 Non Verbal Memory Subtests for ASD

	VIQ	NVIQ	FM-1	VSR-1	AVM-1	VSM-1	ML-1	FM-2	VSR-2	AVM-2	VSM-2	ML-2
VIQ	-	.57*	.24	.23	.31	.27	.18	.37	.20	.44	.30	.29
NVIQ		-	.32	.30	.60*	.40*	.51*	.22	.35	.43	.27	.45
FM-1			-	.25	.38*	.35*	.49*	.37	.04	.46*	.35	.51*
VSR-1				-	.37*	.36*	.52*	.59*	.50*	.62*	.45	.50*
AVM-1					-	.52*	.57*	.31	.46*	.42	.42	.54*
VSM-1						-	.47*	.30	.32	.33	.50*	.50*
ML-1							-	.45*	.33	.47*	.45	.63*
FM-2								-	.37	.69*	.51*	.51*
VSR-2									-	.47*	.45	.43
AVM-2										-	.66*	.54*
VSM-2											-	.56*
ML-2												-

Note. VIQ = Verbal IQ; NVIQ = Nonverbal IQ; FM-1 = Time 1 Facial Memory; VSR-1 = Time 1 Visual Selective Reminding; AVM-1 = Time 1 Abstract Visual Memory; VSM-1 = Time 1 Visual Sequential Memory; ML-1 = Time 1 Memory for Location; FM-2 = Time 2 Facial Memory; VSR-2 = Time 2 Visual Selective Reminding; AVM-2 = Time 2 Abstract Visual Memory; VSM-2 = Time 2 Visual Sequential Memory; ML-2 = Time 2 Memory for Location.

*Significant at $p \leq .001$, with modified Bonferroni correction.

Table 19

Correlations between IQ and TOMAL Time 1 and 2 Delayed Recall Subtests for TDC

	VIQ	NVIQ	MFSD-1	FMD-1	WSRD-1	VSRD-1	MFSD-2	FMD-2	WSRD-2	VSRD-2
VIQ	-	.78*	.36	.27	.10	.10	.36	.04	-.04	-.01
NVIQ		-	.30	.18	.09	.01	.43	.03	.09	-.08
MFSD-1			-	.12	.28	.14	.54	.21	.05	.07
FMD-1				-	-.18	.02	.13	-.12	.16	.19
WSRD-1					-	.12	.50	.53	-.40	.01
VSRD-1						-	.30	.30	-.16	.19
MFSD-2							-	.41	-.27	.36
FMD-2								-	-.10	.38
WSRD-2									-	-.19
VSRD-2										-

Note. VIQ = Verbal IQ; NVIQ = Nonverbal IQ; MFSD-1 = Time 1 Memory for Stories Delayed; FMD-1 = Time 1 Facial Memory Delayed; WSRD-1 = Time 1 Word Selective Reminding Delayed; VSRD-1 = Visual Selective Reminding Delayed; MFSD-2 = Time 2 Memory for Stories Delayed; FMD-2 = Time 2 Facial Memory Delayed; WSRD-2 = Time 2 Word Selective Reminding Delayed; VSRD-2 = Visual Selective Reminding Delayed.

*Significant at $p \leq .001$, with modified Bonferroni correction.

Table 20

Correlations between IQ and TOMAL Time 1 and 2 Delayed Recall Subtests for ASD

	VIQ	NVIQ	MFSD-1	FMD-1	WSRD-1	VSRD-1	MFSD-2	FMD-2	WSRD-2	VSRD-2
VIQ	-	.57*	.45*	.07	.29	.25	.59*	.35	.34	.11
NVIQ		-	.30	.14	.38*	.26	.40	.25	.10	.11
MFSD-1			-	.25	.50*	.35*	.57*	.28	.41	.17
FMD-1				-	.28	.07	.08	.49*	-.14	.21
WSRD-1					-	.33	.45*	.48*	.50*	.24
VSRD-1						-	.44	.36	.25	.27
MFSD-2							-	.29	.50*	.39
FMD-2								-	.50*	.39
WSRD-2									-	.52*
VSRD-2										-

Note. VIQ = Verbal IQ; NVIQ = Nonverbal IQ; MFSD-1 = Time 1 Memory for Stories Delayed; FMD-1 = Time 1 Facial Memory Delayed; WSRD-1 = Time 1 Word Selective Reminding Delayed; VSRD-1 = Visual Selective Reminding Delayed; MFSD-2 = Time 2 Memory for Stories Delayed; FMD-2 = Time 2 Facial Memory Delayed; WSRD-2 = Time 2 Word Selective Reminding Delayed; VSRD-2 = Visual Selective Reminding Delayed.

*Significant at $p \leq .001$, with modified Bonferroni correction.