Working Memory Dysfunction in Children and Young Adults with Autism

With the growing numbers of children diagnosed in the autistic spectrum, there is an increased need to understand and define working memory deficits in the population. A better understanding can inform development of educational tools and therapies designed to ameliorate the most common behavioral impairments. Recent autism research in working memory has been largely driven by two hypotheses: (1) working memory deficits are a result of primary deficits in executive function (EF theory); and (2) working memory deficits are related to how complex information is to process, and that executive function disorders are secondary to larger cognitive deficits (I am calling this “complexity theory” for ease of reference in the remainder of the paper). While there is strong neurological and behavioral evidence supporting the involvement of impaired executive functions in autism, the research is inconclusive as to degree and primacy of involvement. This paper will first present three studies from the perspective of EF theory that suggests not only is executive function impaired in autism, but also may in fact be causal to autism, and then examines one study that found contradictory evidence to this conclusion. Additionally, this paper will examine two studies I categorized as complexity theory that focus on memory deficits found in autism and their relationship to the complexity of the to-be-remembered material.

Autism

Autism, a spectrum disorder, is diagnosed by observation of behavioral and developmental impairments in three general areas: (1) communication, (2) socialization and (3) imagination, exhibited by restricted, repetitive and stereotyped patterns of behavior and interests (Sicile-Kira, 2004). Autism is one of five disorders categorized as
a Pervasive Development Disorder (PDD), the other four being Asperger’s Syndrome, Childhood Disintegration Disorder, Rhett’s Syndrome, and PDD – Not Otherwise Specified (PDD-NOS). All five of these diagnoses are neurological maladies collectively referred to as Autistic Spectrum Disorders. “Autism” will be used throughout this paper to refer to the entire spectrum. Autism is not curable and affects approximately 1 in every 166 children. Additionally, the rate of diagnosis is increasing at an incredible rate of 10-17% each year (Sicile-Kira, 2004).

The autistic spectrum ranges from individuals with low IQs and severe learning disabilities to individuals with high functioning autism (HFA) and Asperger’s Syndrome at the other end. Individuals with HFA and Asperger’s Syndrome have normal or above average IQs and are usually integrated into the larger “normal” society. Autism research has struggled with extreme variability in the population, which often produces inconsistent findings. While the autistic population is a challenging group to study, it is a very important endeavor; defining and describing working memory deficits can help inform the development of tools and therapies designed to ameliorate the most common behavioral autistic impairments.

Working memory in autism

The role of working memory function in autism has been a focus of research for over three decades. In the 1970s and 1980s the “amnesia theory” dominated research which was based on the assumption that the role of memory was key to the underlying cause of autistic behaviors. Abnormalities in the hippocampus helped this theory gain wide acceptance, however subsequent research debunked amnesia theory.

Recent research in working memory in the autistic population has largely used Baddeley’s model, which is composed of four primary components (Baddeley, 2003). The controlling component, the “central executive function”, is responsible for goal-directed behaviors that require planning, flexible strategy employment, voluntary
response inhibition, organized search and directing attention. Under the central executive are three other components, referred to as slave systems. The slave systems include the visuo-spatial sketchpad (visual/spatial working memory), the episodic buffer, and the phonological loop (verbal working memory). A key assumption in Baddeley’s model is that all of the components of working memory draw upon the same resources. In other words, all components use the same gas tank – a kind of “activation fuel”. While both EF and complexity theory use Baddeley’s model to make predictions, they focus on different replicated studies to support their theories.

**Hypothesis backgrounds**

EF theory cites converging evidence from at least five different studies conducted before 1999 using the “Tower of Hanoi” paradigm that has shown impairment in planning skills among autistic individuals. The Tower of Hanoi is a common experimental paradigm used to measure the planning aspect of the central executive. Additionally, autistic subjects have shown impairment in other standard clinical tasks used to measure executive function impairment, including the Wisconsin Card Sorting Test (WCST) which is designed to measure the effect of previously relevant information and its effect on behavior.

Throughout the WCST test the rules for a “right answer” change, requiring the subject to use flexible strategies. Much like patients with frontal lobe damage, autistic individuals can find the first rule with relative ease; however, they are subsequently unable to shift their strategies to find new rules. Consequently they make many more perseveration errors than made by matched controls. While those subscribing to the complexity theory do not debate that these replicable studies point to central executive deficits, they do not believe that central executive dysfunction is the primary explanation for working memory impairments in the autistic population.
Researchers subscribing to complexity theory cite supporting evidence from studies that found that although children with autism recall random words as well as control children, their recall did not improve when compared to control children when the words were put into sentences and semantic meaning was added (Hermalin & O’Conner, 1970). These results have been replicated in similar studies using binary patterns signifying that the deficit is pervasive and not simply limited to semantic-based organization strategies. Other studies have found that although autistic children can benefit from clues that are given to them, they do not create their own semantic, syntactic or temporal sequences to facilitate retrieval (Tager-Flusberg, 1991).

The complexity of memory items was also reported as a factor in a study that found that young children diagnosed with autism had the fewest impairments compared to controls with recall of digits, more with sentences, and the most with stories (Fien et al., 1996). Authors of this study implied that memory function in autism can be distinguished by a dissociation between impaired memory for complex tests and intact memory for simple tests. Connecting this back to Baddeley’s model, this evidence suggests that while autistic individuals may have intact storage capacity, they do not have the same level of activation fuel – so that when a task increases in difficulty it impacts the ability to recall or encode a memory. Both theories provide alternative explanations as to the role of working memory in the autistic behavioral triad.

EF theory researchers argue that deficits in the central executive explain the triad of behaviors. Repetitive behaviors and restricted interests are a result of a lack of flexibility and decreased inhibition. Deficits in social and communication skills are rooted in the inability to learn by experience and difficulty in creating new strategies to navigate the complexity required in social situations.

Complexity theory predicts that memory dysfunction is correlated to complexity of processing, and that all components of working memory are equally impacted. Since
social interaction and communication are complex processes, this theory posits that complexity is the reason autistic people have such a difficult time socially. Additionally, many autistic individuals also have problems understanding facial expressions, another complex processing task.

**EF theory supporting evidence**

Researchers subscribing to the EF theory argue that (1) since the central executive resides in the frontal lobes there will be parallelism to the impairments found in patients with frontal lobe damage, and (2) autistic individuals will not perform as well as matched controls in tasks requiring the central executive. Additionally, the primacy of executive functions in autism, (i.e. the underlying cause of autism), is debated among researchers subscribing to the EF theory.

Strong neurological evidence supporting frontal lobe involvement in autism (the domain of the central executive) in autism has been found using functional magnetic resonance imaging (fMRI) scans during spatial memory tasks (Luna et. al., 2002). fMRI scans reveal activity in the brain by imaging blood flow. In this study the authors predicted that autistic individuals would display less brain activity in the frontal lobe area during spatial memory (working memory) tasks. The study tested two adult groups of subjects, autistic and control, that were matched for IQ.

Results indicated significant decreases in frontal lobe activity for the autistic group while performing spatial memory tasks. The researchers concluded that spatial working memory in autism may be a result of abnormalities in prefrontal circuitry. Furthermore, they suggested that working memory impairments could therefore be a product of central executive dysfunction. Strong behavioral evidence from two different studies also points to significant central executive involvement in autism.

One study providing behavioral evidence of central executive involvement predicted that if individuals with autism suffer primarily from EF disorders, then
impairments should include deficits in temporal order memory and working memory capacity. Concurrently, intact memory should include short and long-term recognition, cued recall and new learning ability (Bennetto, Pennington and Rogers, 1996).

Two groups of subjects were used, one autistic and one normal, matched for verbal IQ and age (approximately 15 years old). The experiment included many classic central executive tasks including the Wisconsin Card Sorting Test and the Tower of Hanoi. Temporal order and recognition memory tasks were also employed. Working memory capacity was measured with digit span (verbal working memory) and reading span tests (overall working memory capacity).

Across a variety of memory tests autistic individuals demonstrated consistent memory patterns similar to patients with frontal lobe lesions. The autistic group’s performance was significantly inferior during central executive tasks compared to controls. Performance of the groups was also found to be significantly different in the temporal order task, but not in the recognition memory tests. Groups did not differ in their digit spans; however, the reading spans of the autistic group were significantly reduced from the controls. The researchers concluded there were strong similarities between autistic individuals and patients with frontal lobe damage. Additionally they asserted that executive dysfunction might actually be the cause of autism.

Additional behavioral evidence supporting EF theory was found in a more recent study using tests that the authors argued measured frontal lobe activity more precisely than the Tower of Hanoi or the Wisconsin Card Sorting Test (Oznoff et. al., 2004). The study used two subtasks of the Cambridge Neuropsychological Test Automated Battery (CANTAB), which has been related to frontal cortex activity through previous studies. One subtask, the “Stockings of Cambridge” (SOC), evaluates planning abilities (similar to the Tower of Hanoi). The other subtask, “Intradimensional/Extradimensional Shift,” evaluates flexibility in strategy employment (similar to the Wisconsin Card Sorting Task).
The researchers predicted that autistic performance would be inferior in these tasks. Additionally they questioned whether or not age was an important variable in autism.

Some earlier studies examining executive deficits in young children did not find significant differences in performance of executive tasks (one example is discussed below). Therefore, researchers predicted that if EF function was secondary (i.e., acquired later in life), then it would get worse with age. Conversely, if it were a primary cause of autism, it would not get worse with age. Two groups of subjects, one autistic and one normal, were matched for age (average age approximately 16 years), IQ, and sex. To examine the potential interaction of executive function deficits and age, the subjects were further divided into three subgroups matched for age.

Significant deficits were found in both planning and set shifting abilities required to in the WCST task across all IQ and age groups, strongly supporting frontal lobe involvement and central executive dysfunction in autism. Subgroup analysis, splitting the groups by age, did not support the suggestion that executive deficits worsen with age, supporting the assertion that executive dysfunction is primary to the cause of autism. However, other studies have found contrary evidence casting doubt on the primacy of EF dysfunction.

*EF theory refuting evidence*

One study finding evidence contrary to the primacy of EF dysfunction used younger children (Griffith et. al, 1999). Researchers conducted two studies: (1) one compared an autistic group of children to a matched control group, average age approximately four years, on eight executive function tasks; and (2) another followed a subset of the original groups for a year to determine if there were any changes to abilities over time. The hypothesis of the first study stated that children with autism would perform significantly worse than controls in executive function tasks. The second
study examined the primacy of EF function asking if children with autism would perform worse on EF tasks as they grow older.

Results of the first study did not support the hypothesis that children with autism perform significantly worse than controls in executive function tasks. Although both groups performed below mental age expectations, autistic children actually performed better than controls on some tasks. The researchers concluded that this ruled out early executive functions deficits in autism, indicating that executive function is not a primary cause of the disorder.

In the second study, the researchers retested subjects one year later on some tasks. Results did not support the hypothesis that children with autism would perform worse over time; however, the control group performed better. The researchers suggest that children with autism may not be growing into a progressing executive disorder, but instead, children without autism may grow out of an executive deficit disorder.

There is no real consensus as to the primacy of EF function in autism. Clearly, there appears to be some frontal lobe involvement and some executive functions are impaired with older autistic children and adults. However, age appears to be an important variable, where younger children do not appear to show the same level of impairments as do older children. Understanding the involvement of executive function impairment in autism is further complicated when factoring in normal frontal lobe development. Normal development continues until the early 20s, so it is possible deficits could not even be seen until children were older. The question remains, is EF function a primary cause of autism or is it part of larger cognitive impairments? Proponents of the level of complexity theory argue the latter.

*Complexity theory supporting evidence*

Complexity theory argues that deficits in working memory are not a product of central executive dysfunction, but are instead part of a larger set of cognitive
impairments. At the core of complexity theory, researchers argue that (1) autistic individuals do not use organizational strategies or context to support memory, and (2) working memory abilities decrease when compared to controls as the to-be-remembered items increase in complexity.

Research supporting complexity theory has come from a study examining the pattern of verbal memory function in high-functioning adolescents and young adults with autism (Minshew and Goldstien, 2001). The study hypothesis was that working memory dysfunction is present in autism; however it is not specific to the central executive. It is instead a result of poor organizing strategies. Furthermore, they posited that working memory dysfunction in autism is one of multiple co-existing deficits related to information complexity. In other words, cognitive impairment is not a pattern explained by any one underlying deficit but becomes evident as complexity increases.

Two groups of subjects, one autistic and one normal were matched for IQ and age (approximately 22 years old). Subjects performed a variety of experiments in an effort to support three interrelated hypotheses.

The first hypothesis predicted that people with autism will not improve in a recall test if words on a memory list are semantically related versus a non-semantically related list. Results supported this hypothesis, and the researchers concluded that autistic individuals have intact recall memory but the semantic meaning is used less efficiently to aid learning semantically similar words.

The second and third experimental paradigms tested the hypotheses that autistic memory performance would become increasingly impaired as task complexity increased when compared to controls. They compared performance on simple tests, (including immediate recall) to more complex tests, (including delayed recall). The autistic group did poorly on delayed versus immediate recall tests. Authors argued that this is an indication that autistic individuals do not use organizational structures, resulting in a
more shallow level of encoding. This experiment provided strong support for the level of complexity theory but did not attempt to profile the areas of working memory where deficits were most pronounced.

Additional support for complexity theory came from a study intended to determine the profile of working memory deficits for children with autism and whether those deficits followed a pattern similar to adults with autism (Williams et. al., 2006). Two groups of subjects, one autistic, and one not, average age approximately 12 years, matched for IQ, performed a variety of tests from the Wide Range Assessment of Memory and Learning (WRAML), normed for children. The standard version of the WRAML is a test commonly used to assess the profile of working memory in autistic adults. The tests included nine subtests divided into visual, auditory and learning domains. Researchers examined (1) whether or not complexity or the modality (verbal or visual) would have any effect on the results, and (2) whether or not the autistic group would use strategies to facilitate memory. Additionally, the researchers employed tasks that tested each component of working memory from Baddeley’s model separately in an attempt to profile overall working memory in autism.

Results indicated that autistic children have a much different working memory profile than a normally developing control group. The autistic group displayed inferior memory for complex visual verbal tests compared to controls. There was not a significant difference in group performance in simpler tasks involved in associative learning ability, verbal working memory and recognition memory.

Spatial working memory was also impaired for the autistic group when defined by the ability to remember a location (considered complex); however visual spatial learning was not impaired (considered a more simple task). Delayed recall also had mixed results where there was no significant difference in the two groups except for complex recall involving stories.
Additionally, the working memory profile for autistic children was very similar to that found in autistic adults. The only difference between groups was in the sentence and story memory tasks. The researchers attributed this to a lack of understanding of basic story structure that adults either learned or developed through exposure to reading.

The authors concluded that their results supported that level of complexity is the critical variable to working memory dysfunction in autism. They argue that confirmation of this theory requires both the display of intact abilities for simple tasks, such as simple word recall, and impairment in complex tasks such as story recall. They further suggest that impairments are due to an inability to create and use strategies to facilitate memory.

There is much more consensus in research using the level of complexity hypothesis than in research from the EF perspective; however, there is not as much research using the former hypothesis. While complexity theory provides a convincing explanation of the social and communication deficits found in autism, it does not provide a satisfying explanation for the third aspect of the behavioral triad, repetitive and restricted behaviors. Additionally, while it does not explicitly rule out central executive involvement in working memory deficits, proponents do not believe that impaired central executive functions are causal to autism.

Conclusion

There is no consensus for a profile of working memory in autistic individuals young or old. There is strong evidence that some central executive functions are impaired in autism. However, the question remains: are working memory deficits part of a primary executive dysfunction, or are working memory deficits related to how complex the information is to process, and central executive function disorders are secondary to larger cognitive deficits? Using Baddeley’s model, the question is a debate between two ideas (1) impairment to the working memory master component (central executive) is
primary, and subsequent effects on slave systems result because the master is unable
to correctly direct attention and (2) an overall lack of activation fuel is affecting all the
components of working memory equally, but the fuel shortage is more dramatic when
more fuel is needed for complex material.

   It is important to assess the role of working memory in the triad of common
autistic behaviors in order to develop therapies that may ameliorate these behaviors and
help autistic people integrate into society more easily. The complexity theory provides a
better explanation for the communication and social deficits since social interactions
require shifting strategies and distinguishing different contexts.

   EF theory does a more satisfying job of explaining repetitive behaviors and
restricted interests because the central executive is involved in flexible strategy
employment and voluntary response inhibition. Additionally, executive functions begin to
develop in the first year of life and autism is usually diagnosed when EF impairments
would begin to show up, around the age of three. Furthermore, neurological evidence
provides strong support for the involvement of the frontal lobes in autism. Since the
central executive is believed to reside in the frontal lobes, this is strong support of
involvement of EF function in autism.

   However, deficits in executive function are not specific to autism and are found in
a range of disorders including Attention Deficit Hyperactivity Disorder (ADHD) and
Tourette’s syndrome. Those that argue against the primacy of EF function argue this as
evidence that impairment in EF cannot be sufficient to actually cause the spectrum of
symptoms associated with autism.

   The conflicting evidence of working memory impairments in autism complicates
the argument, since some studies have found no working memory deficits in very young
children or in those with high-functioning autism. Research in this area is further
complicated by the range of capabilities displayed in the population. As the autistic
population grows there is a pressing need to understand both the profile of memory and the causes of memory deficits in order to develop tools and therapies that might provide early intervention in order to ameliorate common autistic behaviors.

Areas for future research in this area should include longitudinal studies of autistic individuals beginning at an early age that profile (1) impairment of executive dysfunction over many ages using the same individuals and (2) whether or not the complexity of the to-be-remembered item is a factor at a younger age. Subjects should be from a wide range of IQ’s in an attempt to profile the progression of memory dysfunction over time. Understanding the range of profiles over time could help inform therapists now on the expected working memory capabilities of their patients. It would also more accurately inform those creating educational tools as to who their audience is and what could be expected from them.

Additionally, future research should aim to determine the differences in executive dysfunction in autism when compared to ADHD and Tourette’s syndrome. While there has been strong evidence that executive dysfunction in autism has a different profile than in the ADHD population (Guerts et. al, 2004), similar studies comparing the Tourette’s and autistic population were not conclusive (Ozonoff & Strayer, 2001). If similarities exist, then therapies aimed at ameliorating executive dysfunction might be shared. If the profiles are extremely different, then it may help in early diagnosis and proper early intervention for respective populations.
References


