Autism As An Information Processing Disorder:  
A Neurocognitive Model

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Autism is a behaviorally defined neurodevelopmental disorder that impairs social, language, and adaptive functioning. Examination of the cognitive origins of the behaviors characteristic of autism has led to a greater understanding of this complex disorder and has resulted in several models of autism. The recognition of deficits in theory of mind in individuals with autism yielded better understanding of the communicative and social impairments (Baron-Cohen, Leslie & Frith, 1985). The weak central coherence model offers a cogent explanation for the failure of individuals with autism to see the gestalt or to ascribe meaning to acquired information (Firth, 1989). The executive dysfunction model (Pennington et al., 1997), while not explaining the developmental course and not precisely fitting the profile of deficits (Griffith, Pennington, Wehner, & Rogers, 1999; Ozonoff & Strayer, 2001), has yielded new understanding about the level of impairment beyond a description of positive and negative symptoms. However, none of these models has accounted for the myriad of cognitive and behavioral manifestations of autism.

Minshew and Goldstein (1998) have proposed a model of autism that is based upon models of information processing and brain at the neural systems levels. A comprehensive examination of the performance of individuals with high functioning autism across domains revealed a pattern of deficits in which tasks that required simple information processing were performed at or above the level of normal controls while tasks that required processing of complex information proved to be more difficult (Minshew, Goldstein & Siegel, 1997). Specifically, impairments occurred in skilled motor, complex memory, complex language, concept formation, and problem solving domains with intact or superior performance in the attention, simple memory, simple language, rule learning, and visuospatial domains. Decreasing performance with increasing complexity is not an observation that is unique to individuals with autism; however, these individuals show deficits at lower levels of complexity than expected relative to age and general ability level (Minshew, Johnson & Luna, 2001). This profile of deficits explains why the average IQ scores of high functioning individuals with autism fail to predict age appropriate adaptive behavior and social function (Minshew & Goldstein, 1998). The selective impairments in higher order cognitive functions means that individuals with autism must accomplish tasks using lower order abilities resulting in inefficiencies of learning and oddities of performance associated with autism.

Following these initial studies, Minshew and her colleagues examined differences in the way individuals with autism process information in a number of different cognitive domains. Results from these investigations revealed a pattern across domains of a lack of use of organizational strategies that help to lessen information processing demands. For example, within the memory domain, high functioning individuals with autism generally have good rote memory; however, they do not usually use implicit organizational strategies to enhance memory, leading to decreasing performance as the complexity of the stimulus increases (Minshew & Goldstein, 2001; Williams, Goldstein, & Minshew, in 2005; Williams, Goldstein, & Minshew, under review). Additionally, a disassociation between the concept identification and concept formation aspects of abstract reasoning has been documented in individuals with autism (Minshew, Meyer & Goldstein, 2002). The result of this deficit in concept formation is a lack of cognitive flexibility with an associated inability to spontaneously form schemata or paradigms that organize information (Goldstein, Williams, & Minshew in press).

The understanding of autism as a disorder of complex information processing or higher order integrative functioning coupled with findings from functional magnetic resonance imaging (fMRI) studies has led to a model of the underlying neuropathology of autism, the underconnectivity model (Just et al., 2004). Minshew, Just and colleagues view the disorder as the behavioral manifestations of a deficit in the coordination of processing by the neural systems both within and across cortical areas. This processing problem is thought to be the result of two interrelated neurodevelopmental abnormalities (Minshew, Johnson & Luna, 2001). The first is an abnormal development of connections of secondary and heteromodal association cortex that disrupts the integration of cortical systems needed to subserve higher cognitive processes. The second is a concurrent disruption of local circuit organization in highly specialized brain regions.

This profile of cognitive strengths and weaknesses helps us to understand how individuals with autism process incoming stimuli and learn new information. We hypothesize that they form strong neural connections in the initial stages of the
learning process that reduce their ability to flexibly alter and reorganize these connections when given new information. Results of recent fMRI studies (Koshino et al., 2005) suggest that even on tasks that individuals with autism perform as well as typical controls, they rely on lower level cognitive abilities. This pattern of deficits results in learning impairments that are seen as the failure to form prototypes for new concepts so that concepts are narrowly defined with only one or two common meanings. Individuals with autism fail to generate organizational strategies even when the organization is inherent in the stimuli. They fail to implement learned strategies at appropriate times unless given explicit cues or are trained through associative learning. While commonly thought to be strong visual learners, individuals with autism may actually have difficulty learning from complex visual stimuli. Because of their propensity to use lower order cognitive abilities, individuals with autism focus on details and fail to form these into a larger whole. They have a related problem with saliency or determining what is the important information and focusing on it.

In our presentation we will discuss the implications of this learning style for the design of therapeutic intervention with individuals with autism. We will also talk about the impact of these cognitive differences on social and adaptive functioning focusing particularly on the challenges faced by older children, adolescents, and adults with high functioning autism.

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References


Biographical Information:

Dr. Williams, Assistant Professor, University of Pittsburgh, is an SLP with 20 years of clinical experience prior to acquiring her doctorate. She has published studies and conducted numerous presentations in the area of developmental disorders and autism. Currently she is studying concept formation and language processing in high-functioning adolescents and adults with autism using behavioral measures and fMRI.

Dr. Minshew, Professor, Psychiatry/Neurology, University of Pittsburgh; board-certified child neurologist; Director of an NICHD Collaborative Program of Excellence in Autism. Her research has characterized autism as selectively involving complex information processing with underdevelopment of wiring of neocortical systems that link brain regions to perform increasingly more complex cognitive functions.