

Brief Report: Neuropsychology of Autism: A Report on the State of the Science¹

Geraldine Dawson²

University of Washington, Seattle

An extensive literature on the neuropsychology of autism exists. The literature is complex and sometimes inconsistent. The complexity comes in part from the fact that an understanding of the neuropsychology of autism depends upon knowledge of the development and assessment of normal brain functioning, a nascent and challenging topic itself. In particular, developmentally sensitive neuropsychological tests across a wide range of functional domains are lacking. The inconsistency found across studies arises for at least two reasons. First, tremendous heterogeneity exists in the behavioral expression of autism, and studies differ substantially in the subject populations they examine. Indeed, it would be surprising if one neuropsychological profile applied to all persons with autism, in view of their widely varying language, cognitive, and social capabilities. Second, whether or not impairments are found in a given study is critically dependent upon the comparison group used (e.g., mentally retarded vs. normal vs. learning disabled) and the variable on which the groups are matched (e.g., IQ vs. language vs. nonverbal ability). Currently, there are no common rules that investigators use to choose their control groups and matching strategies. This probably impedes progress in the field.

Despite these caveats, some basic conclusions can be derived from the literature. To begin, it is clear that persons with autism have neuropsychological impairments in a wide range of domains, a fact that suggests that autism probably involves dysfunction of multiple regions rather than

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²All correspondence should be sent to Geraldine Dawson, Department of Psychology, Box 351525, University of Washington, Seattle, Washington 98195.

Table 1. Neuropsychological Findings in Autism

Domain	Impaired function	Spared function
Intelligence	Verbal, abstract, sequential	Visuospatial organization
Attention	Orient Shifting/disengaging Selective attention	Sustained attention
Memory (Higher functioning)	Mild long-term memory, esp. complex information Strategies for encoding complex info Sequential, abstract information	Paired associate learning Auditory rote memory Cued recall
Memory (Lower functioning)	Short- and long-term memory Declarative memory	Discrimination learning Operant learning
Language (Higher functioning)	Pragmatics Prosody Comprehension of complex verbal information	Phonology Syntax
Language (Lower functioning)	Severe expressive and receptive language (e.g., mutism)	
Executive functions	Working memory Inhibition Planning/organization Flexibility/set shifting	

of only one brain region. Further, in every domain, except possibly executive functions, impairments are not found across the board (Table 1). Some brain functions are spared within each affected domain.

Most studies reveal large individual differences in certain neuropsychological domains. Whereas most persons with autism have frontal lobe impairments (Ozonoff, Pennington, & Rogers, 1991; Rumsey & Hamburger, 1988), great variability exists in the areas of language and memory. Language difficulties may range from mutism to mild pragmatic difficulties. Severely affected persons with autism may exhibit impairments in basic memory functions, such as visual recognition memory, which are known to be mediated by the medial temporal lobe (hippocampus and amygdala) (Barth, Fein, & Waterhouse, 1995). Less affected persons have more subtle memory problems, such as problems in working memory and in the encoding of complex verbal material, which are indicative of higher cortical dysfunction (Ameli, Courchesne, Lincoln, Kaufman, & Grillon, 1988; Minshew & Goldstein, 1993; Ozonoff et al., 1991).

Another potentially important finding in autism research is that individuals with autism have more difficulty when the task involves social interaction. Early in life, young children with autism have more difficulty (a) imitating body actions than imitating toy actions, (b) orienting to social stimuli than orienting to nonsocial stimuli, and (c) pointing to show than pointing to indicate own need (Curcio, 1978; Dawson, Meltzoff, & Oster-

Table II. Hypothesized Neural Substrates for Early Symptoms of Autism^a

Symptom	Possible impairment	Proposed substrate ^b	Reference
Orienting to social stimuli	Recognition of affective significance of stimuli Social stimulus-reward associations	AMYG	LeDoux, 1987
Motor imitation	Disengagement/shift attention Perception of body movements Cross-modal association Long-term memory Representation of action plans Motor planning and execution	AMYG AMYG AMYG, HIP FL FL, BG	Jones & Mishkin, 1972 Courchesne et al., 1994 Brothers et al., 1990 Murray & Mishkin, 1985 Mishkin & Appenzeller, 1987 Goldman-Rakic, 1987 Damasio & Maurer, 1978
Joint attention	Orienting to social stimuli Perception of gaze direction Rapid shifts in attention	See above AMYG CER	See above Brothers et al., 1990 Courchesne et al., 1994
Empathy	Perception of emotion Motor imitation	AMYG See above	Adolphs et al., 1994 See above

^aParts of this table were included in figure 3 of "From mind to molecule: Researches try to unravel the complexity of autism," *Journal of NIH Research*, 7, 1995. Dawson (1995) is the original source of this material.

^bAMYG = amygdala; HIP = hippocampus; CER = cerebellum; FL = frontal lobe; BG = basal ganglia.

ling, 1995; DeMeyer et al., 1972). Later, individuals with autism have more difficulty (d) forming a theory of mind than forming physical second-order representations, (e) comprehending pragmatic language rules than comprehending syntactic rules, and so on (Leekam & Perner, 1991; Tager-Flusberg, 1989). This pattern of findings raises the question of whether or not autism involves dysfunction of a brain system specialized for social cognition. Animal and human lesion studies have led some investigators to suggest that parts of the medial temporal lobe (amygdala, hippocampus, and entorhinal cortex) and the orbital frontal lobe are likely to constitute such a brain system (Allman & Brothers, 1994; Brothers, 1990; Damasio, 1994; LeDoux, 1994). As illustrated in Table II, studies suggest that the amygdala, in particular, may be linked to a cluster of early autistic symptoms, consisting of difficulties in social orienting, motor imitation, joint attention, and empathy (Dawson et al., 1995; Mundy, Sigman, Ungerer, & Sherman, 1986; Osterling & Dawson, 1994; Smith & Bryson, 1994; Sigman, Kasari, Kwon, & Yirmiya, 1992). The amygdala and related brain structures appear to mediate motivational, attentional, and representational processes required for social orienting, imitation, joint attention, and empathy. Furthermore, some investigators have hypothesized that the variability in functioning found in autism may be accounted for by differences in the extent of medial temporal lobe dysfunction (Bachevalier, 1994; Dawson, 1995; Fein & Waterhouse, 1985). Based on an animal model, Bachevalier (1994) proposed that

individuals with extensive dysfunction of the limbic system, involving both the hippocampus and amygdala, will show more severe memory and social impairments than those with selective dysfunction of only the amygdala. This view is not incompatible with a model proposed by Courchesne, Chisum, and Townsend (1994) in which early cerebellar dysfunction is hypothesized to affect the development of limbic regions in a variable way. Consistent with Bachevalier's model, in a study of young children with autism, Dawson, Meltzoff, Rinaldi, and Osterling (1996) found that performance on a medial temporal lobe task (delayed nonmatching to sample) correlated strongly with degree of impairment in social orienting, imitation, joint attention, and empathy. In contrast, only motor imitation ability was found to be correlated with performance on a task that taps dorsal lateral prefrontal functioning. These findings do not indicate that the frontal lobe and other higher cortical regions (e.g., association and parietal cortex) do not play an important role in autism (Courchesne et al., 1994; Minshew, Goldstein, Muenz, & Payton, 1992; Rogers & Pennington, 1991). Other studies have found an association between executive function skill and autistic symptoms, including theory-of-mind impairments (McEvoy, Rogers, & Pennington, 1993; Ozonoff et al., 1991). The Dawson et al. (1996) findings, however, do support the notion that degree of medial temporal lobe dysfunction may amount in part for the variability found in early symptom expression in autism.

The great variability in autistic symptom expression raises the question of whether or not there are distinct neuropsychological subtypes of autism. Evidence for distinct subtypes comes from the work of Rapin (in press) who has been examining this question in a study of approximately 200 children with pervasive developmental disorder. This and other research suggest that more severely affected persons tend to have lower IQs, severely impaired language, and memory impairments on both medial temporal lobe and prefrontal memory tasks. Less severely affected persons tend to have higher IQs, mild language impairments, and memory impairments only on tasks that require working memory or complex encoding strategies (see Table I). Other support for a subtype model comes from studies that have documented a clustering of certain autistic symptoms in children of differing social ability (Castelloe & Dawson, 1993; Volkmar, Cohen, Bregman, Hooks, & Stevenson, 1989), and the existence of different patterns of brain activity in individuals classified by social subtypes (Dawson, Klinger, Panagiotides, Lewy, & Castelloe, 1995). Future research may eventually lead to the conclusion that the heterogeneity in autism is better characterized as a continuum of impairment than as distinct subtypes. Regardless, it is almost certain that different neuropsychological profiles will exist for individuals functioning at each end of the autistic spectrum. The challenge

of the future is to characterize those profiles in order to pinpoint more effective and individualized intervention strategies for persons with autism.

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