

## Autism as a Disorder of Complex Information Processing<sup>1</sup>

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This chapter reviews the evidence leading to the proposal of the neurobehavioral model, or conceptual construct, of autism as a disorder of complex information processing that spares the visual-spatial system. This model is a multiple primary cognitive deficit model proposing that the pattern of deficits within and across cognitive domains in autism is a reflection of complex information processing demands.

### NEUROBEHAVIORAL MODELS IN NEUROBIOLOGIC CONTEXT

Neurobehavioral models for autism are hypotheses about the cognitive basis of behavior and its neural representation in the brain. Numerous such models have been proposed for autism in the decades since a neurologic origin gained acceptance. These models, and the large body of research they arose from and led to, have resulted in major progress in the characterization of the neurocognitive basis of autism with several major consequences. First, this research has led to substantial improvements in diagnostic criteria, recognition of affected individuals, and treatment. Second, recent developments in the definition of structural and functional abnormalities of the brain have culminated in the recognition that the brain in autism

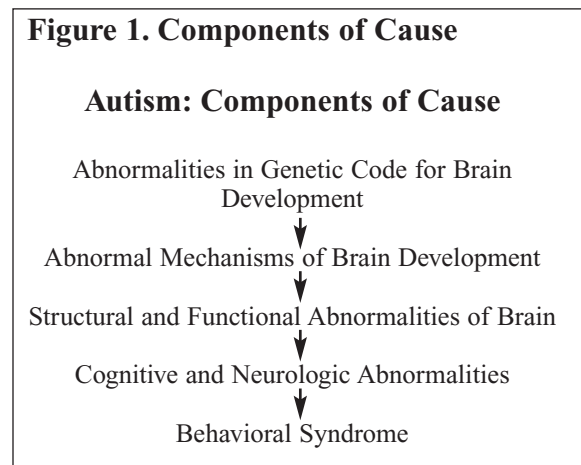
reflects the unique effects of disruption of the dynamics of brain development. A third milestone resulting from this research has been the recognition of partially affected family members and the resulting appreciation of autism as a family genetic disorder with multiple probable genetic loci. Collectively, these research contributions have led to the current conceptualization of the neurobiology of autism as originating with familial abnormalities in the genome that code for brain development. Multiple families of gene abnormalities are anticipated, reflecting various clinical phenomena. These gene abnormalities are expected to code for various abnormal mechanisms for brain development, which culminate in the structural and functional abnormalities of the brain seen in autism. These functional and structural abnormalities constitute the neural basis for the cognitive impairments underlying the behavior that defines autism (see Figure 1). The achievement of this conceptualization of the neurobiology of autism has been the product of decades of research and has made

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the long-term goal of developing corrective neurobiologic interventions for autism finally conceivable. The eventual attainment of this goal is dependent on achieving a detailed characterization of each of the elements in this neurobiologic chain of events. The continuing investigation of the cognitive and neural basis of autism in future research can be expected to play as significant a guiding role in reaching this goal as it did in making such a goal feasible.

**Figure 1. Components of Cause**



### **EVOLUTION IN NEUROBEHAVIORAL MODELS FOR AUTISM**

The investigation of the cognitive and neural basis of autism has led to numerous neurobehavioral models in the 35 years since a neurologic origin for autism first gained acceptance (Rimland, 1964). These models reflect a stepwise series of progressively improving approximations of the underlying pathophysiology that resulted from research investigating important hypotheses about the neurocognitive basis of autism. Current neurobehavioral models are a composite reflection of the accumulated knowledge of decades of research as well as the considerable remaining unknowns about autism and about the normal human brain, cognition, and behavior. Neurobehavioral models are thus,

by definition, temporary conceptual constructs that organize existing findings into testable hypotheses for further investigation.

The earliest neurobehavioral models for autism emerged in the 1960s and 1970s and generally proposed a single primary deficit in an aspect of information acquisition as the cognitive basis for this behavioral syndrome. Therefore, the earliest models hypothesized deficits in sensory perception, brainstem attentional or arousal mechanisms, or associative memory. These models were ultimately abandoned when the previously demonstrated abnormalities in post-rotary nystagmus, brainstem auditory-evoked potentials, temporal horn ventricular size on imaging, and associative memory were found to be the result of the inclusion of a substantial number of autistic subjects with coexisting causes for these findings. Repetition of these studies with autistic subjects screened to exclude those with other disorders causing brain damage failed to provide evidence of abnormalities (Campbell, Rosenbloom, & Perry, 1982; Creasey et al., 1986; Courchesne & Lincoln, 1985; Courchesne, Hicks, & Lincoln et al., 1985; Damasio, Maurer, Damasio, & Chui, 1980; Dunn, 1989; Minshew & Goldstein, 1993; Ornitz, Atwell, Kaplan, & Westlake, 1985; Ornitz, Sugiyama & deTraversay, 1993; Prior, Tress, Hoffman, & Bolt, 1984; Rumsey, Grimes, Pikus, Duara, & Ismond, 1984). However, even the latter studies provided little neuropsychologic evidence documenting the status of sensory perception, attention, and associative memory abilities in autism.

A second, shorter-lived group of neurobehavioral models emerged in the 1970s, proposing a left hemisphere-language acquisition defect or a lack of hemispheric specialization as the basis for autism (reviewed in Minshew, 1994). By the mid-1980s, however, neuropathologic (Bauman & Kemper, 1985) and neurophysiologic studies (reviewed in Minshew,

1991) had consistently demonstrated a bilaterally symmetric pattern of brain involvement in autism. In addition, evidence of right hemisphere language deficits involving prosody, gesture, and facial expression was emerging, highlighting the limitations of drawing conclusions about the brain localization for autism based on the localization of a single cognitive deficit.

In 1980, the first formal information processing model for autism was proposed. This model was based on the first report in autism of attenuation or absence of auditory P300 evoked potentials with sparing of visual P300 potentials, which led to the hypothesis of a selective auditory information processing defect (Novick, Kurtzberg, & Vaughan, 1979; Novick, Kurtzberg, Vaughan, & Simpson, 1980). In light of the intact behavioral performance of the subjects, Novick and colleagues (1980) proposed that the neurophysiologic abnormality reflected the reliance by parietal cortex on less efficient neural pathways for the processing of auditory information. The disparity observed between auditory and visual P300 potential abnormalities in autism was replicated by subsequent investigators (Dunn, 1989), and led to questions about the involvement of visual information processing and the posterior regions of the cerebral hemispheres. These neurophysiologic findings were among the first data demonstrating the central involvement in autism of information analysis and evaluation rather than of information acquisition. These data were also of major significance for providing documentation of the consistent and reliable conduction of sensory information to the cerebral cortex and of the consistency of sensory perception by individuals with autism (Minschew, Sweeny, & Bauman, 1997).

In the latter 1980s and early 1990s, several major cognitive findings were reported that led to a number of new neurobehavioral

models. In 1988, a study of neuropsychologic functioning across domains in 10 autistic men with average group mean verbal and performance IQ scores documented a cognitive profile characterized by dramatic deficits in conceptual reasoning abilities, relatively intact language, memory, and motor abilities, and intact sensory perception and visual-spatial abilities (Rumsey & Hamburger, 1988). Based on this profile, Rumsey and Hamburger proposed a core deficit in a broad class of verbal and non-verbal conceptual reasoning abilities, but had difficulty relating this deficit conceptually to the behavioral syndrome of autism. Ozonoff, Pennington, and Rogers (1991) replicated this overall profile and accumulated evidence of executive function deficits, which led to their proposal of an influential executive dysfunction-frontal systems model for autism (Ozonoff, Pennington, & Rogers, 1991). In a second significant study, Ozonoff, McMahon, & Filloux (1994) investigated the specific cognitive components in autism responsible for executive dysfunction on the Wisconsin Card Sorting Test. They identified the cognitive flexibility component as the major source of impaired performance, and the inhibition of prepotent responses as making a modest contribution. Notably, they found that components related to shifting attention between different features of an object and to inhibiting responses failed to demonstrate impairments, leading Ozonoff and colleagues to propose that autistic individuals' perseverative focus of attention on details had a conceptual rather than a perceptual basis. This study was also significant for demonstrating that complex cognitive tasks received contributions from multiple component processes that could be separated with appropriate procedures and that the impaired performance of different neuropsychiatric populations could be traced to dysfunction of different components.

A second major recent contribution to neurobehavioral conceptualizations of autism was the recognition of “theory of mind” deficits as a major cognitive mechanism underlying the abnormal social behavior in autism. The identification of this cognitive ability and its impairment in autism demystified social behavior in autism and brought it clearly into the realm of cognitive psychology. It was also a milestone, and likely the first of many, for having identified a previously unsuspected cognitive ability responsible for an important aspect of human behavior (Tooby & Cosmides, 1995). The model proposed for theory of mind abilities in autism was also notable for highlighting the role of multiple cognitive abilities acting in concert in novel ways to subservise complex human behavior (Baron-Cohen, 1995). Thus, the impairment in the capacity for making inferences about the mental beliefs and knowledge of others was seen as functionally linked to deficits in the social use of eye contact. This model thus converged with reports on autism of deficits in the use of eye contact to achieve shared attention (Sigman, 1996), another newly recognized cognitive or neurologic function contributing to social behavior. These two newly recognized abilities provided clear examples of the many yet-to-be defined cognitive abilities that contribute to the complex cognitive and behavioral competencies impaired in autism, and of the likely need to reconceptualize the cognitive contributions to other impaired abilities in autism as new research findings are reported.

A third major influence on recent neurocognitive models for autism pertains to reports of attentional deficits. Deficits in attention have been proposed repeatedly throughout the course of autism research in an attempt to explain the autistic individual’s intense focus on details, on the one hand, and a lack of interest in people, on the other.

Although recent research studies summarized previously have provided evidence for a conceptual basis for these abnormalities in behavior, deficits in attention continue to be reported and proposed as the primary cognitive basis for behavior in autism. Deficits in selective attention, attention to extra-personal space, and shifting attention have been among those recently proposed (Courchesne et al., 1993; Ornitz, 1988; Townsend & Courchesne, 1994). The first two of these deficits were inferred from neurophysiologic abnormalities in the absence of impairments in cognitive performance and imaging abnormalities involving the parietal lobe, respectively. The shifting attention deficit was documented with a cognitive paradigm, but the paradigm also had substantial executive function and working memory demands in addition to the demand for an attentional shift at the perceptual level. The multiple demands of this task made it impossible to determine the cognitive origin for the impaired performance in autism without investigating these contributions individually. A number of subsequent studies attempted to clarify the role of attentional processes in autism. Collectively, these studies examined the reflexive and voluntary or executive control of attention in individuals with autism of varying levels of ability (reviewed in Burack, Enns, & Johannes, 1997). These studies provided evidence that abnormalities in attentional focus in autism are related to the information processing aspects of the tasks and the voluntary or executive control of attention, and not to deficits in reflexive orienting abilities. These latter studies were also of major importance in highlighting the influence of developmental level or general ability level on the expression of deficits in autism, and the limitations of conclusions about core deficits that did not consider these influences.

Up to this point, neurobehavioral models for autism generally were single primary cognitive deficit models, proposing a clinically apparent deficit in a single cognitive domain or modality as underlying the social, communication, and odd nonsocial behavior in autism. By the early 1990s, however, substantive evidence of deficits in several higher-order cognitive abilities had emerged, posing a major question for the validity of single primary deficit models. As most of the cognitive and neuropsychologic studies in autism had focused on a single cognitive domain, their design precluded the identification of potential deficits in other domains and the consideration of their significance in the various neurobehavioral models.

### **PROFILE OF NEUROPSYCHOLOGIC FUNCTIONING IN AUTISM**

Examination of cognitive functioning across domains within the same subject sample provided an obvious opportunity for addressing the issue of single versus multiple co-existing cognitive deficits in autism. It also provided an opportunity for observing the pattern of these deficits, which itself might contain additional important clues to the underlying neurobiology. One of the first studies to investigate neuropsychologic functioning across domains in a group of autistic subjects screened to exclude those with causes of brain dysfunction other than autism was that of Rumsey and Hamburger (1988). This study was viewed as remarkable for the demonstration of dramatic impairments in the reasoning domain that were not explainable by deficits in other domains. However, less noticed was the characterization of the language, memory, and motor domains as “relatively intact,” whereas sensory perception and visual-spatial abilities were described as intact. Examination of the test

battery revealed only a few tests and mixed results in the language, memory, and motor domains, whereas there were a larger number and broader range of tests for the reasoning domain. The relatively intact domains reflected a combination of good performance on tests of simpler abilities and impairments on tests of more complex abilities, especially in the memory and language areas. A number of investigators subsequently replicated this profile, emphasizing the evidence for executive dysfunction but again relying on few tests in the language and memory domains, with the same mixed results.

One study attempted to address this issue by expanding the memory and language test battery and by separately considering simple and complex abilities at analysis (Minschew et al., 1992). This study of 15 nonmentally retarded individuals with autism revealed intact function on memory tests of simple associative processes and on language tests of basic skills such as word fluency, reading decoding, and spelling. Memory deficits were documented on delayed recall measures, suggesting that information encoding was not sufficiently supported by organizing strategies. Language deficits were documented on tests of higher-order abilities, such as comprehension of idioms, metaphors, and ambiguous sentences. This pattern of findings in the language and memory domains suggested the presence of a dissociation in autism between simple and complex abilities. The second major finding of this study was the absence of impairments on the Wisconsin Card Sorting Test (WCST). Deficits on this test had come to be viewed as a hallmark of the abstraction deficit in autism as a result of the extensive investigation of executive dysfunction based on this test. Instead, abstraction deficits were demonstrated on the Goldstein-Scheerer Object Sorting Test with a test of verbal reasoning and in the capacity

to shift concepts. The absence of deficits on the WCST in this study was attributed to the higher level of function of the autistic subjects compared to prior studies. In these subjects, the deficit was better characterized by deficits on concept-formation tests than by rule-learning tests, such as the WCST. The third significant finding of this study was the presence of deficits on Part A, but not on Part B, of the Trail Making Test. Deficits on Part B are typically viewed as evidence of problems with executive function or shifting attention, as subserved by the frontal lobes. Part A has minimal cognitive demands and serves as practice for Part B; the major demand of part A is on psychomotor skills. The intact performance by the subjects with autism on Part B, but with impairments on Part A, suggested the presence of psychomotor slowing. Review of the Rumsey and Hamburger (1988) Trail Making Test data revealed that their subjects had exhibited greater deficits on Part A than on Part B, as well as exhibiting evidence of psychomotor slowing on a finger-tapping task.

Several of the findings from the preceding study were amplified in follow-up studies. Concept formation ability was investigated further with the Twenty Questions Procedure, which requires subjects to identify a preselected object from an array using a maximum of 20 questions. The most efficient strategy is to formulate constraint-seeking questions that involve characteristics shared by several objects, which can eliminate several alternatives at once and thus progressively narrow the possibilities to the target item. Four trials were administered in a study comparing the problem-solving skills of nonmentally retarded individuals with autism and matched control subjects. The autistic subjects solved significantly fewer of the four trials and used a significantly smaller number of constraint-seeking questions. Their impaired performance on this test of the

concept formation aspect of abstraction was contrasted with their intact performance on tests of the rule-learning aspect of abstraction, a less challenging aspect of abstraction. The mixed pattern of results in the language and memory area in the Minshe et al. study (1992) led to an in-depth examination of the language domain investigating the hypothesis of a dissociation between preserved simple abilities and impaired complex abilities (Minshe, Goldstein, Taylor, & Siegel, 1994; Minshe, Goldstein, & Siegel, 1995). In these studies, the simple language category was comprised of tests of mechanical skills, such as verbal fluency, mechanical reading, word recognition, spelling, phonetic analysis, and simple calculation. The complex language category included tests of interpretive abilities, such as reading comprehension, understanding of the metaphorical aspects of spoken and written language, and verbal reasoning. The performance of high-functioning subjects with autism was compared to that of normal community volunteers matched by age, gender, race, IQ, and socioeconomic status (SES). The results of the study indicated that the autistic subjects did as well and often better than control subjects on the tests of mechanical language skills, but significantly more poorly on tests of complex interpretive skills. These studies provided additional evidence suggesting that subjects with autism had selectively failed to acquire the higher-level interpretive language abilities expected on the basis of their age, verbal IQ score, and basic language skills.

### **EVIDENCE FOR A COMPLEX INFORMATION PROCESSING DISORDER**

In light of these findings, a third study of the profile of neuropsychologic functioning was designed to further characterize the

pattern within and across domains in a large group of rigorously defined subjects with autism and individually matched controls. The test battery was expanded and designed to address the neuropsychologic deficits hypothesized by various neurobehavioral models for autism, as well as to address the hypothesis of selective involvement of higher-order cognitive abilities related to generalized dysfunction of association cortex. The battery was composed of valid and reliable neuropsychologic tests assessing the major cognitive domains of attention, sensory perception, motor function, language, memory, reasoning, and visual-spatial abilities (see Table 1 in the chapter appendix, "Results of Neuropsychological Tests Assessing the Major Cognitive Domains.") The visual-spatial domain was included because its status was important in completing the profile of cognitive functioning in autism, although visual-spatial abilities have long been considered a strength of individuals with autism. A range of abilities was considered within each domain to address the various hypothesized deficits, and both verbal and visual modalities were assessed where appropriate. The large number of measures relevant to the assessment of simple and complex language and memory abilities in both the visual and auditory modalities required subdivision of these cognitive domains into simple and complex categories for separate analysis. In other domains, the number of tests was fewer and individual consideration of the tests within domains was relied upon to characterize the features related to deficits and intact abilities. Tests in each domain were considered as multivariate sets, and stepwise discriminate function analyses were used to evaluate the accuracy of each set in correctly classifying cases into autistic and control groups. Classification accuracy was assessed with Cohen's kappa, an index of strength of

agreement for nominal scales. Tests included and not included in the regression equations and their order of entry provided additional information on which tests had the most discriminatory power. Individual t-tests were computed to clarify performance on tests not included in the regression equations (see Table 2, chapter appendix).

Tests in the attention, sensory perception, simple memory, and visual-spatial domains did not yield satisfactory classification accuracy, providing evidence of intact basic information acquisition abilities and intact information processing in the visual-spatial domain. *Kappa* scores in the fair to good agreement range (0.40-0.75) were obtained for the motor, simple language, complex language, complex memory, and abstract reasoning domains. For the simple language category, the significant *kappa* score reflected superior performance by the subjects with autism relative to control subjects, in contrast to the motor, complex memory, complex language, and reasoning categories where the significant *kappas* reflected impairments.

Examination of tests entered and not entered into the regression equations, order of entry, and individual t-test results provided additional evidence about the nature of the deficit pattern. The attention domain was most notable for the absence of evidence of deficits. Only tests with a motor component—the letter and number cancellation tasks—were entered into the regression equation, and these failed to achieve significant classificatory accuracy. Performance on these two tests was notable for the low rate of errors by both subject groups and the absence of a predilection for any quadrant; thus, there was no support for a hypothesized deficit in attention to extra-personal space. In the motor domain, it was of note that discriminatory accuracy was achieved with the Grooved Pegboard Test and Trail Making Test, Part A,

the two tests of skilled motor sequences. In contrast, there was no difference between subjects with autism and control subjects on the test of simple or isolated motor movements (Finger-tapping Test). T-tests revealed significantly poorer performance on Part A, but not Part B, of the Trail Making Test, which is consistent with prior observations and the assignment of Part A to the motor domain and Part B to the reasoning domain. In the complex language and complex memory domains, test entry was notable for including both verbal and visual tests, thus failing to support the hypothesis of a selective auditory processing deficit in autism. In the reasoning domain, the WCST and the Halstead Category Test (two tests of the rule-learning aspect of abstract reasoning) failed the tolerance test consistent with the previously reported findings in nonmentally retarded individuals with autism (Minshew et al., 1992). The first test passing the tolerance test for the reasoning domain was the Twenty Questions Procedure, which is a concept formation test. This was followed by the Picture Absurdities subtest of the Binet scales, which requires consideration of context and a conceptual framework in order to identify incongruities, and the Trail Making Test, Part B, which challenges working memory and shifting cognitive sets (executive function). The selection of these three tests suggests that the reasoning deficit in autism involves a broad range of conceptual abilities as previously proposed by Rumsey and Hamburger (1988), and that executive dysfunction or cognitive inflexibility might be too narrow to encompass the deficit.

The profile of cognitive functioning in these nonmentally retarded autistic adolescents and adults was therefore defined by deficits in concept formation, complex memory, complex language, and skilled motor abilities and by intact or superior function in

the attention, sensory perception, simple memory, simple language, rule-learning, and visual-spatial areas. The implications of these findings are several.

## RESEARCH IMPLICATIONS

The characterization of the cognitive profile in terms of both deficits and intact abilities is significant, as it demonstrates the distinctions between autism and general mental retardation, on the one hand, and the developmental specific learning disabilities, on the other. The two-part characterization also demonstrates the selective impact of autism on higher-order abilities. Thus, the presence of age- and IQ-appropriate performance on tests of spelling, reading, arithmetic, and visual-spatial abilities distinguishes autism from the developmental specific learning disabilities and the nonverbal learning disability syndrome. The intact language, memory, arithmetic, rule-learning and visual-spatial abilities account for the attainment of IQ scores in the average range. The deficits in problem solving, concept formation, complex language and complex memory abilities explain the failure of the average IQ scores to be accurate predictors of adaptive behavior and function in society. This dissociation between intact and deficit skills also explains the clinical observation that abstraction, communication, and social abilities fall rapidly (or disproportionately) with declining IQ in the autistic population as compared to the nonautistic, mentally retarded population, and the lower adaptive function of mentally retarded individuals with autism compared to mentally retarded individuals without autism of the same general level ability.

In addition to demonstrating the selective impact of autism on higher-order cognitive abilities, the documented intact abilities fail to support neurobehavioral models that

hypothesize clinically apparent deficits in sensory perception, attention at the perceptual level, and associative memory as the basis for autism. The integrity of these basic abilities also demonstrates that the deficits documented in concept formation, complex memory, complex language, and skilled motor abilities are not secondary to deficits in more elementary abilities.

A second unique feature of cognitive functioning in the subjects with autism was the pattern of intact simpler abilities in domains demonstrating deficits. Perhaps contrary to expectations, a deficit in the abstract reasoning domain, for example, did not mean that all abstraction was impaired. Rather, deficits in each domain involved the highest level abilities expected on the basis of an individual's age and IQ, while leaving simpler abilities intact or even enhanced. That is, in each domain, deficits appeared to correspond to the highest level tasks, thus depending on the most cognitively advanced abilities, whereas intact function appeared to correspond to the simplest or most basic skills. This pattern conformed to the neurophysiologic pattern reported for autism of impaired, late cognitive potentials and intact earlier potentials. Across domains, complexity also appeared to account for the predilection of deficits for those domains with the highest demands on information processing. The consistency of this pattern within and across domains and with the neurophysiologic pattern suggests that it reflects a neurobiologic feature or principle of brain structure and function.

As one way of probing the validity of this conceptualization or characterization of cognitive functioning in autism, the cognitive profile defined in this study was compared with that reported by Tallal for a disorder of early or simple information processing (Jernigan, Hesselink, Sowell, &

Tallal, 1991; Johnston, Stark, Mellits, & Tallal, 1981; Neville et al., 1993; Tallal & Piercy, 1973; Tallal et al., 1996). This comparison revealed that the cognitive profile in autism was the converse of that described by Tallal for children with developmental specific learning impairment (SLI). As in autism, the neuropsychologic profile in children with SLI involved multiple domains but included the attention domain as well as the sensory perception, motor, memory, and language domains. Unlike autism, the deficits involved the elementary or simple abilities; namely, basic attentional processes, sensory perception, elementary motor, simple memory, and simple language abilities. This profile was found to correspond to a disturbance in early information processing, resulting in the failure to acquire information dependent on the first 100 msec of information processing. In contrast, higher-order interpretative and reasoning skills were intact, and the children could sometimes use these abilities to fill in or infer missing information.

Evidence of a deficit in complex abilities in the motor domain also supports the neurobiologic validity of a complex information processing construct in autism. That is, the presence of a dissociation between simple and complex abilities in an area of minor clinical involvement would also suggest that the dissociation reflects a fundamental feature of the neurobiology. The coexistence of a similar pattern across domains suggests that the deficits are dependent on a common neural substrate or organizing principle of the brain.

In arriving at the characterization of the cognitive profile in autism as reflecting a complex information processing disorder, consideration was given to the ways that complexity is defined. Within cognitive theory, complexity is defined in several ways, including number of elements contained in the stimulus material as well as the multiplicity of

cognitive processes involved in task performance. The latter definition involves emergent abilities that are not directly reducible to simpler elements of cognitive function (i.e., the reductionist fallacy). Thus, the cognitive capacity to comprehend extended blocks of language is not simply reducible to vocabulary and grammar skills, but requires another level of language abilities in order to comprehend the meanings beyond those implicit to vocabulary and the arrangement of words into sentences. The model proposed here does not distinguish between these definitions of complexity, particularly because they are related in the sense that, as the number of elements increases, there is typically an increase in the number of cognitive processes needed for task performance.

The application of a complexity construct to the cognitive profile in autism requires several constraints or specifications to accurately reflect the data from which it was derived. First, the data in this study define deficits by complexity within domains, not independent of domains; thus, the definition of complexity conveyed in this model is domain-related. That is, although any language skill might be viewed from a cognitive perspective as more complex than any motor skill, the deficits found do not conform to a cognitive ranking of relative complexity independent of domain. Rather, the deficit pattern appears to conform to the fact that different cognitive functions are represented by separate neurologic systems in the brain. Second, visual-spatial abilities involve complex information processing but were found to be intact; thus, the disorder of complex information processing in autism must be stipulated to spare the visual-spatial domain. Because the visual-spatial system is a separate neural system, it is reasonable to assume that this neural system could be spared through various neurobiologic mechanisms

without invalidating a complex information processing model for cognitive functioning in other domains. Third, this model was derived from the study of nonmentally retarded adolescents and adults with autism. If it is to be applied to younger or lower-functioning individuals with autism, it is clear that complexity in terms of cognitive function has to be conceptualized in relation to age and IQ. The specific expression of the complex information processing deficit is therefore going to float as a reflection of the age and general ability level of the individual.

Several key aspects of the clinical syndrome of autism were not assessed in this study because of the time-intensive nature of experimental measures or the lack of sufficiently challenging measures for nonmentally retarded individuals with autism. Consequently, the test battery did not assess social or nonverbal language abilities, although deficits in these abilities are implicit to the diagnosis of autism and were documented with the structured instruments used for diagnosis, which were the Autism Diagnostic Interview (LeCouteur et al., 1989; Lord, Rutter, & LeCouteur, 1994) and the Autism Diagnostic Observation Schedule (Lord, Rutter, & Goode, 1989). Nonetheless, the deficits in these areas can be conceptualized within a complex information-processing model. Theory of mind skills are therefore viewed as a higher-order inferential, cognitive ability. Similarly, the modulation of eye contact and facial expression for communication purposes and the comprehension and expression of satire, irony, and innuendo in prosody are likewise viewed as higher-order complex, information-processing skills. On the other hand, deficits were found in complex memory skills that are not obviously related to the clinical criteria for autism. The data supporting their presence is clear, so the issue is how such an impairment might relate

to the clinical deficits. The evolution of theory of mind abilities in relation to autism provides a model for considering the existence of a previously unrecognized cognitive contribution to the clinical manifestations of autism (Baron-Cohen, 1995). The memory data from the present study provided evidence of intact rote memory for simple information in limited amounts but a reduced capacity for remembering information as its complexity increased. This reduced memory capacity applied to an increasing number of units of the same kind, such as words in a sequence and branch points in a maze, as well as to an increase in the intrinsic complexity of the material, as in the case of stories and the Rey-Osterreith complex figure (Minsheu et al., 1996). Thus, these subjects with autism have difficulty remembering increasing amounts of information and discerning the intrinsic organizational structure of information that normally supports memory. Given that social interactions, communication, and problem-solving situations typically involve the presentation of large amounts of information, it would seem likely that a memory impairment of the type found would contribute to impaired function. As proposed by Toobes and Cosmides (1995) in their foreword to Baron-Cohen's book describing the evolution of the theory of mind data and construct in autism, there are many as-yet-undescribed cognitive abilities that are performed so automatically that their existence is not suspected. The theory of mind model described by Baron-Cohen further suggests that cognitive abilities also may act in concert in ways not currently described to support complex capabilities in humans, and that these interactions may also be disrupted in autism. Consistent with this, it has been proposed that the social and language systems must interact in order for communication to be related to a social context and that

these interactions are disrupted in autism. Such an interaction provides a cognitive and neural basis for the use of language for communication. Similarly, it is probable that the memory system interacts at a cognitive and neural systems level with the social, language, and reasoning systems to support the cognitive functions impaired in autism. Ultimately, these relationships will be explored and elucidated with experimental cognitive procedures and fMRI.

### SUMMARY

In summary, this study of neuropsychologic functioning in autism provided evidence of the co-existence of deficits in multiple domains within a single-subject group, supporting a multiple primary cognitive deficit model for the cognitive basis of behavior in autism. No evidence was found of deficits in attention, sensory perception, or associative memory to support neurobehavioral theories hypothesizing clinically apparent deficits in these abilities as the basis of behavior in autism. Within affected domains, impairments consistently involved the most complex tasks dependent on higher-order abilities whereas intact or superior function was found on simpler abilities within the same domains. Across domains, complex information-processing demands also provided an explanation for the particular constellation of deficits that define autism; that is, those domains with the highest complex information-processing demands. The neuropsychologic profile for autism characterized in this study is consistent with the evoked potential pattern of abnormal late, endogenous potentials and preserved earlier potentials, and the converse of the neuropsychologic and neurophysiologic pattern described for a simple or early information-processing disorder. The presence of such a

common denominator within and across domains would suggest that impairments are dependent on a common feature of neuronal organization. As such, there is likely to be a larger class than currently appreciated of yet-to-be-defined cognitive abilities impaired as a result of this disturbance in neuronal organization. Theory of mind abilities and the deficits in complex memory identified in this study are examples of the unknown features of the cognitive basis of autism to be defined in future research. Both of these impairments also highlight the emerging recognition of the importance of disruption in the interactions between different cognitive functions and neural systems as the basis for certain aspects of behavior in autism. ■

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## Appendix

### RESULTS OF NEUROPSYCHOLOGIC TESTS ASSESSING THE MAJOR COGNITIVE DOMAINS

**Table A1. Discriminant Analysis Results By Domain and By Order of Entry**

Domain	Tests Failing Tolerance Test	Tests Passing Tolerance Test	% Correct	% Jackknife	Kappa <sup>1</sup>
Attention	Serial Digit Learning; Digit Span; Continuous Performance	Letter Cancellation; Number Cancellation	66.7	66.7	.33
Sensory Perception	Luria-Nebraska Tactile Scale: Touch, Position, Finger Position and Stereognosis items	Finger Tip Writing; Luria-Nebraska Sharp/Dull Tactile Scale item	64.6	62.5	.29
Motor	Finger Tapping; Developmental Test of Visual Motor Integration	Grooved Pegboard; Trail Making A	75.8	75.8	.52 <sup>1</sup>
Simple Language	WAIS-R Vocabulary	K-TEA Reading Decoding; K-TEA Spelling; WRMT-R Word Attack; Controlled Oral Word Association	71.2	66.7	.42 <sup>1</sup>
Complex Language	WRMT-R Passage Comprehension; TLC- Metaphoric Expression	K-TEA Reading Comprehension; Verbal Absurdities; Token Test	72.7	65.2	.45 <sup>1</sup>
Simple Memory	Paired Associates; 3 Word Short Term Memory; Maze Recall	CVLT Trial 1	65.2	65.2	.30
Complex Memory	Paired Associates-Delayed; CVLT Long Delay	NVSRT-Consistent Long Term Retrieval; WMS-R Logical Memory- Delayed Recall; Rey Figure-Delayed Recall	77.3	75.8	.55 <sup>1</sup>
Reasoning	Category Test; Wisconsin Card Sort Test	20 Questions; Picture Absurdities; Trail Making B	75.8	72.7	.52 <sup>1</sup>
Visual-Spatial	WAIS-R Picture Completion, Object Assembly	WAIS-R Block Design	56.1	56.1	.12

<sup>1</sup>Indicates a significant between-group difference in performance.

**Table A2. Psychometric Data Used for Discriminant Analysis**

Tests Entered into Prediction Equations	Autistic Group		Control Group		p
	<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>	
<u>Attention Domain</u>					
WAIS-R Digit Span	9.88	3.81	10.52	2.46	.424
Serial Digit Learning-Correct Responses	16.52	8.17	17.42	7.91	.648
Continuous Performance Test-Mean Reaction Time Correct Responses	0.34	0.62	0.23	0.66	.487
Letter Cancellation-Omissions	1.09	1.63	0.45	1.00	.061
Number Cancellation-Omissions	3.27	4.03	4.39	5.38	.342
<u>Sensory Perception Domain</u>					
Luria-Nebraska Tactile Scale:					
Simple Touch Errors	0.29	0.55	0.17	0.48	.407
Stereognosis Errors	0.46	0.59	0.21	0.42	.096
Sharp-Dull Discrimination Errors	0.88	0.80	0.58	0.72	.189
Position Sense Errors	0.00	0.00	0.08	0.41	.328
Finger Position Errors	0.67	1.27	0.46	1.02	.535
* Halstead-Reitan: Fingertip Number Writing-Errors	5.38	4.30	2.79	2.84	.019*
<u>Motor Domain</u>					
Finger Tapping-Dominant Hand	44.27	13.78	45.19	16.24	.805
Developmental Test of Visual-Motor Integration-Total Points	15.42	32.43	22.18	31.69	.465
* Grooved Pegboard-Dominant Hand-Time in Seconds	86.73	18.30	70.67	16.03	.000*
* Trail Making A-Time in Seconds	31.52	15.81	20.45	7.99	.001*
<u>Simple Language Domain</u>					
Controlled Oral Word Association (FAS)-Number of Words	36.00	13.31	34.00	16.18	.586
WAIS-R Vocabulary	9.45	3.02	9.70	2.26	.713
K-TEA Spelling	102.58	16.93	100.91	11.50	.642
Woodcock Reading Mastery-Word Attack	107.24	11.55	103.52	15.53	.273
K-TEA Reading Decoding	97.48	13.60	102.79	10.19	.078

*Continued*

**Table A2.** *Continued*

Tests Entered into Prediction Equations	Autistic Group		Control Group		p
	<u>M</u>	<u>SD</u>	<u>M</u>	<u>SD</u>	
<u>Complex Language Domain</u>					
Token Test (number correct)	18.03	2.19	18.42	5.19	.690
* K-TEA Reading Comprehension	91.36	14.43	103.06	12.45	.001*
* Woodcock Reading Mastery-Passage Comprehension	92.27	15.04	104.27	14.34	.002*
* Test of Language Competence-Metaphoric Expression (scaled score)	6.85	3.25	9.42	3.70	.004*
* Binet Verbal Absurdities-Raw Score	9.30	3.64	12.48	3.97	.001*
<u>Simple Memory Domain</u>					
Maze 1 Recall (correct/incorrect)	0.42	0.61	0.52	0.57	.534
3 Word Short Term Memory-Number of Correct Sequences	3.24	3.04	2.91	3.15	.663
Paired-Associate Learning-Number Correct	42.55	23.13	48.76	24.21	.290
CVLT A List-Trial 1 Number Correct	4.50	3.90	6.30	3.90	.072
<u>Complex Memory Domain</u>					
Paired-Associates-Delayed Recall	16.00	7.46	17.45	6.13	.390
CVLT A List-Long Delay	7.00	5.49	9.00	5.55	.146
* WMS-R Logical Memory-Delayed Recall-Elements	5.58	5.79	8.45	6.02	.052*
* Nonverbal Selective Reminding-Consistent Long-term Retrieval	19.94	15.09	37.39	16.09	.000*
* Rey-Osterrieth Figure-Delayed Recall-Number of Elements	16.83	8.58	21.94	7.49	.012*
<u>Reasoning Domain</u>					
Halstead Category Test (errors)	46.24	28.71	40.73	22.46	.388
Wisconsin Card Sorting Test-Perseverative Errors	16.45	15.48	13.27	11.13	.342
Trail Making B (time in seconds)	65.48	37.19	52.42	23.31	.093
* Binet Picture Absurdities (raw score)	20.00	11.46	27.52	6.12	.002*
Questions (% constraint seeking)	35.49	23.82	56.08	14.02	.000*
<u>Visual-Spatial Domain</u>					
WAIS-R Picture Completion	8.76	2.22	9.21	2.27	.415
WAIS-R Object Assembly	9.88	3.63	9.73	2.88	.852
WAIS-R Block Design	10.79	3.25	9.70	2.14	.113
Rey-Osterrieth-copy score	31.30	4.80	33.09	3.75	.096