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# AUTISM AS A DISORDER OF COMPLEX INFORMATION PROCESSING

**Nancy J. Minshew[1]* and Gerald Goldstein[2]**

[1]Department of Psychiatry, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania

[2]VA Pittsburgh Healthcare System, Pittsburgh, Pennsylvania

Numerous neurobehavioral models for autism have been proposed in the decades since a neurologic origin gained acceptance. Research related to these models has resulted in substantial progress in the characterization of the neurocognitive basis of autism. The culmination of considerable research has resulted in support for a neurobehavioral model of autism as a disorder of complex information processing systems. This is a multiple primary cognitive deficit model proposing that the pattern of deficits within and across domains in autism is a reflection of complex information processing demands. This article will first provide an overview of the evolution of neurobehavioral models in autism and then present findings leading to the conceptualization of this model for autism. 
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**Key Words:** autism; neurobehavioral models; information processing; cognitive profiles

This article reviews evidence for the neurobehavioral model or conceptual construct of autism as a disorder of complex information processing that spares the visuospatial system. In this multiple primary cognitive deficit model, the pattern of deficits within and across cognitive domains in autism reflects 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 in the decades since a neurologic origin for autism 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. This research has led to substantial improvements in diagnostic criteria, recognition of affected individuals, and treatment. 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. This research has also led to the recognition of partially affected family members and the 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 in affected individuals. These functional and structural brain abnormalities constitute the neural basis for the cognitive impairments underlying the behavior that defines autism (see Fig. 1). Achieving this conceptualization of the neurobiology of autism has been the product of decades of research and has made the long-term goal of developing corrective neurobiologic interventions for autism at last conceivable. Attaining this goal depends on achieving a detailed characterization of each of the elements in the neurobiologic chain of events that results in autism. 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.

## EVOLUTION IN NEUROBEHAVIORAL MODELS FOR AUTISM

Investigation of the cognitive and neural basis for autism has led to numerous neurobehavioral models since a neurologic origin for autism first gained acceptance [Rimland, 1964]. Research investigating hypotheses about the neurocognitive basis of autism has led to a stepwise series of progressively improving approximations of the underlying pathophysiology. Current neurobehavioral models reflect knowledge gained from decades of research, as well as the many remaining unknowns about autism and, more generally, about the normal human brain, cognition, and behavior. Thus, neurobehavioral models are 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. Such models typically proposed a single primary deficit in some aspect of information acquisition as the cognitive basis for this disorder. For example, the earliest models hypothesized deficits in sensory perception, brainstem attentional or arousal mechanisms, or associative memory. These

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*Correspondence to: Nancy J. Minshew, University of Pittsburgh School of Medicine, 3811 O'Hara Street, Bellefield Towers, Suite 430, Pittsburgh, PA 15213. E-mail: minshewnj@msx.upmc.edu

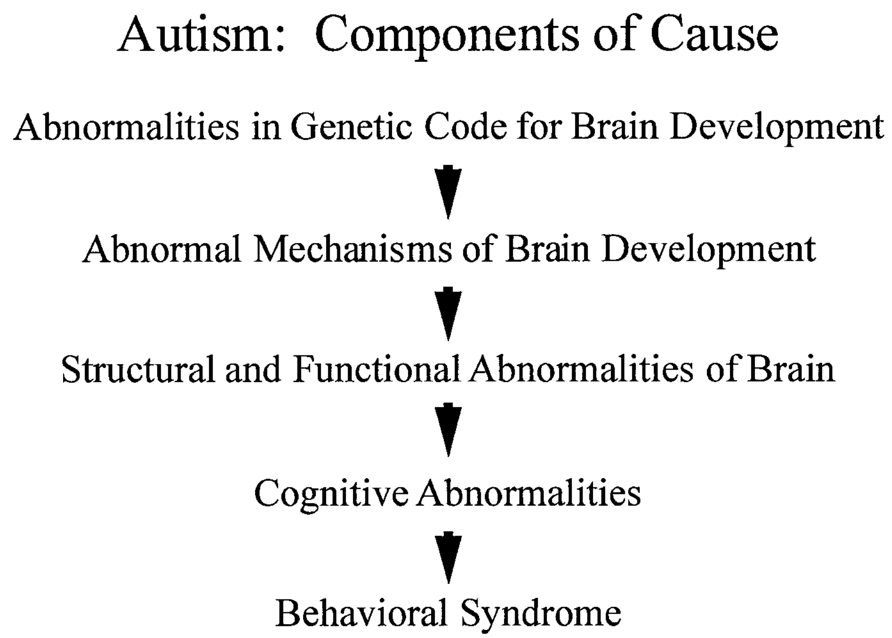

Fig. 1. Proposed components of cause for autism.

models were ultimately abandoned, however, when previously demonstrated abnormalities in post-rotary nystagmus, brainstem auditory evoked potentials, temporal horn ventricular size on imaging, and associative memory were found to result from including a substantial number of autistic participants with coexisting disorders causing brain damage. Samples screened to exclude those with other disorders failed to provide evidence of abnormalities [Campbell et al., 1982; Creasey et al., 1986; Courchesne and Lincoln, 1985; Courchesne et al., 1985; Damasio et al., 1980; Grillon et al., 1989; Minshew and Goldstein, 1993; Ornitz et al., 1985, 1993; Prior et al., 1984; Rumsey et al., 1984]. However, even these 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. These models proposed that the basis for autism was a left hemisphere-language acquisition defect or lack of hemispheric specialization [reviewed in Minshew, 1994]. By the mid-1980s, however, neuropathologic [Bauman and Kemper, 1985] and neurophysiologic studies [reviewed in Minshew, 1991] had consistently demonstrated a bilaterally symmetric pattern of brain involvement. In addition, there was emerging evidence of right hemisphere language deficits involving prosody, gesture, and facial expression. These findings highlighted 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. The model was based on the first report in autism of attenuation or absence of auditory P300-evoked potentials with sparing of visual P300 potentials; this led to the hypothesis of a selective auditory information processing defect [Novick et al., 1979, 1980]. In light of the intact behavioral performance of their participants, Novick and colleagues proposed that the neurophysiologic abnormality reflected the reliance by parietal cortex on less efficient neural pathways for the processing of auditory information [Novick et al., 1980]. The disparity observed between auditory and visual P300 potential abnormalities in autism was replicated by subsequent investigators [reviewed in Dunn, 1994], 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 information acquisition. These data were also of major significance for documenting consistent, reliable conduction of sensory information to the cerebral cortex and consistent sensory perception by individuals with autism [Minshew et al., 1997].

In the late 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 marked deficits in conceptual reasoning abilities, relatively intact language, memory, and motor abilities, and intact sensory perception and visuospatial abilities [Rumsey and Hamburger, 1988]. Based on this profile, Rumsey and Hamburger proposed a core deficit in a broad class of verbal and nonverbal conceptual reasoning abilities. However, they had difficulty relating this deficit conceptually to the behavioral manifestations of autism. Ozonoff et al. [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. In a second major contribution, Ozonoff and colleagues [1994] investigated the specific cognitive components in autism responsible for executive dysfunction on the Wisconsin Card Sorting Test (WCST). 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, components related to shifting attention between different features of an object and to inhibiting responses failed to demonstrate impairments. This finding led 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 had contributions from multiple component processes that could be separated with appropriate procedures. It also demonstrated that the impaired performance of different neuropsychiatric populations could be traced to dysfunction on 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 [see Travis and Sigman, 1998]. 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 and 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 subserve 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 in autism of deficits in the use of eye contact to achieve joint attention [Sigman, 1996], another newly characterized cognitive or neurologic function contributing to social behavior. These two newly characterized 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. Such deficits have been proposed repeatedly to attempt to explain the intense focus of individuals with autism on details, on the one hand, and their lack of interest in people on the other. Although the recent research summarized above has provided evidence for a conceptual basis for these behavioral abnormalities, deficits in attention continue to be reported and proposed as a primary cognitive basis for behavior in autism. Deficits in selective attention, attention to extrapersonal space, and shifting attention [Courchesne et al., 1993; Ornitz, 1988; Townsend and Courchesne, 1994] have been among those recently proposed. 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. That paradigm, however, 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 make it impossible to determine the cognitive origin for the impaired performance in autism without investigating these contributions individually. A number of studies thereafter attempted to clarify the role of attentional processes in autism. Collectively, these studies examined the reflexive and voluntary or executive control of attention in autistic individuals of varying levels of ability [reviewed in Burack et al., 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 for highlighting the influence of developmental level or general ability level on the expression of deficits in autism, and the limitations of basing conclusions about core deficits without considering 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, thus posing a major question for the validity of single primary deficit models. As most cognitive and neuropsychologic studies in autism had focused on a single cognitive domain, their designs 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 participant sample provided an obvious opportunity for addressing the issue of single versus multiple coexisting 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.

Rumsey and Hamburger [1988] conducted one of the first studies to investigate neuropsychologic functioning across domains. Participants were individuals with autism that had been screened to exclude those with other causes of brain damage. This study was viewed as remarkable because it demonstrated 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 visuospatial abilities were described as ''intact.'' Examination of the test battery revealed only a few tests in the language, memory, and motor areas, which produced mixed results. By contrast, the reasoning domain had included a larger number and broader range of tests. Notably, 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 evidence for executive dysfunction but again relying on few tests in the language and memory domains, which produced the same type of 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 [Minshew et al., 1992]. This study of 15 nonmentally retarded autistic individuals 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 abilities and complex abilities. The second major finding of this study was the absence of impairments on the 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. Abstraction deficits were instead demonstrated with a test of verbal reasoning and in the capacity to shift concepts on the Goldstein-Scheerer Object Sorting Test. The absence of deficits on the WCST in this study was attributed to the higher level of function of the autistic participants compared to prior studies. In these individuals, 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 the Trail Making Test Part A, but not on Part B. 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 on Part B and impairments on Part A in these individuals suggested psychomotor slowing. Review

**Table 1. Discriminant Analysis Results By Domain and By Order of Entry**

| Domain | Tests Failing Tolerance Test | Tests Passing Tolerance Test | % Correct | % Jackknife | Kappa[1] |
|---|---|---|---|---|---|
| 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[1] |
| 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[1] |
| Complex Language | WRMT-R Passage Comprehension; TLC-Metaphoric Expression | K-TEA Reading Comprehension; Verbal Absurdities; Token Test | 72.7 | 65.2 | .45[1] |
| 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[1] |
| Reasoning | Category Test; Wisconsin Card Sort Test | 20 Questions; Picture Absurdities; Trail Making B | 75.8 | 72.7 | .52[1] |
| Visual-Spatial | WAIS-R Picture Completion, Object Assembly | WAIS-R Block Design | 56.1 | 56.1 | .12 |

[1] Indicates a significant between group difference in performance.

of the Rumsey and Hamburger [1988] Trail Making Test data revealed that their participants had also 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 Minshew and colleagues' [1992] study were amplified in follow-up studies. Concept formation ability was investigated further with the Twenty Questions Procedure, which requires participants 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 so as to eliminate several alternatives at once and, thus, to 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 controls. Participants with autism 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. Minshew and colleagues [1992] mixed pattern of results in the language and memory area led to an in-depth examination of the language domain investigating the hypothesis of a dissociation between preserved simple abilities and impaired complex abilities [Minshew et al., 1994, 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 participants with autism was compared to that of normal community volunteers matched on age, gender, race, IQ, and SES. Participants with autism did as well and often better than controls on the tests of mechanical language skills, but significantly poorer on tests of complex interpretive skills. These studies provided additional evidence suggesting that autistic individuals 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 provide further characterization of the pattern within and across domains in a large group of rigorously defined participants with autism and individually matched controls. An expanded test battery was designed to address the neuropsychologic deficits hypothesized by various neurobehavioral models for autism, as well as our own 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 visuospatial abilities (see Table 1). Although visuospatial abilities have long been considered a strength of individuals with autism, the visuospatial domain was included because its status was important in completing the profile of cognitive functioning. A range of abilities was considered within each domain to address the various hypothesized deficits. 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. 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 as to tests with the most discriminatory power. Individual *t*-tests were computed to clarify performance on tests not included in the regression equations (Table 2).

Tests in the attention, sensory perception, simple memory, and visuospatial domains did not yield satisfactory classification accuracy, providing evidence of intact basic information acquisition abilities and intact information processing in the visuospatial domain. Kappa scores in the fair-to-good agreement range (.40–.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 individuals with autism relative to controls, 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 (Table 1). 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 participant groups and the absence of a predilection for any quadrant. Thus, there was no support for a hypothesized deficit in attention to extrapersonal space. In the motor domain, discriminatory accuracy was achieved with the Grooved Pegboard Test and Trail Making Test, Part A, the two tests of skilled motor sequences. By contrast, there was no difference on the test of simple or isolated motor movements (Finger Tapping Test). *T*-tests revealed significantly poorer performance on Trails A but not Trails B, 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; this is consistent with our previously reported findings in nonmentally retarded individuals with autism [Minshew

**Table 2. Psychometric Data Used for Discriminant Analysis**

| Tests Entered into Prediction Equations | Autistic Group M | Autistic Group SD | Control Group M | Control Group SD | p |
|---|---|---|---|---|---|
| Attention Domain | | | | | |
| 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 |
| Sensory Perception Domain | | | | | |
| 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* |
| Motor Domain | | | | | |
| 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* |
| Simple Language Domain | | | | | |
| 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 |
| Complex Language Domain | | | | | |
| 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* |
| Simple Memory Domain | | | | | |
| Mazel 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 |
| Complex Memory Domain | | | | | |
| 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* |
| Reasoning Domain | | | | | |
| 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* |
| *20 Questions (% constraint seeking) | 35.49 | 23.82 | 56.08 | 14.02 | .000* |
| Visual-Spatial Domain | | | | | |
| 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 |

et al., 1992]. The first test passing the tolerance test for the reasoning domain was the Twenty Questions procedure, 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 proposed by Rumsey and Hamburger [1988]. Executive dysfunction or cognitive inflexibility might be too narrow to encompass the deficit.

Thus, the profile of cognitive functioning in these nonmentally retarded autistic adolescents and adults was 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 visuospatial areas. There are several implications of these findings.

The characterization of the cognitive profile in terms of both deficits and intact abilities is significant. 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 visuospatial abilities distinguishes autism from the developmental specific learning disabilities and the Nonverbal Learning Disability syndrome. Intact language, memory, arithmetic, rule-learning, and visuospatial abilities account for the attainment of IQ scores in the average range. Deficits in problem solving, concept formation, complex language, and complex memory abilities account for 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 accounts for the clinical observation that abstraction, communication, and social abilities fall rapidly (i.e., disproportionately) with declining IQ in the autistic population as compared to the nonautistic mentally retarded population. It is also consistent with the lower adaptive function of mentally retarded autistic individuals compared to nonautistic mentally retarded individuals of the same general level of ability.

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

Another unique feature of cognitive functioning in the participants with autism was the pattern of intact simpler abilities in domains demonstrating deficits. Contrary perhaps 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 the individuals 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 and, thus, dependence on the most cognitively advanced abilities. Intact function appeared to correspond to the simplest or most basic skills. This pattern conformed to the electrophysiologic pattern reported for autism—impaired late, cognitive potentials and intact earlier potentials [Minshew, 1991; Minshew et al., 1997]. 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 basic 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 [Tallal and Piercy, 1973; Tallal et al., 1996; Johnston et al., 1981; Neville et al., 1993; Jernigan et al., 1991]. This comparison revealed that the cognitive profile in autism was the converse of that described by Tallal and colleagues for children with developmental specific language impairment (SLI). As in autism, the neuropsychologic profile in SLI children involved multiple domains, but including the attention, sensory perception, motor, memory, and language domains. Unlike autism, the deficits in SLI 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 SLI children could sometimes use these abilities to fill in or infer missing information.

Evidence of a deficit in complex abilities in the motor domain in autism also supports the neurobiologic validity of a complex information processing construct. That is, the presence of a dissociation between simple and complex abilities in an area of minor clinical involvement would 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 increase, 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 our 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, visuospatial 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 visuospatial domain. Since the visuospatial 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 autistic adolescents and adults and, 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 autistic individuals. Thus, 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, the Autism Diagnostic Interview [LeCouteur et al., 1989; Lord et al., 1994] and the Autism Diagnostic Observation Schedule [Lord et al., 1989]. Nonetheless, the deficits in these areas can be conceptualized within a complex information processing model. For example, theory of mind skills are 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. Conversely, deficits were found in complex memory skills that are not obviously related to the clinical criteria for autism. Because data supporting their presence is clear, 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 [Minshew et al., 1996]. Thus, these autistic participants have difficulty remembering increasing amounts of information and with 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 Tooby 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, we would propose that the memory system will be demonstrated to interact 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.

In summary, this study of neuropsychologic functioning in autism provided evidence of the coexistence 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 particular aspects of behavior in autism. ■

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