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Can Standard Measures Identify Subclinical Markers of Autism?[1]

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This study compared the executive function and theory-of-mind abilities of siblings of autistic individuals to those of siblings of learning-disabled controls. Three different analyses of the dependent measures provided convergent support for a potential subclinical marker in the executive function domain. No group differences in theory-of-mind abilities were found. However, power analyses revealed that the measures employed in this study, which are typically used with autistic individuals, were not sufficiently sensitive to detect any group differences that might exist in "unaffected" family members. Suggestions for future research are provided, including the need to develop more sensitive tasks that produce larger effects and measure more elementary cognitive operations.

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INTRODUCTION

There is growing evidence that genetic factors play some role in the etiology of autism. While the familial recurrence risk is relatively small, it is 50 to 100 times the rate predicted by the population prevalence of the disorder (Folstein & Rutter, 1988). Although familial aggregation by itself does not indicate genetic etiology, a significantly higher concordance rate for autism in monozygotic (MZ) than dizygotic (DZ) twins has provided additional support for the role of genetics in autism (Folstein & Rutter, 1977; Smalley, Asarnow, & Spence, 1988).

Exactly what is inherited is not yet clear, however. Several studies have suggested that a genotype with variable expressivity may produce autism in one family member and more broadly defined cognitive, social, and behavioral impairments in other family members (Folstein & Rutter, 1977, 1988). Recent research has focused on identifying *subclinical markers* of autism; these have been defined as "biological and behavioral measures that are presumably more proximal to the underlying gene (or genes) involved in autism than the clinical syndrome" (Smalley & Asarnow, 1990, p. 271). As defined by Smalley and Asarnow, potential subclinical markers must satisfy two criteria: They must detect differences between autistic and nonautistic subjects and they must also be present in a higher proportion of relatives of autistic people than relatives of nonautistic individuals.

Efforts to explore familial factors in the transmission of the disorder must be sensitive to the early practice of "parent blame" in the field of autism, lest they unwittingly reopen old debates regarding causation (Jensen, 1991). Nevertheless, the study of subclinical manifestations of autism in family members is essential for a more precise specification of the processing deficits involved in the disorder, better understanding of their neurobiological origins, refinement of treatment techniques, and early identification of affected children. Thus, while great care must be taken to avoid the implication that parents are to blame for their child's difficulties, exploration of genetic factors in the causation of autism is vitally important to future growth of the field.

Initial investigations of subclinical markers focused on cognitive variables, with mixed success. Studies found that the rate of mental retardation, learning disabilities, and language disorders was elevated in siblings of autistic individuals (August, Stewart, & Tsai, 1981; Minton, Campbell, Green, Jennings, & Samit, 1982). It was later suggested that cognitive deficits were markers of mental retardation, independent of autism, since they appeared to cluster in relatives of retarded, but not normal IQ, autistic

probands (Baird & August, 1985). However, a study by Freeman et al. (1989) failed to confirm either pattern of results.

More recently, studies have turned to investigation of social and emotional variables. Wolff, Narayan, and Moyes (1988) found that parents of autistic children demonstrated significantly less empathy, rapport, social openness, and smiling during a semistructured interview than parents of children with other handicaps. Smalley and Asarnow (1990) found that siblings of autistic individuals performed poorly on an emotion discrimination task, in comparison with their performance on a visual perception measure, while control siblings did not show this pattern. Finally, Landa, Folstein, and Isaacs (1991) found that parents of autistic children produced significantly poorer spontaneous narratives than control parents. These results are consistent with the hypothesis that something is transmitted in the families of autistic children that is broader and milder than autism per se. This is a critical point, as further investigation of subclinical markers would not be indicated if the inheritance of autism appeared to be an all-or-none phenomenon.

Recently there has been great interest in identifying underlying processing mechanisms that might account for a wide range of autistic symptoms. Two promising candidates for so-called "primary deficits" of autism have received attention in the literature: executive function and theory of mind. Several studies have found striking executive function deficits (e.g., in planning, working memory, and cognitive flexibility) in autistic individuals (McEvoy, Rogers, & Pennington, 1993; Prior & Hoffmann, 1990; Rumsey & Hamburger, 1988, 1990). Ozonoff, Pennington, and Rogers (1991) found that executive function variables were best able to discriminate between autistic individuals and controls. This body of research has led to the suggestion that executive function deficits may be primary to autism (Harris, 1993; Hughes & Russell, 1993; Ozonoff, in press; Pennington, in press). Other investigations have found that autistic children are severely impaired, relative to controls, in predicting the mental states of others, leading to an alternate suggestion that theory-of-mind deficits are central to the disorder (Baron-Cohen, 1989; Baron-Cohen, Leslie, & Frith, 1985, 1986; Perner, Frith, Leslie, & Leekam, 1989).

Since we might expect that primary deficits of autism are also the ones that are heritable, a next logical step in the search for potential subclinical markers is to examine executive function and theory-of-mind abilities in family members. This was the goal of the present investigation. To our knowledge, it is the first study to examine these domains in relatives of autistic probands.

METHOD

Subjects

Probands

Eighteen high-functioning autistic subjects were matched with 18 learning-disabled (LD) controls on the basis of Full-scale IQ (FSIQ), gender, socio-economic status (SES), and ethnic background; these two proband groups participated in a previous research investigation (Ozonoff et al., 1991).

All autistic probands met DSM-III-R criteria for either Autistic Disorder (n = 14) or Pervasive Developmental Disorder Not Otherwise Specified (n = 4) and received scores above 27 on the Childhood Autism Rating Scale (CARS; Garfin, McCallon, & Cox, 1988; Schopler, Reichler, & Renner, 1988). Autistic probands were recruited through the Autism Society of Colorado and local clinicians; recruitment was done without knowledge of the presence of deficits in the proband's family. Two selection criteria were used: First, the proband had to have intellectual abilities in the nonretarded range of functioning (e.g., FSIQ > 70), and second, the proband had to have at least one sibling between the ages of 8 and 18. By including only nonretarded probands, we could be relatively certain that any deficits found among their siblings were independent of the familiality of mental retardation.

Diagnoses of LD probands included attention deficit hyperactivity disorder, dyslexia, expressive language disorder, and other learning disabilities. All LD probands received scores below 20 on the CARS, indicating that they did not manifest autistic symptoms. See Table I for descriptive characteristics of both proband groups.

Siblings

One sibling (between the ages of 8 and 18 years) of each proband was recruited to participate in the present study. No autistic probands had more than one sibling in the appropriate age range, but 5 LD probands did. In these cases, a sibling was randomly chosen for participation, without knowledge of that individual's characteristics or functioning level.

One sibling of an autistic proband had been previously diagnosed as autistic; all other siblings in both groups were apparently developing normally and had not been previously identified. The two groups of siblings did not differ in age (autistic siblings: M = 11.8 years, SD = 4.1; control siblings: M = 12.5 years, SD = 3.5) or gender (M:F ratio: autistic siblings = 10:8, control siblings = 12:6).

Table I. Descriptive Characteristics of the Proband Sample

	Autistic probands $n = 18$	LD probands $n = 18$
Mean full scale IQ (*SD*)	89.8 (13.5)	95.0 (15.9)
Mean CARS score (*SD*)	34.8 (4.6)	18.3 (2.9)[a]
Mean socioeconomic status (*SD*)	45.6 (10.4)	44.3 (13.2)
Gender (male:female)	16:2	16:2
Race (white:non-white)	15:3	16:2

[a] $p < .0001$.

Measures

The following experimental battery was administered during one 2-hour testing session. The order of the measures was counterbalanced across subjects. Details of the tasks and their scoring can be found in the primary sources cited below.

Intellectual

The six subtests that load most highly on the Verbal Comprehension and Perceptual Organization factors of the WISC-R and WAIS-R (Information, Similarities, Vocabulary, Comprehension, Block Design, Object Assembly; Sattler, 1988) were used to prorate Verbal (VIQ), Performance (PIQ) and FSIQ.

Executive Function

Wisconsin Card Sorting Test (WCST; Heaton, 1981). This measure of cognitive flexibility requires subjects to sort cards by color, shape, and number. Unbeknownst to the subject, the sorting rule changes after 10 consecutive cards have been correctly sorted; the subject's sorting strategy must be modified accordingly. The dependent variable is the average number of trials taken to complete a category. This summary score is inflated by errors, failures to maintain set, and perseverative responses (e.g., when the subject continues sorting by a previously correct category despite negative feedback).

Tower of Hanoi (Borys, Spitz, & Dorans, 1982). This ring-transfer task requires subjects to plan a sequence of moves that transforms an initial configuration of rings into a "tower," in which the rings are arranged by size on a designated peg. The dependent variable is a planning efficiency score derived from the number of trials required to complete the problem correctly (see Borys et al., 1982, for more detail on the scoring procedure). Both the 3-ring (TOH3) and 4-ring (TOH4) versions of the task were administered to all subjects.

Theory of Mind

Second-Order Belief Attribution Task. This task was administered and scored as described by Baron-Cohen (1989). Subjects watch while a story is acted out with toys; in the story, two children, John and Mary, play in a park. At the end of the story, subjects are asked to predict Mary's belief about John's whereabouts (the Belief Question) and then explain why Mary holds this belief (the Justification Question). The Belief Question was scored in a pass/fail manner. The Justification Question was scored according to the number of mental state attributions made by the subject: 0 (e.g., no mental state attributions were made), 1 (e.g., mental states were attributed to only one character) and 2 (e.g., the subject accounted for the mental states of both characters).

Fox and Grapes Task (Flavell, Botkin, Fry, Wright, & Jarvis, 1968). In this task, the subject reads a familiar fable and then modifies it so that it can be understood by a young child. The dependent variable is the number of simplifying recodings the subjects makes, including (a) substitutions, in which an expression in the text is replaced by a simpler one that is more easily comprehended by a young child (e.g., "he said" for "quoth he"); (b) additions, in which something is added to clarify or supplement the text (e.g., "to get the tempting morsel, *which is the grapes*"); and (c) deletions, in which something is removed that is considered confusing or inessential. These categories were coded as described by Flavell et al. (1968).

Apple–Dog Task (Flavell et al., 1968). In this measure, the subject is shown 7 pictures and asked to narrate the story it illustrates. Three pictures are then removed and the subject is asked to tell the story from the point of view of "Mrs. Smith," who has just entered the room and seen only the 4 remaining pictures. The subject must suppress his own knowledge and tell the story from the other person's perspective. A score of 1 is given if the subject correctly presents "Mrs. Smith's" point of view, using the 4-picture sequence. A score of 4 is given if the subject tells the story from his own (7-picture) perspective. Intermediate scores of 2 and 3 are given for varying levels of perspective-taking, as described by Flavell et al. (1968).

RESULTS

Preliminary Analyses

Performance on the TOH3 was at ceiling, with the maximum score possible on the test only 1 standard deviation from the group means (autistic siblings: $M = 31.2$, $SD = 4.2$; control siblings: $M = 32.4$, $SD = 4.0$). Previous studies have demonstrated that TOH3 performance reaches adult levels by age 12 (Welsh, Pennington, & Groisser, 1991). Because of ceiling effects and developmental inappropriateness for most subjects, this measure was excluded from further analyses.

Group Differences

Independent sample t tests and chi-square tests were used to examine group differences in performance on the experimental measures. As can be seen in Table II, control siblings performed significantly better than autistic siblings on the 4-ring version of the Tower of Hanoi. A nonsignificant trend on the WCST ($p < .15$) suggested that autistic siblings tended to take more trials to complete sorting categories than control siblings. In the theory-of-mind domain, however, no group differences were evident. Finally, there were no statistically significant differences between the sibling groups on VIQ, PIQ, or FSIQ, replicating previous studies (Baird & August, 1985; Freeman et al., 1989).

Discriminant Analysis

A discriminant function analysis was performed to evaluate how well the sibling groups could be empirically distinguished from each other on the basis of test performance. The TOH4 was most highly correlated with the function. When this variable alone was used in the analysis, an overall classification accuracy rate of 75% was achieved (Wilks's lambda = .86, $p = .02$); when other variables were entered, prediction accuracy decreased. Thus, the TOH4, by itself, was a relatively powerful discriminator between the groups.

Distribution Analyses

To examine whether a subset of autistic siblings demonstrated deficits on the experimental measures, the number of subjects performing 1.5 standard deviations below the control group mean was calculated for each

Table II. Group Performances on Experimental Measures

	Autistic siblings (n = 18)		LD siblings (n = 18)		
	M	*SD*	*M*	*SD*	p^a
Verbal IQ	104.8	17.1	104.8	8.0	.99
Performance IQ	108.9	12.2	107.6	14.0	.75
Full-scale IQ	107.4	14.8	106.2	10.9	.78
TOH4	4.7	3.7	8.0	4.5	.02
WCST	29.3	29.1	18.5	7.5	.14
2nd-order belief	0.72	0.46[b]	0.78	0.43[c]	.70[f]
2nd-order justification	1.4	0.70[d]	1.4	0.62[e]	.82[f]
Fox-and-grapes	4.3	2.8	4.2	3.5	.98
Apple–dog	1.6	1.2	1.4	0.9	.62

[a] Independent sample t tests (df = 34) except where noted.
[b] Pass:fail = 13:5.
[c] Pass:fail = 14:4.
[d] Belief attributions: 2nd order:1st order:0 order = 9:7:2.
[e] Belief attributions: 2nd order:1st order:0 order = 9:8:1.
[f] Data analyzed with chi-square test.

of the 7 continuous variables used in the study. As can be seen in Table III, more siblings of autistic probands than siblings of LD probands fell in this range on most measures. A chi-square test of association revealed that the difference in proportions across the 7 measures was statistically significant, $\chi^2(1) = 3.84$, $p = .05$. Further examination of the data revealed that the same 3 autistic siblings performed most poorly on all variables, with 3 other autistic siblings performing poorly on selected measures.

Power Analyses

For group differences to be evident, the measures used must have sufficient power. Especially in the study of familial deficits, it is important that we use sensitive tests that can identify subtle differences in "unaffected" family members. This study employed measures that are widely used at the present time. However, since this is the first study to investigate these domains in the families of autistic individuals, it was not clear if these tasks would be sensitive enough to detect any differences that might be present. Therefore, *post hoc* power analyses were conducted to examine whether these measures are appropriate for research in which large samples are prohibitive.

Table III. Subjects Falling 1.5 Standard Deviations Below the Control Group Mean

	Autistic siblings (n = 18)	LD siblings (n = 18)
Verbal IQ	6	1
Performance IQ	1	0
Full-scale IQ	1	1
TOH4	5	3
WCST	4	1
Fox-and-grapes	0	0
Apple–dog	3	4
Total (proportion)	20 (.159)	10 (.079)[a]

[a] $\chi^2 = 3.84$, $p = .05$.

On the measure that demonstrated the largest group difference, the TOH4, an effect size of .79 was calculated (Kraemer & Thiemann, 1987). Assuming this effect size and 80% power, approximately 28 subjects per group are required to reliably demonstrate group differences on a two-tailed t test at the .05 level of significance. At $p = .01$, approximately 46 subjects per group are necessary. The effect sizes of the theory-of-mind measures were much smaller, requiring substantially larger samples to reliably find group differences. Thus, given the logistical difficulty of large-sample research on autism, the TOH4 is the only measure even marginally powerful enough to use with unaffected family members.

DISCUSSION

This study sought to identify potential subclinical markers of autism by examining the familiality of executive function and theory-of-mind deficits in siblings of autistic children. Three different analyses of the dependent measures provide convergent support for a potential subclinical marker in the executive function domain. First, a statistically significant group difference was found on one executive function variable, the Tower of Hanoi 4-ring problem, and a nonsignificant trend in the same direction was demonstrated on the WCST. Second, a discriminant function analysis found that the TOH4, by itself, correctly classified 75% of subjects into

groups. In a previous study of the affected probands of these siblings, the Tower of Hanoi was also the most powerful diagnostic discriminator (Ozonoff et al., 1991), correctly classifying 80% of probands. Finally, the scores of a subset of autistic siblings were significantly depressed relative to control siblings on several measures, including the TOH4 and WCST.[3] Thus, it appears that there is something transmitted in autistic families, perhaps falling in the executive function domain, that is not transmitted in control families. Since this is a family study, however, it is not possible to identify the basis of this transmission as genetic, environmental, or transactional.

No significant group differences were found on the theory-of-mind variables, nor were these variables good discriminators of the groups. One explanation is that impairment in this domain is not a likely subclinical marker of autism. However, another possibility exists.

Power analyses reveal that these measures, which are typically used with autistic individuals, are not sufficiently sensitive to detect group differences in family members, with this particular sample size. Even in the executive function domain, which was relatively more powerful in detecting group differences, unrealistically large samples would be required to reliably demonstrate such differences. What is needed are more powerful discriminating tasks that produce larger effects.

In addition, it is necessary to develop measures that tap more elementary cognitive operations. Traditional executive function and theory-of-mind tasks, such as those used in the present study, are complex and require several cognitive processes for successful completion. For example, performance on the Tower of Hanoi demands a variety of intact functions, from planning and temporal ordering of potential moves, to spatial and visual imagery skills, to holding a large amount of information in working memory. Deficits in any of these operations could account for the group differences seen on this task. Measures that tap unitary, specific cognitive processes may yield not only larger effects and better group discrimination but also a more precise understanding of the mechanisms underlying autism.

[3]While the distribution analyses may provide support for a potential subclinical marker in the executive function domain, it is also possible that the differences found in the subset of autistic siblings were driven by the low VIQ of several subjects (see Table III). While there were no group differences in VIQ, PIQ, or FSIQ in the overall sample (n = 36), the VIQ of the "affected" subset of autistic siblings was lower than that of the LD siblings. If VIQ is important to performance on the TOH or WCST, then the deficits seen in this subset of subjects may not represent subclinical markers in the executive function domain but may instead reflect lower intellectual capacity. Thus, the results of the distribution analyses must be interpreted with some caution.

Information-processing paradigms developed within cognitive psychology may be applicable to this type of research. These paradigms focus on simple cognitive operations, such as target detection or response inhibition, and provide continuous variables (e.g., reaction time) which are inherently more powerful than restricted range accuracy data (Kraemer & Thiemann, 1987). Thus, information-processing measures may be capable of uncovering group differences obscured by standard neuropsychological and social–cognitive measures.

Courchesne et al. (in press) have recently used measures of attention switching with autistic individuals. Huge effect sizes were found, with more than 6 standard deviations separating the performance of the autistic group from that of the control group. Although these measures have not yet been used with family members, it may be this type of task that proves most fruitful in our continued search for subclinical markers of autism. Clearly, a priority for future research is the development of more sensitive, dynamic measures for use with nonautistic family members. The present investigation suggests that executive function may be a promising domain worthy of further exploration.

Finally, while research on siblings is an acceptable method of screening for familial deficits and subclinical markers, it is not a very powerful design for definitively testing a genetic hypothesis. Under most models of genetic transmission, fewer than 50% of siblings would be affected; in addition, the affected subset would have to display such pronounced deficits that their poor performance alone would produce group differences. Consequently, twin studies that compare MZ-DZ concordance rates of a broader phenotype, possibly including executive function deficits, are another important step in our quest to identify subclinical markers of autism.

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