

## Neurocognitive Function and Joint Attention Ability in Young Children with Autism Spectrum Disorder Versus Developmental Delay

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Studies have shown that young children with autism are not impaired on prefrontal tasks relative to what would be expected for their mental age, raising questions about the executive dysfunction hypothesis of autism. These studies did not include ventromedial prefrontal tasks, however. The present study examined whether young children with autism spectrum disorder (ASD) are impaired on ventromedial prefrontal tasks, and whether performance on such tasks is correlated with a core autism symptom, joint attention ability. Seventy-two 3- to 4-year-old children with ASD, 34 3- to 4-year-old developmentally delayed children, and 39 12- to 46-month-old typically developing children, matched on mental age, were administered ventromedial and dorsolateral prefrontal tasks and joint attention tasks. Children with ASD performed similarly to comparison groups on all executive function tasks, indicating that at this early age, there is no autism-specific pattern of executive dysfunction. Ventromedial, but not dorsolateral, prefrontal task performance was strongly correlated with joint attention ability, however. The ventromedial prefrontal cortex is hypothesized to play a role in the development of joint attention and possibly some aspects of the autistic syndrome.

### INTRODUCTION

Autism is a developmental disorder characterized by qualitative impairments in social interaction and communication and a restricted range of activities and interests. Evidence for a biological etiology for autism is well established (Bailey, Phillips, & Rutter, 1996). Although it has been over 5 decades since autism was recognized as a clinical syndrome (Kanner, 1943), the precise nature of brain dysfunction underlying this disorder remains to be elucidated. It is well documented that individuals with autism have impairments in processing of social and emotional information, as evident in tasks assessing face and emotion recognition, imitation of body movements, interpretation and use of gestures, and theory of mind (Baron-Cohen, Tager-Flusberg, & Cohen, 1994; Davies, Bishop, Manstead, & Tantam, 1994; Dawson, Meltzoff, Osterling, & Rinaldi, 1998; Hobson, Ouston, & Lee, 1988a, 1988b; Mundy, Sigman, Ungerer, & Sherman, 1986; Smith & Bryson, 1994; Teunisse & DeGelder, 1994). Even on simple attention tasks, such as orienting to auditory stimuli, children with autism show more severe impairments when the stimuli are social in nature (Dawson, Meltzoff, Osterling, Rinaldi, & Brown, 1998). This pattern of behavioral impairments suggests that autism is related to dysfunction of a brain system involved in social cognition. Animal and human lesion studies indicate that parts of the medial temporal lobe (amygdala, hippocampus, and entorhinal cortex) and the ventromedial prefrontal cortex are likely to comprise a brain system specialized for social processing (Barbas, 1995; Brothers, 1990; Dama-

sio, 1994; LeDoux, 1994). Evidence for involvement of the medial temporal lobe in autism includes neuropsychological, neuroimaging, animal lesion, and autopsy studies (Bachevalier, 1994; Baron-Cohen et al., 1999, 2000; Barth, Fein, & Waterhouse, 1995; Bauman & Kemper, 1994; Dawson, Meltzoff, Osterling, & Rinaldi, 1998; Howard et al., 2000). Evidence for involvement of the ventromedial prefrontal cortex in autism is less direct and is primarily based on findings of deficiencies in social cognition and theory of mind in patients with ventromedial prefrontal damage (Cicerone & Tanenbaum, 1997; Damasio, Tranel, & Damasio, 1990; Stone, Baron-Cohen, & Knight, 1998).

In earlier articles (Dawson, 1996; Dawson, Meltzoff, Osterling, & Rinaldi, 1998), Dawson and colleagues proposed, similar to other investigators (Bachevalier, 1994; Baron-Cohen & Ring, 1994), that autism involves dysfunction of parts of the medial temporal lobe (MTL; amygdala, hippocampus, and entorhinal cortex) and the ventromedial cortex. Dawson and colleagues tested this hypothesis by administering neuropsychological tasks to children with autism and examining their performance in relation to severity of autism symptoms (Dawson, Meltzoff, Osterling, & Rinaldi 1998). It was found that, compared with developmentally matched children with Down syndrome and those with typical development, children with autism were impaired on the delayed nonmatching to sample task (DNMS). In studies of nonhuman primates and human adults, lesions to hippocampus

and ventromedial prefrontal cortex—specifically, the orbital prefrontal region—have been shown to impair performance on the DNMS while failing to impair performance on other memory tasks, such as the delayed response task (Bachevalier & Mishkin, 1986; Diamond & Goldman-Rakic, 1989; Kowalska, Bachevalier, & Mishkin, 1991; Meunier, Bachevalier, & Mishkin, 1997; Zola-Morgan & Squire 1993; Zola-Morgan, Squire, & Amaral, 1989). In the Dawson, Meltzoff, Osterling, and Rinaldi (1998) study, it was also found that children with autism were impaired on the delayed response task (similar to the A not B task in infants). In studies of nonhuman primates, it has been shown that lesions to the dorsolateral prefrontal cortex result in impaired performance on the delayed response task, and that lesions of the MTL do not affect performance on this task (Diamond & Goldman-Rakic, 1986, 1989; Goldman, Rosvold, & Mishkin, 1970). Furthermore, it was found that performance on the DNMS task was strongly correlated with severity of autism symptoms, whereas there was little or no correlation between performance on the delayed response task and autism symptom severity.

In the present study, the hypothesis that autism is related to dysfunction of the MTL–ventromedial prefrontal circuit was tested by examining the relation between patterns of neuropsychological functioning and severity of a core autism symptom (joint attention) in a relatively large group of young children with autism spectrum disorder (ASD). The present study extended research conducted by Dawson, Meltzoff, Osterling, and Rinaldi (1998) in three ways. First, whereas the children with autism in the Dawson et al. study were, on average, 5.5 years of age, the children in the present study were 3 to 4 years. By studying younger children, the intention was to minimize compensatory factors that may influence performance as children mature and receive treatment.

Second, the number of neuropsychological tasks was expanded to better assess whether children with ASD show differential performance on tasks that tap the dorsolateral prefrontal cortex versus the MTL–ventromedial prefrontal circuit. Previous studies that assessed executive function in young children with autism primarily used tasks that tap the dorsolateral prefrontal cortex. For example, Griffith, Pennington, Wehner, and Rogers (1999) administered a large battery of executive function tasks to a comparably young sample of children with autism to test the hypothesis that autism is related to impairments of the prefrontal cortex. This hypothesis was originally proposed by Damasio and Maurer (1978) and later expanded on by Rogers and Pennington (1991). Several studies of adults, adolescents, and older children

with autism (Bennetto, Pennington, & Rogers, 1996; Hughes & Russell, 1993; Ozonoff, 1995; Ozonoff, Pennington, & Rogers, 1991; Prior & Hoffman, 1990) have demonstrated executive function impairments in autism. Griffith et al. found that preschool-age children with autism were no more impaired on executive function tasks than children with mental retardation. These authors (p. 828) concluded “these results raise serious questions about the executive dysfunction hypothesis of autism.” This conclusion may be premature, however, because tasks assessing ventromedial prefrontal function were not included in their battery. Given that there is increasing evidence for MTL–ventromedial prefrontal dysfunction in autism, a more thorough assessment of ventromedial prefrontal function in autism is warranted.

The third way the present research extended the Dawson, Meltzoff, Osterling, and Rinaldi (1998) study was its use of multiple measures of core constructs to allow for a better test of the hypothesis that severity of autism symptoms is correlated with degree of dysfunction of the MTL–ventromedial prefrontal circuit. Dawson, Meltzoff, Osterling, and Rinaldi (1998) found a strong relation between severity of autism symptoms and performance on the DNMS, a task that animal studies suggest is mediated by both MTL and ventromedial prefrontal cortex. In contrast, little or no relation was found between severity of autism symptoms and performance on the A not B task, which taps the dorsolateral prefrontal cortex. In the present study, the hypothesis that core autism symptoms are related to MTL–ventromedial prefrontal dysfunction was more carefully tested by administering multiple assessments of three domains—(1) dorsolateral prefrontal functioning, (2) MTL–ventromedial prefrontal functioning, and (3) joint attention ability—and examining the relations among these domains.

There are two reasons for focusing on joint attention and its relation to neurocognitive ability. First, impairments in joint attention in autism have been extremely well replicated, and such impairments are both specific and virtually universal in young children with autism (Filipek et al., 1999; Mundy, 1995). Joint attention impairments appear to be present by 1 year of age (Osterling and Dawson, 1994; Mundy et al., 1986) and are part of the diagnostic criteria for autism (DSM-IV; American Psychiatric Association, 1994). Joint attention is the ability to coordinate attention between interactive social partners with respect to objects or events, or to share an awareness of the objects or events (Mundy et al., 1986). Classic joint attention behaviors include responding to another’s gaze shifts and protodeclarative pointing.

Second, there has been interest in the role of execu-

tive function in the development of joint attention and theory of mind (Ozonoff et al., 1991; Pennington & Ozonoff, 1996; Rochat, 1999). One study of older high-functioning individuals with autism found a correlation between executive function ability and theory-of-mind skills (Ozonoff et al., 1991). Because joint attention ability has been hypothesized to be a precursor of theory-of-mind ability (Rochat, 1999), it was hoped that this study would shed light on the contribution of different aspects of executive function to early theory-of-mind development. One study of young children with autism found that joint attention ability was correlated with performance on a task that taps dorsolateral prefrontal functioning (McEvoy, Rogers, & Pennington, 1993). This correlation, however, was only partially replicated in a subsequent study of young children with autism (Griffith et al., 1999).

It was predicted that joint attention ability would be more closely correlated with performance on tasks that tap the MTL–ventromedial prefrontal circuit, than with performance on tasks that tap the dorsolateral prefrontal cortex. This prediction was based on the hypothesis that early emerging symptoms of autism, such as an impairment in joint attention, are related to core affective, motivational, and social impairments that can be linked to dysfunction of MTL and ventromedial prefrontal cortex (for more elaborate discussion of this hypothesis, see Baron-Cohen et al., 2000; Dawson, 1996). The animal and brain damage literatures suggest that the MTL, particularly the amygdala, is critical for social perception, such as recognition of faces and facial expressions (Aggleton, 1992; Jacobson, 1986); recognition of the affective significance of stimuli and stimulus-reward associations (LeDoux, 1987); perception of body movements and gaze direction (Brothers, Ring, & Kling, 1990); and for certain cognitive abilities that are likely to be important for social perception, such as cross-modal association (Murray & Mishkin, 1985). It is further hypothesized that such early dysfunction of the MTL has downstream consequences for the development of higher order functions, particularly those mediated by the ventromedial prefrontal cortex with which the amygdala has close functional and anatomical connections. In particular, the ventromedial prefrontal cortex has been shown to be important for establishing, generalizing, and inhibiting stimulus–reward associations, and thus plays an important role in “emotional learning”; that is, in assigning and flexibly applying and modifying social reward values (Rolls, 1990; Rolls, Hornak, Wade, & McGrath, 1994). Dawson and colleagues (Dawson, Carver, & McPartland, 2000a; 2000b; Dawson, Osterling, Rinaldi, Carver, &

McPartland, 2001) have argued that impairment in assigning and flexibly modifying social reward values may be a core feature of autism.

The present study assessed three groups of young children who were developmentally matched on mental age: Children with ASD, children with developmental delay (DD) without autism, and children with typical development. Three tasks that tap dorsolateral prefrontal cortex (A not B, A not B with invisible displacement, and spatial reversal), three tasks that tap ventromedial prefrontal cortex (two versions of delayed nonmatching to sample with brief delay, and object discrimination reversal), and three tasks that assess joint attention ability were administered. Delayed nonmatching to sample assesses rule-learning ability (specifically, the ability to abstract the quality of novelty and associate it with reward) and visual recognition memory (Diamond, Churchland, Cruess, & Kirkham, 1999). It has been linked to the amygdala and hippocampus and ventromedial prefrontal cortex, including the entorhinal cortex and orbital prefrontal cortex, in lesion studies with monkeys (Bachevalier & Mishkin, 1986; Kowalska et al., 1991; Meunier et al., 1997; Zola-Morgan & Squire, 1993; Zola-Morgan et al., 1989) and in human amnesic patients (Squire, Zola-Morgan, & Chen, 1988). In the present study, only a brief delay (5 s) was used in the DNMS, thus minimizing the memory aspects of the task. Thus, the child’s score best reflected the rule-learning aspect of performance (i.e., novelty is associated with reward). Animal studies have suggested that the rule-learning aspect of the DNMS is linked to the ventromedial prefrontal cortex (Meunier et al., 1997). The object discrimination reversal (ODR) task assesses children’s ability to modify their behavioral response when a particular response is no longer rewarded. Performance on this task is severely impaired by early and late lesions to the orbitofrontal region in monkeys (Butter, 1969; Butters, Butter, Rosen, & Stein, 1973; Goldman-Rakic, Isseroff, Schwartz, & Bugbee, 1983; Jones & Mishkin, 1972; Mishkin, 1964), and by lesions of the ventromedial prefrontal region in human adults (Damasio, 1994; Damasio et al., 1990; Rolls, 1990; Rolls et al., 1994). In addition, Dias, Robbins, and Roberts (1996) showed that the object discrimination reversal task could clearly dissociate dysfunction of the ventromedial prefrontal cortex from dysfunction of the dorsolateral prefrontal cortex in monkeys.

The A not B task requires both working memory and response inhibition. It has been linked to the dorsolateral prefrontal cortex based on both human infant studies and animal lesion studies (Diamond & Goldman-Rakic, 1986, 1989; Goldman et al., 1970). Lesions to the medial temporal lobe and parietal cortex

in the adult animal do not disrupt performance on this task (Diamond & Goldman-Rakic, 1989; Diamond, Zola-Morgan, & Squire, 1989). The spatial reversal task also requires both working memory (maintaining a response set during a delay) and problem solving and concept formation (generalizing a rule to respond correctly; Espy, Kaufmann, McDiarmid, & Glisky, 1999).

Children in the three groups were compared in terms of their performance on both types of neurocognitive tasks and joint attention ability. Structural equation modeling (SEM) was used to assess the hypothesis that ventromedial prefrontal ability is more closely related to joint attention than dorsolateral prefrontal ability. In addition to providing information relevant to the nature of brain dysfunction in autism, it was hoped that the role of the prefrontal cortex in the development of early social cognition would be elucidated.

## METHOD

### Participants

Three groups of children participated in the study: (1) 72 children with ASD, which included 49 children with autism and 23 children with Pervasive Developmental Disorder, Not Otherwise Specified (PDDNOS); (2) 34 children with DD without autism; and (3) 39 children with typical development. Groups were matched on mental age based on composite age equivalence scores on the Mullen Scales of Early Learning (Mullen, 1997). The DD group included 31 children with idiopathic DD and 3 children with Down's syndrome. Participants were recruited from local parent advocacy groups, public schools, the Department of Developmental Disabilities, clinics, hospitals, and the University of Washington Infant and Child Subject Pool. Exclusionary criteria included the presence of a neurological disorder of known etiology (for ASD group only; e.g., fragile X), significant sensory or motor impairment, major physical abnormalities, history of serious head injury, and/or neurolog-

ical disease. Children with typical development were excluded if they exhibited unusually high or low cognitive ability ( $\pm 1$  SD) as assessed by their composite score on the Mullen Scales of Early Learning (Mullen, 1997). Table 1 presents demographic and descriptive information, including gender, ethnicity, socioeconomic status (SES) based on the Hollingshead Four Factor Index of Social Status (Hollingshead, 1976), chronological age, and Mullen composite mental age for the three groups of children. Groups did not differ in terms of Mullen composite mental age, SES, or ethnicity. Groups differed in terms of gender composition, with the DD group having a significantly greater percentage of females than the ASD and typical groups,  $\chi^2(2, N = 145) = 11.391, p < .01$ . Because the children with typical development were matched to the clinical groups on mental age, this group had significantly lower chronological age than both the ASD group,  $t = -10.867, p < .001$ , and the DD group,  $t = 10.482, p < .001$ .

Children with ASD were administered a diagnostic evaluation consisting of the Autism Diagnostic Interview-Revised (ADI-R; Lord, Rutter, & LeCouteur, 1994) and the Autism Diagnostic Observation Schedule-Generic (ADOS-G; Lord, Rutter, Goode, & Heemsbergen, 1989). Both instruments assess the symptoms of autism according to DSM-IV criteria (American Psychiatric Association, 1994). In addition, clinicians made a clinical judgment of diagnosis based on presence or absence of symptoms of autism as defined in the DSM-IV. Diagnosis of autism was defined as meeting criteria for autism on the ADOS-G, the ADI-R, and based on clinician judgment. In addition, if a child received a diagnosis of autism based on the ADOS-G and clinical diagnosis, and came within 2 points of meeting criteria on the ADI-R, the child was considered to have autism. Diagnosis of PDDNOS was defined as meeting criteria for PDDNOS on the ADOS-G, meeting criteria for autism on the ADI-R or missing criteria on the ADI-R by 2 or fewer points, and meeting criteria for PDDNOS based on clinical diagnosis. Children with DD were administered the ADOS-G. These children did not meet criteria for

**Table 1** Demographic Characteristics of Sample

Group	N, Male:Female	European American:Other (N)	SES M (SD)	Chronological Age (Months)			Composite Mental Age (Months)		
				Min.	Max.	M (SD)	Min.	Max.	M (SD)
Autistic spectrum disorder	72, 60:12	50:22	47.4 (11.5)	34	52	43.5 (4.3)	11.8	46.8	25.2 (8.8)
Developmental delay	34, 18:16	22:12	47.4 (14.0)	33	57	44.8 (5.3)	12.3	42.8	27.6 (8.2)
Typical development	39, 30:9	28:11	49.5 (12.1)	12	46	27.1 (8.9)	13.5	45.5	28.4 (9.1)

autism or PDDNOS on the ADOS-G or based on clinical diagnosis, nor did they show elevated symptoms on these measures.

### Procedure

Testing occurred over the course of five sessions. The child's parent was present for all testing. Children were given food snacks and praise as reward for sitting at the table when necessary, and were provided breaks as needed. All sessions were videotaped. On tasks in which children were required to search for rewards, food items or small toys served as reinforcers. Children also received praise for correct responses.

### Ventromedial Prefrontal Neuropsychological Tasks

*Delayed nonmatching to sample.* The DNMS procedure used in this study was based on the paradigm described by Diamond et al. (1999). The child was shown a novel junk object (the sample) covering a wooden well. When the child displaced the junk object, he or she was able to retrieve a reward (toy or dry snack such as cheerios) from the well. The sample was removed and a delay of 5 s was imposed. After the delay, the sample was again presented to the child along with a novel junk object (the nonmatching sample). The child was able to retrieve a reward only by reaching to the nonmatching sample. If the child reached incorrectly, the experimenter showed the child that the reward was with the nonmatching object, but the child was not allowed to retrieve the reward. New stimuli were used on each trial with a 15-s intertrial interval, and trials were administered until the child had reached criterion performance (reaching for the novel object on five consecutive trials) or a maximum of 19 trials had been administered. Side of reward was varied quasirandomly according to a Gellerman series. The dependent variables were the percent of trials on which the child reached correctly and the number of errors to criterion.

Two versions of the DNMS were administered. The first utilized actual objects as the samples, as described above. The second version was part of a separate study on social versus nonsocial memory and, instead of junk objects, digital photos of toys enclosed in plastic picture frames were used to cover the wells. On this second version, each child was administered 24 trials regardless of performance. The dependent variables were the percent of trials on which the child reached correctly to the novel photograph and the number of errors to criterion. The two versions of the DNMS are referred to as DNMS-objects and DNMS-pictures, respectively.

*Object discrimination reversal.* This task required the child to learn which of two repeatedly presented objects was associated with a reward (regardless of the object's location). After the child demonstrated that he or she had made an association between the object and reward by reaching for the correct object five times consecutively, a reversal occurred and the child was required to learn that the reward now was under the other object. This continued until the child had been administered up to two reversals, or failed to reach criterion within 25 trials. The dependent variables for ODR performance included percent of times the child met criterion for correct performance, total number of errors to criterion, and total number of perseverative errors (an error following an error).

### Dorsolateral Prefrontal Neuropsychological Tasks

*A not B task.* The child was seated across the table from the examiner. Two identical blue cups were placed on the table to the child's right and left and equidistant from the child. While the child observed, a reward was placed under the cup on one side (starting side was varied randomly across children within groups). An assistant lowered a white foam-board screen that obscured the child's view of the cups for 5 s. Then the screen was lifted and the child was prompted to find the reward. If the child reached correctly, he or she retrieved the reward. If the child reached incorrectly, the examiner showed the child the location of the reward, but the child was not permitted to retrieve the reward. After an interval of 15 s, another trial was administered, hiding the reward on the same side. The side of hiding was reversed after the child reached correctly for two consecutive trials. After two reversals followed by two consecutive correct choices, the delay was increased to 12 s. Trials continued at 12 s until the child had attained two more reversals followed by two consecutive correct choices or a maximum of 24 trials had been administered. Dependent variables included percent of trials in which the child reached correctly at 5 s, percent of correct reversal trials at 5 s, percent of trials in which the child reached correctly at 12 s, and percent of correct reversal trials at 12 s.

*A not B with invisible displacement.* This task is a more challenging version of the A not B task. The child was seated across the table from the examiner. A green box with an open side facing the child was presented at the center of the table, and a reward was placed inside. A cover was draped across the entrance of the box, obscuring the reward from the child's view. While the child watched, the experimenter slid the box to the right or left of the child (starting side

was varied randomly across subjects within groups). An assistant placed a white foam board between the child and the box for a delay of 5 s. During the delay, the experimenter placed an identical, but empty, green box on the other side of the table, equidistant from the child. After the delay, the screen was lifted, and the child was prompted to find the reward. The child received the reward if he or she reached correctly. If the child reached incorrectly, the examiner showed the child the location of the reward, but the child was not permitted to retrieve the reward. After an intertrial interval of 15 s, another trial was administered, with the reward hidden on the same side. The side of hiding was reversed after the child reached correctly for two consecutive trials. Trials continued until the child reached correctly two consecutive times following the third reversal or a maximum of 14 trials had been administered. The dependent variables were percent of trials in which the child reached correctly and percent of reversal trials in which the child reached correctly.

*Spatial reversal.* The spatial reversal task is similar to the A not B tasks in that it assesses spatial memory for location of a hidden object. The child does not see the object hidden, however. The spatial reversal procedure used in this study was based on the paradigm developed by Kaufman, Leckman, and Ort (1989). The child was seated across the table from the examiner. With a white foam-board screen obscuring the child's view, two identical red cups were placed on the table equidistant from the child to his or her right and left and baited with a reward. The screen was lifted and the child was prompted to find the reward. The chosen side was designated the preferred side, and subsequent trials hid the reward on the same side. If the child reached for the preferred stimulus, he or she retrieved the reward. If he or she reached incorrectly, the child did not retrieve the reward and the experimenter stated, "Let's try again," without providing corrective feedback. After a child met criterion by choosing correctly on four consecutive trials, the side of hiding was reversed. This procedure was continued through 20 trials. The dependent variables were percent of trials in which the child reached correctly and percent of reversal trials in which the child reached correctly.

### Joint Attention Tasks

*Butterworth measure of joint attention.* This assessment was based on a method originally developed by Butterworth and Jarrett (1991) to assess joint attention skills in infants and toddlers. The child was seated across from a familiar examiner and allowed to play

with a mildly interesting toy. Four colored cardboard crosses, approximately 20 cm high, were mounted on the wall at the child's eye level, 157 cm from the center of the room. The crosses were placed 30° to the right and left, behind and in front of the child.

There were two types of joint attention probes, each delivered twice and each accompanied by an intake of breath to express surprise: (1) examiner gazing at the cross, and (2) examiner pointing to the cross. The examiner administered the probes after first securing the child's attention and establishing eye contact. Type of joint attention probe and the location of the stimuli were counterbalanced across participants. The examiner and an assistant coded live whether the child looked toward the stimulus. An error was defined as a failure to turn eyes toward the stimulus within 15 s from delivery of the stimulus. Any coding discrepancies, which were rare, were resolved immediately following the task by viewing the videotape. An additional coding from videotape by coders blind with respect to diagnosis and hypotheses was obtained for a random subset of participants (19% of total sample). Intraclass correlation coefficients for the live versus videotape coders were .81 for the joint attention task. The dependent variable was the percent of trials in which the child failed to respond correctly.

*ADOS-G: Response to joint attention.* This ADOS-G item assesses whether the child's attention is directed to a distant object as a function of the examiner's use of gaze and/or pointing (Lord et al., 1989). The item was coded based on the child's response to joint attention probes included in the play interview. While the child was playing quietly, the experimenter placed him or herself directly in front of the child and established eye contact by calling the child's name or, if necessary, providing a physical prompt. On making eye contact, the experimenter said, "Look [Child's name]," and looked toward a toy that had been placed in front and 65° to the side of the child. If the child did not respond to this joint attention probe by following the examiner's gaze to the toy, it was repeated, appending the phrase, "Look at that" to the verbal prompt. If the child failed to respond to this bid, the examiner stated, "[Child's name], look at that," and pointed to the toy. Scores ranged from 0 to 2. A score of 0 indicated that the child had successfully used the orientation of the examiner's face and eyes as a cue to attend to the toy. A score of 1 indicated that the child had required a point to attend to the toy. A score of 2 indicated that the child had not responded to any of the joint attention probes or that the experimenter had been unable to obtain the child's attention to administer the joint attention probe after five attempts.

*ADOS-G: Initiation of joint attention.* This ADOS-G item assesses whether the child makes attempts to direct an adult's attention toward an object neither of them is touching and that this is not a request for the object (Lord et al., 1989). This item was scored based on the examiner's judgment of the child's attempts at protodeclarative attention bids throughout the course of the entire play interview. Scores ranged from 0 to 2. A score of 0 indicated that on at least one occasion the child directed an adult's attention to a distal object by gazing at the object, establishing eye contact, and redirecting gaze to the object. Using a point or a vocalization was acceptable but not necessary to receive a score of 0. To obtain a score of 0 a child must have successfully integrated attention to the adult and attention to the distal object. A score of 1 indicated that on at least one occasion a child partially referenced a distal object by either looking at the object and pointing or vocalizing, or by looking or pointing at an adult without redirecting attention back to the object. The child may have demonstrated attention to the adult or attention to the object, but he or she failed to integrate the two in a bid for joint attention. A score of 2 indicated that the child did not initiate a bid for joint attention to reference a distal object.

## RESULTS

### Group Differences in Joint Attention Ability

A multivariate analysis of variance (MANOVA) was conducted on the three joint attention measures used in this study to assess for overall group differences. Children with ASD exhibited the expected impairment in joint attention, having significantly higher scores that indicated a greater impairment, (Wilks'  $F(6, 252) = 18.22, p < .001$ ). Examination of univariate ANOVAs revealed greater impairment for children with ASD on each measure (Table 2). Based on post hoc Tukey honestly significant difference tests, it was found that the children with DD and typical development did not significantly differ.

### Group Differences in Neurocognitive Ability

A series of univariate analyses of variance (ANOVA) was conducted to test the hypothesis that young children with ASD perform less well on ventromedial prefrontal tasks than do mental age-matched children with DD and typical development. Both measures of overall performance (e.g., percent correct, number of errors to criterion) and, where possible, specific errors (e.g., number of perseverative errors) were examined for all neurocognitive tasks.

**Table 2** Joint Attention (JA) Ability in Young Children with Autism Spectrum Disorder (ASD), Developmental Delay (DD), and Typical Development

JA Measures	ASD		DD		Typical Development		<i>p</i>
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	
Butterworth JA	.53	.37	.27	.31	.11	.18	<.001
ADOS—initiate JA	1.08	.75	.12	.33	.05	.23	<.001
ADOS—respond JA	.85	.82	.24	.55	.05	.23	<.001

Note: ADOS = Autism Diagnostic Observation Schedule.

Descriptive statistics for these variables for each group are presented in Table 3. There was a substantial floor effect for performance on the spatial reversal task, with 90% of children scoring at the floor on the variable reflecting percent correct reversals. For the rest of the tasks, children in all groups showed a similar range of levels of performance, indicating that the tasks were developmentally appropriate for the groups of children being tested. Given the large number of comparisons, a conservative  $\alpha$  level of  $p < .01$  was adopted. Across all neurocognitive tasks administered, no significant group differences,  $p < .01$  were found. Object discrimination reversal showed a trend toward group differences, overall errors:  $F(2, 131) = 4.31, p = .015$ ; and perseverative errors:  $F(2, 131) = 2.77, p = .066$ . Tukey post hoc analyses on these two variables showed that no pairwise comparison among the three groups was significant beyond  $p < .01$ . Thus, in these samples of children matched on mental age, there was little evidence for prefrontal impairment in 3- to 4-year-olds with ASD, relative to the developmentally matched comparison groups. Similar results were obtained when the children with PDDNOS were removed from the sample, and analyses were repeated.

### Relation between Neurocognitive Ability and Joint Attention

Structural equation modeling was used to test the hypothesis that performance on the ventromedial prefrontal tasks is more strongly associated with joint attention abilities in young children with ASD than is performance on the dorsolateral prefrontal tasks. The use of latent variable SEM takes the reliability of measurement into account by using the latent factor underlying multiple indicators of executive function constructs and accounting for the degree to which the various indicators covary with one another. Distinguishing between various prefrontal tasks via an explicit measurement model in SEM allowed us to re-

**Table 3 Neurocognitive Performance in Young Children with Autistic Spectrum Disorder (ASD), Developmental Delay (DD), and Typical Development**

	ASD		DD		Typical Development		<i>p</i>
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>	
Ventromedial prefrontal tasks							
Object discrimination reversal							
Percent of criteria met	.66	.42	.63	.41	.57	.46	.58
No. of errors to criterion	9.88	4.44	12.97	6.18	11.50	4.65	.02
No. of perseverative errors	3.09	3.16	4.71	4.31	3.13	2.60	.07
DNMS-objects with short delay							
Percent correct	.68	.22	.74	.23	.75	.19	.24
No. of errors to criterion	4.65	3.85	3.68	3.95	3.74	3.48	.34
DNMS-pictures with short delay							
Percent correct	.64	.23	.63	.22	.68	.19	.55
No. of errors to criterion	4.68	2.80	4.47	2.65	3.97	2.31	.42
Dorsolateral prefrontal tasks							
A not B							
Percent correct (5-s delay)	.86	.16	.85	.18	.81	.17	.42
Percent correct reversals (5-s delay)	.73	.33	.76	.34	.69	.34	.70
Percent correct (12-s delay)	.77	.18	.73	.22	.71	.24	.32
Percent correct reversals (12-s delay)	.55	.39	.51	.34	.47	.37	.62
A not B with invisible displacement							
Percent correct	.66	.21	.68	.20	.66	.15	.89
Percent correct reversals	.38	.34	.55	.39	.43	.35	.10
Spatial reversal							
Percent correct	.66	.13	.61	.14	.64	.13	.28
Percent correct reversals	.03	.10	.08	.22	.06	.19	.42

Note: DNMS = delayed nonmatching to sample.

fine our test of the relation between neurocognitive performance and joint attention. Spatial reversal was not used in the SEM analyses because of the substantial floor effect found for this task (90% of participants were at floor level on this task, suggesting that it may have been too challenging for this young sample). Ranges and standard deviations for the remaining neurocognitive tasks were similar. The latent factors for neurocognitive performance were constructed from the following measures: ventromedial factor (ODR: percent of times criterion was met; DNMS-objects: percent correct; and DNMS-pictures: percent correct) and dorsolateral factor (A not B, 5 s: percent correct; A not B, 12 s: percent correct; A not B with invisible displacement: percent correct). The joint attention factor was derived from the Butterworth measure of joint attention (percent failed), and ADOS-G scores on initiation and response to joint attention. These analyses were conducted on covariance matrices using the EQS program (Bentler, 1995). Parameter estimates were based on maximum likelihood estimation. Table 4 displays the correlations among the variables used in the SEM.

The two neurocognitive factors were composed using a confirmatory factor analysis model in which the variance of the latent factor was fixed at 1, and the variance of each indicator was free to vary. This allows for a significance test on the magnitude of each indicator's loading on the latent factor. The two neurocognitive factors were allowed to covary with one another. To assess the degree to which these latent neurocognitive factors uniquely explained variability in joint attention ability in children with ASD, structural paths were tested from the ventromedial and dorsolateral factors to joint attention (see Figure 1). It was hypothesized that the path between the ventromedial and joint attention factors would be negative (better performance on ventromedial tasks relates to lower scores on the joint attention impairment factor) and significantly different from 0, whereas the path between the dorsolateral and joint attention factors would not differ from 0. Overall, this model provided a good fit to the data,  $\chi^2(24, N = 46) = 32.04$ , comparative fit index (CFI) = .90, root mean square error of approximation (RMSEA) = .09. The standardized path coefficients are presented in Figure 1 and show

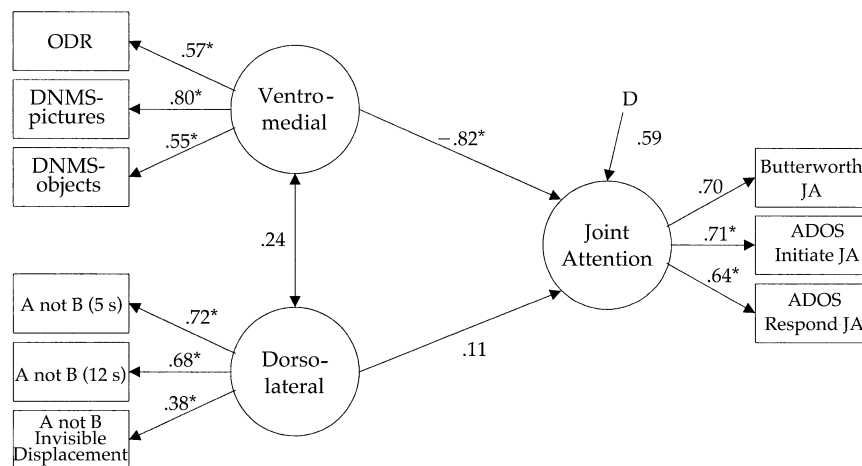
**Table 4** Correlations between Variables Used in Structural Equation Models

	Butterworth JA	ADOS—Initiate JA	ADOS—Respond JA	Object Discrimination Reversal	DNMS- Pictures	DNMS- Objects	A not B (5 s)	A not B (12 s)	A not B with Invisible Displacement	Mullen Composite Age
ADOS—initiate JA	.47									
ADOS—respond JA	.53	.40								
Object discrimination reversal	-.41	-.39	-.27							
DNMS-pictures	-.37	-.52	-.37	.45						
DNMS-objects	-.22	-.29	-.32	.31	.45					
A not B (5 s)	-.10	.05	-.08	-.08	.08	.15				
A not B (12 s)	.13	.02	-.22	-.06	.22	.15	.50			
A not B with invisible displacement	-.10	-.30	.05	.08	.31	.03	.295	.21		
Mullen composite age	-.71	-.52	-.545	.44	.44	.35	.03	.05	.25	
<i>M</i>	.57	1.11	.85	.67	.68	.69	.86	.80	.67	25.25
<i>SD</i>	.36	.71	.84	.43	.21	.23	.15	.18	.18	9.34

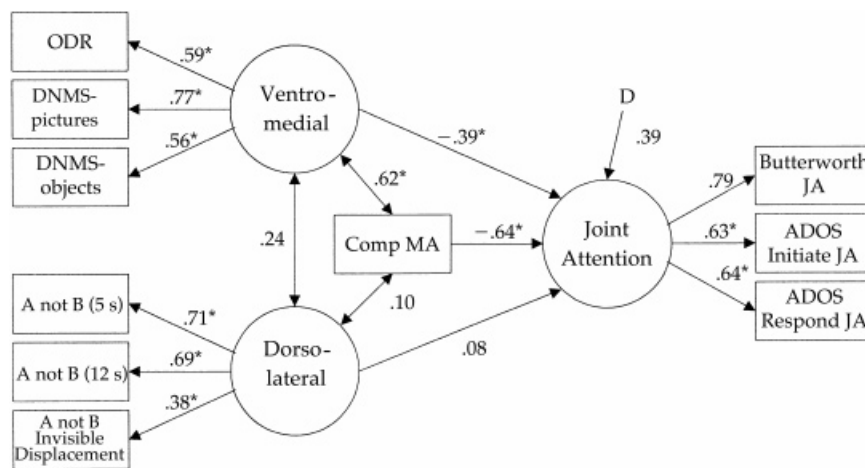
Note: JA = joint attention; ADOS = Autism Diagnostic Observation Schedule; DNMS = delayed nonmatching to sample.

that, as hypothesized, the direct path between the ventromedial factor and joint attention was significant, parameter estimate =  $-.204$ ,  $z = 3.58$ ,  $p < .001$ , whereas the direct path between the dorsolateral factor and joint attention was not, parameter estimate =  $.026$ ,  $z = .55$ , *ns*. A second model was run in which the ventromedial–joint attention path and the dorsolateral–joint attention path were constrained to be equal. This constrained model yielded a poorer fit to the data,  $\chi^2(25, N = 46) = 40.50$ , CFI = .808, RMSEA = .12, and the Lagrangian multiplier test (Bentler, 1995) on this equality constraint indicated that the ventromedial–joint attention path was significantly greater than the dorsolateral–joint attention path,  $\chi^2(1, N = 46) = 7.27$ ,  $p = .007$ .

To further test the specificity of the relation between ventromedial functioning and joint attention, composite mental age was added to the model. In this model (shown in Figure 2), the Mullen composite age equivalence score was added as a measured variable with correlational paths between both neurocognitive factors and a structural path to the joint attention impairment factor. Thus, in this model the direct path from ventromedial to joint attention assessed the unique contribution of the ventromedial factor beyond that of the dorsolateral factor and mental age. The standardized path coefficients are presented in Figure 2. The overall fit,  $\chi^2(30, N = 46) = 39.55$ , CFI = .92, RMSEA = .09, was similar to that of the earlier model. The direct path between mental age and joint



**Figure 1** Structural model of the relations among joint attention and neurocognitive abilities. ODR = object discrimination reversal; DNMS = delayed nonmatching to sample; ADOS = Autism Diagnostic Observation Schedule; JA = joint attention.



**Figure 2** Structural model of the relations among joint attention, neurocognitive abilities, and mental age. ODR = object discrimination reversal; DNMS = delayed nonmatching to sample; ADOS = Autism Diagnostic Observation Schedule; JA = joint attention.

attention was larger than for either neurocognitive factor. The unique contribution of the ventromedial prefrontal factor remained significant, however, parameter estimate =  $-.108$ ,  $z = -1.96$ ,  $p = .05$ . In summary, the relation between ventromedial prefrontal performance and joint attention ability was significant, whereas the relation between dorsolateral prefrontal performance and joint attention ability was not, parameter estimate =  $-.022$ ,  $z = .55$ , *ns*. Furthermore, the specific ventromedial prefrontal–joint attention relation remained significant, even when mental age was added to the model.

## DISCUSSION

The hypothesis that children with ASD would perform worse on neurocognitive tasks that tap the MTL–ventromedial prefrontal circuit than would children with DD and typical development was tested with a battery of tasks assessing the MTL–ventromedial prefrontal circuit versus the dorsolateral prefrontal cortex. Similar to Griffith et al. (1999), it was found that young children with ASD performed similarly to mental age-matched children with DD and typical development on tasks that assess executive functions. These results suggest that both children with autism and DD have impaired executive function, but that autism is not associated with a unique pattern of executive function impairment at this age. It is unlikely, therefore, that executive function impairment will be a good early diagnostic indicator of autism, because performance on executive function tasks by young children with autism appears to be similar to that of other children of comparable mental age.

As suggested by Griffith et al. (1999), it is possible that the executive function impairments that characterize autism are not evident until later in life. Studies that have found executive function impairments in individuals with autism relative to controls have assessed older children and adults (e.g., Dawson, Meltzoff, Osterling, & Rinaldi, 1998; Hughes & Russell, 1993; McEvoy et al., 1993; Ozonoff, 1995; Pennington & Ozonoff, 1996; Prior & Hoffman, 1990). Executive function skills are just emerging during the early preschool period (Diamond & Goldman-Rakic, 1989); thus, a lack of syndrome-specific executive function deficits in very young children is not surprising. The present sample is being followed longitudinally to assess the development of executive function ability. Autism-specific prefrontal impairments may not be apparent until the frontal lobe is more mature. Frontal impairment may be secondary to experience-driven effects of MTL dysfunction. Studies with monkeys (Bachevalier, 2001) showed that early MTL damage disrupts development of the circuitry and functions of the prefrontal cortex. Amygdala dysfunction, in particular, would be expected to disrupt very basic aspects of social processing during early life. Evidence for amygdala dysfunction in autism is mounting (Baron-Cohen et al., 2000). In fact, in the same sample of children who participated in the present study, impairments in very basic aspects of social cognition, such as face recognition, were found (Dawson, Carver, Meltzoff, McPartland, & Webb, in press). Amygdala dysfunction would likely interfere with the later development of some prefrontal functions, which develop rapidly during the preschool period and are facilitated by social interactions (Dawson,

Ashman, & Carver, 2000; Dawson, Panagiotides, Grofer Klinger, & Hill, 1992). If such a developmental disruption exists, one would expect a lag in frontal lobe development that becomes wider over time when compared with other clinical populations in which social interactions are not as compromised.

Our second hypothesis was that performance on tasks that have been linked to the MTL–ventromedial prefrontal circuit would be more highly correlated with joint attention ability than performance on tasks linked to the dorsolateral prefrontal cortex. Support for this hypothesis was found. As expected, children with ASD performed worse on the joint attention tasks than did children with DD and typical development. Moreover, structural equation analyses revealed a significant relation between MTL–ventromedial prefrontal task performance and joint attention ability, even after controlling for the significant effects of mental age level on joint attention ability. In contrast, performance on the dorsolateral prefrontal tasks was not related to joint attention performance.

These findings provide clues regarding the role of executive function in the development of joint attention. Dawson and colleagues (Dawson et al., 2000a, 2000b; Dawson, Carver, Meltzoff, McPartland, & Webb, in press) have theorized that rule learning regarding the relations between stimuli and reward is critical for many aspects of social functioning, including the development of joint attention and theory-of-mind ability. Expectations regarding the anticipated reward value of a stimulus begin to motivate attention by the second half of the first year of life (Ruff & Rothbart, 1996). Establishing such expectations regarding the anticipated reward value for social stimuli may be especially difficult for children with autism because social reward feedback (e.g., a smile in response to a behavior) is relatively less predictable and variable compared with nonsocial reward feedback (e.g., a sound in response to pushing a button; Dawson & Lewy, 1989). Gergely and Watson (1999) showed that in contrast to typically developing infants and toddlers, children with autism show a strong preference for highly contingent, nonvariable (i.e., perfect rather than imperfect) contingency feedback. Thus, unlike the normal infant whose attention is drawn to imperfect contingent feedback, which is characteristic of social interactions, the child with autism is drawn toward the less variable feedback of nonsocial stimuli (Dawson & Lewy, 1989; Gergely & Watson, 1999). Alternatively, it is possible that autism involves a fundamental deficit in social attention related to motivation and sensitivity to social reward (Dawson, Carver, Meltzoff, McPartland, & Webb, in press; Mundy & Neal, 2000).

Advanced joint attention taxes the ability to estab-

lish rules regarding stimulus-reward associations even further by requiring the coordination between children's own expectations regarding the reward value of a stimulus and those of others. For example, when showing an object to another person, children must coordinate their own interest in the object with expectations that their mother will likely respond in a rewarding way by showing a similar interest in the object. Other joint attention abilities further require children to flexibly modify their association between a stimulus and reward in response to varying reward feedback. For example, in the standard social referencing situation, children must hold on line and possibly inhibit their own initial expectations regarding the reward value of a novel stimulus (e.g., This object looks like it might be scary) and must then incorporate the feedback provided by another person to form a new expectation regarding the reward value of a novel stimuli, which serves to guide motivation and behavior (e.g., Mom's face indicates that this object is going to be rewarding). In this and other ways, the ability to establish, generalize, and modify expectations regarding the association between stimuli and their reward value—a skill mediated by the ventromedial prefrontal cortex and assessed by tasks such as ODR—may play an important role in the development of joint attention and other aspects of social processing. In future studies, it would be of interest to examine the relation between individual differences in performance on ventromedial prefrontal tasks, such as the ODR task, and joint attention and other social abilities in a normative population. In the present sample of typically developing children, there was too little variability in their joint attention ability to adequately assess this relation. The question of whether impairments on ventromedial prefrontal tasks are characteristic of autism at a later age will need to be addressed in future studies with older individuals. Furthermore, it may be important to vary whether the feedback is social (e.g., praise) or nonsocial (e.g., food or toy) in nature, because children with autism may have specific deficits in their ability to respond to social reward (Dawson et al., 2000a, 2000b; Dawson, Carver, Meltzoff, McPartland, & Webb, in press; Mundy & Neal, 2000). The results of the present study, however, provide some beginning evidence that variation in ability to perform tasks related to the ventromedial prefrontal cortex may contribute to a core impairment in autism, namely, joint attention ability.

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