

Examining the Relationship between Executive Functions and Restricted, Repetitive Symptoms of Autistic Disorder

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The executive function theory was utilized to examine the relationship between cognitive process and the restricted, repetitive symptoms of Autistic Disorder (AD). Seventeen adults with AD were compared to 17 nonautistic controls on a new executive function battery (Delis-Kaplin Executive Function Scales). Restricted, repetitive symptoms were measured by a variety of instruments (i.e., the Autism Diagnostic Observation Schedule, Autism Diagnostic Interview-Revised, Gilliam Autism Rating Scale, and the Aberrant Behavior Checklist). The study replicated the executive function profile that has been reported in adults with AD. In addition to the replication findings, the study found several executive processes (i.e., cognitive flexibility, working memory, and response inhibition) were highly related to the restrictive, repetitive symptoms of AD; whereas, other executive process (i.e., planning and fluency) were not found to be significantly correlated with restricted, repetitive symptoms. Similarly, we found an executive function model consisting of relative strengths and deficits was the best predictor of restricted, repetitive symptoms of autism. The implications for the executive function theory and how the theory predicts core symptoms of autism are discussed.

KEY WORDS: executive function; restricted, repetitive symptoms; stereotyped behaviors; autistic symptoms; Delis-Kaplin Executive Function Scales.

EXAMINING THE RELATIONSHIP BETWEEN RESTRICTED, REPETITIVE SYMPTOMS OF AUTISTIC DISORDER AND EXECUTIVE FUNCTIONS

Evidence suggests that individuals with Autistic Disorder (AD) have impaired capacities to execute mental control necessary for maintaining a problem-solving strategy to obtain a future goal. Such a

capacity has become known as the executive function theory of AD (Hughes & Russell, 1993; McEvoy, Rogers, & Pennington, 1993; Ozonoff & McEvoy, 1994; Ozonoff, Rogers, & Pennington, 1991; Ozonoff, Strayer, McMahon, & Filloux, 1994; Pennington & Ozonoff, 1996; Rumsey & Hamburger, 1988; Russell, Jarrold, & Henry, 1996; Turner, 1999). The executive function theory of AD has garnered much attention as a possible explanation for the manifestation of autistic symptoms (Hughes & Russell, 1993; McEvoy *et al.*, 1993; Ozonoff *et al.*, 1991; Prior & Hoffmann, 1990; Rumsey & Hamburger, 1988). Most recently, the theory has provided an avenue to explore the least studied cluster of autistic symptoms (i.e., restricted, repetitive behaviors) (Turner, 1997, 1999).

Although restricted, repetitive behaviors are a hallmark of AD, few theories have examined their etiology, and only a handful of studies have examined the prevalence of such symptoms in the autistic

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population (Campbell *et al.*, 1990; Rumsey, Rapoport, & Sceery, 1985; Volkmar, Cohen, & Paul, 1986). Several theories have been developed concerning the etiology of the stereotyped behaviors that are observed in autism. A theory of restrictive, repetitive behaviors was developed from a developmental-operant perspective (Lovass, Newsom, & Hickman, 1987; Rincover, 1978). The general notion of this theory is that stereotyped behaviors develop from “self-stimulatory” behaviors that are present in all infants, and that autistic individuals fail to acquire more mature behaviors that replace these rudimentary “self-stimulatory” behaviors. One could speculate that early deficits in frontal lobe function could result in the stagnation of early development and limit one’s ability to screen out environmental triggers, which then might result in the stereotyped behaviors.

Other researchers tie aberrant behaviors to neurobiological abnormalities. Most recently Pierce and Courchesne (2001) found stereotyped behaviors frequently observed in AD were negatively correlated with cerebellar vermis lobules VI–VII, and these behaviors were also found to be positively correlated with frontal lobe volume. Other researchers, Lewis and Baumeister (1982) postulated that stereotyped behaviors could result from an abnormality in the nigro-striatal dopamine tract in the basal ganglia. The nigro-striatal tract is thought to regulate responsiveness to stimulation and sensorimotor integration. Lewis and Baumeister’s theory of stereotyped behaviors provides preliminary evidence for a relationship between stereotyped behaviors and the frontal lobes. Based on these aforementioned studies, we see that restrictive, repetitive behaviors could be a result of abnormal frontal lobe functioning. Moreover, researchers have suggested that restrictive, repetitive behaviors are excessive, more severe forms of the tendency to perseverate and possibly due to the inability to generate novel solutions or the inability to shift one’s set (Ridley, 1994; Turner, 1999), which are mediated by frontal lobe functioning.

In 1994, Ridley postulated that stereotyped behaviors and perseverations result from a failure to use an appropriate level of representations in mental processing. He theorized that the frontal lobes generate “higher-order mental representations” that consist of information which does not exist in the individual’s environment. These higher-order representations are specific rules, generalizations, and concepts that allow the individual to respond in a creative manner to novel stimuli or situations that are not directly dictated by the environment. Conse-

quently, when a motor program is initiated by the primary motor cortex, the frontal lobes must enact a higher-order mechanism to stop the simple motor program. If the higher-order representation is not enacted, then a perseveration or stereotyped behavior is observed. Ridley’s theory suggests that a flaw in the generation and initiation of higher-order representations, imprecise representation, or both, create the constellation of restricted, repetitive behaviors observed in autism.

Recently, Turner (1997) suggested two distinct and dissociable executive hypotheses. Similar to Ridley’s higher-order representation theory, Turner’s theory suggests that restricted, repetitive symptoms of AD are a direct extension of the tendency to perseverate and are due to flawed frontal lobe processing. However, she postulated that individuals with AD become “locked into” one specific behavior or thought because they are unable to control their attention, thus preventing them from inhibiting ongoing or elicited behaviors. This hypothesis is referred to as a failure of behavioral inhibition. The second component of Turner’s hypothesis is that individuals with AD lack the ability to generate novel responses without unambiguous prompting. The paucity of generating novel ideas or behaviors manifests itself as a repetitive display of particular behaviors, which she refers to as the impaired generative ability hypothesis. Therefore, Turner’s theory provides two distinct hypotheses as to how flawed executive processing could be related to the vast array of restricted, repetitive behaviors observed in individuals with AD. In contrast, Ridley’s theory indirectly suggests that multiple impaired executive processes are the cause of restricted, repetitive symptoms in AD.

Although neither the literature nor theory has determined the most plausible cause of restricted, repetitive symptoms in AD, the literature examining the executive processes in individuals with AD strongly indicates that individuals with AD have impaired cognitive flexibility (Bennetto, Pennington, & Rogers, 1996; Pascualvaca, Fantie, Papageorgiou, & Mirsky, 1998; Ozonoff, 1995; Ozonoff & McEvoy, 1994; Ozonoff *et al.*, 1991, 1994; Prior & Hoffmann, 1990; Rumsey & Hamburger, 1988, 1990; Szatmari, Tuff, Finlayson, & Bartolucci, 1990) and planning abilities (Bennetto *et al.*, 1996; Hughes, Russell, & Robbins, 1994; Ozonoff & McEvoy, 1994; Ozonoff *et al.*, 1991; Prior & Hoffmann, 1990).

In contrast to these well-documented executive deficits, the literature on AD does not appear to indicate that individuals with AD have significant

impairments in response inhibition (Bryson, 1983; Eskes, Bryson, & McCormick, 1990; Hughes, Russell, & Robbins, 1994; Ozonoff & Jensen, 1999; Ozonoff & Strayer, 1997) and working memory abilities (see Russell *et al.*, 1996; Ozonoff & Strayer, 2001; however, note that Bennetto *et al.*, 1996 found working memory deficits). The literature on the executive processes in AD is less clear on individuals with AD ability to generate novel ideas (i.e., fluency and generativity), although there appears to be a tendency for individuals to have difficulties with generativity. Several fluency studies have reported that individuals with AD have impaired verbal fluency abilities relative to control groups (Boucher, 1977, 1988; Lewis & Boucher, 1995; Minshew, Goldstein, Muenz, & Payton, 1992; Rumsey & Hamburger, 1988; Turner, 1999; Williams, Moss, Bradshaw, & Rinehart, 2002), which is in support of Ridley's and Turner's theories on the origin of restrictive, repetitive symptoms. Other fluency studies, however, have failed to detect a difference between autistic and nonautistic groups on similar fluency tasks (Boucher, 1988; Minshew, Goldstein, & Siegel, 1995; Scott & Baron-Cohen, 1996; Turner, 1999).

Similar to the negative finding reported on measures of verbal fluency, it is important to note that several researchers have not found cognitive flexibility deficits in individuals with AD (Minshew, Goldstein, & Siegel, 1997; Minshew *et al.*, 1992; Schneider & Asarnow, 1987). Similarly, there are mixed findings on the executive function theory's ability to discriminate between AD and Asperger's syndrome (see Manjiviona & Prior, 1999; Miller & Ozonoff, 2000; Rinehart, Bradshaw, Brereton, & Tonge, 2002). However, the executive theory appears to effectively discriminate between AD and other disorders (Ozonoff, 1999; Pennington, 1996). Given these caveats and in examining the breadth of executive function literature, we can see an executive function profile emerge. This profile consists of relative strengths in working memory and response inhibition and relative deficits in cognitive flexibility, planning, and fluency.

The primary goal of the present study was to examine the relationship between restricted, repetitive behaviors and the executive profile of AD. The present study set out to initially replicate the executive profile of AD using a new executive function protocol and well-established neuropsychological tests. The second portion of the present study was aimed at investigating the relationship between executive processes and restrictive, repetitive symp-

toms. Based on the executive function theory of AD and the profound cognitive flexibility deficits reported in the AD literature, we predicted that deficits in cognitive flexibility would be positively correlated with restricted, repetitive symptoms. Similarly, we hypothesized that impaired executive processes (i.e., cognitive flexibility, planning and fluency) would predict a uniquely significant portion of variance in restricted, repetitive behaviors, whereas, those executive processes which do not appear to be impaired (i.e., working memory and response inhibition) would not predict a uniquely significant portion of variance.

METHODS

Participants

Seventeen adults with AD, between 19 and 42 years of age ($M = 29$ years), were compared to 17 healthy nonautistic controls, between 18 and 45 years of age ($M = 29$ years). The inclusion criteria for participants with AD were that they (a) fell within the age range of 17–45 years, (b) have a Performance IQ (PIQ) score of 70 or greater on the Wechsler Adult Intelligence Scale-Third Edition (WAIS-III; Psychological Corporation, 1998), (c) have been previously diagnosed with autism by a licensed psychologist, or board certified child adolescent psychiatrist, pediatrician, or neurologist, and (d) met criteria for autism on the Autism Diagnostic Interview—Revised (ADI-R; Lord, Rutter, & LeCouteur, 1994), Autism Diagnostic Observation Schedule-Generic (ADOS-G; Lord, Rutter, DiLavore, & Risi, 2001; Lord *et al.*, 1989, 1997), and the Gilliam Autism Rating Scale (GARS; Gilliam, 1995). The following were inclusion criteria for the nonautistic group: (a) falling within the age range of 17–45 years, (b) not having a history of mental retardation, psychiatric illness, or head trauma, and (c) no psychotropic or illicit drugs use in the past seven days. The primary investigator verified these criteria for the nonautistic group participants during a prescreening telephone interview with each participant.

Procedure

Diagnostic Assessment

The diagnostic assessment of autistic participants consisted of three independent components.

First, individuals with autism were given the ADOS-C. Once the structured interaction was finished, the same research assistant administered the ADI-R with the parent or caregiver. The second component of the diagnostic assessment consisted of the parent or caregiver completing GARS and the Aberrant Behavior Checklist-Community (ABC-C; Aman & Singh, 1986). The final component of the diagnostic evaluation was the completion of the ABC-C by the research assistant who performed the neuropsychological assessment. In order to avoid potential demand characteristics, the research assistant who completed the neuropsychological assessment typically did not have access to information gathered from the ADOS or structured interview ADI-R. Individuals in the nonautistic sample did not undergo a diagnostic assessment. However, an ABC-C for the nonautistic group was completed based on behaviors observed during intellectual and neuropsychological testing. The protocols were scored in accordance with each measure's guidelines. Research assistants who administered the diagnostic procedures also scored and tabulated the ADI-R and ADOS-G algorithms. The primary experimenter tabulated the scores for the ABC-C and the GARS.

Intellectual and Neuropsychological Assessment

Research assistants with a Master's degree were trained and evaluated on the standard administration of the Delis-Kaplin Executive Function Scales (Delis *et al.*, 1996), the Wisconsin Card Sorting Test (Grant & Berg, 1948), and the WAIS-III. Scoring procedures for each test in the experimental protocol were obtained from the appropriate test manual (Delis, Kaplan, & Kramer, 2001; Heaton, Chelune, Talley, Kay, & Curtiss 1993; Psychological Corporation, 1998). The following subtests from the D-KEFS were administered: the California Verbal Fluency Test, the California Design Fluency Test, the California Stroop Test, the Tower of California, and the California Trails Test. These tests were selected because they most closely match previous measures utilized in the autism research and because they measure the five executive function domains outlined by Pennington and Ozonoff (1996).

Wechsler Adult Intelligence Scale-Third Edition

The Wechsler Adult Intelligence Scale-Third Edition (WAIS-III) is comprised of 12 subtests that measure a variety of intellectual abilities (Psychological Corporation, 1998). The test produces a Verbal IQ

(VIQ) and Performance IQ (PIQ) that are each derived from scores on six separate subtests. The WAIS-III's technical manual reports the reliability and validity of the subtests and the three IQ scores. The technical manual describes a factor analysis that reveals four index scales: Verbal Comprehension, Perceptual Organizational, Working Memory, and Processing Speed. The WAIS III technical manual also provides comprehensive reliability and validity data on each of the index scales and individual measures. The primary measures from the WAIS-III utilized in the current study were the Working Memory Index and the letter-number sequencing test. Both are described as measures of working memory.

Delis-Kaplin Executive Function Scale

The Delis-Kaplin Executive Function Scale (D-KEFS) is an instrument used to assess an individual's ability to successfully engage in flexible, abstract, creative, and planned behavior (Delis *et al.*, 2001). The age range for the D-KEFS is between 16 and 89 years, but select subtests can be administered starting at age 8 years.

The subtests of the D-KEFS employed in the current study are modifications of existing "classic" executive function tests, with a greater emphasis on analyzing performance in terms of component skills, especially cognitive flexibility (e.g., as on the California Trail Making Test). These new tests do not yield single global scores of performance, but rather provide several measures of component processes that may underlie various executive functions. Recently, these tests were found to be sensitive to the executive function deficits of adults with focal frontal lesions (Baldo, Shimamura, Delis, Kramer, & Knight, *in press*) and of children with fetal alcohol syndrome (Mattson & Riley, 1995). Several of these new executive function tests place maximum demands on cognitive shifting abilities in either verbal or nonverbal modalities. The D-KEFS technical manual (Delis *et al.*, 2001) provides a detailed description of the D-KEFS subtests and details of how each subtest empirically met reliability and validity standards. The following executive functioning tasks from the D-KEFS were employed in the current study, and a brief synopsis is provided on each: California Trail Making Test, California Design Fluency Test, California Stroop Test, Tower of California Test, and California Verbal Fluency Test. These measures were selected because of the theoretical neuropsychological constructs each

instrument measures (i.e., cognitive flexibility, fluency, working memory, planning, and response inhibition), their reliability and validity, and because of their similarity to previously utilized neuropsychological instruments employed in autism research.

The California Trail Making Test is a revised version of the Trail Making Test in the Halstead-Reitan Battery. The California Trail Making Test consists of five conditions that assess visual-motor sequencing, visual scanning, number-letter switching, and motor speed. The number-letter switching task requires participants to alternate between connecting numbers and letters, which is proposed to be a measure of cognitive flexibility. Given the ages of participants involved in the current study, the internal consistency for the California Trail Making Test were reported to range between .69 and .74 and standard error of measure ranged from 1.44 to 1.66.

The California Design Fluency Test has three conditions that require participants to make unique designs or patterns by connecting dots with straight lines. In the first condition, participants are presented with five filled dots and are asked to connect four of the dots with four straight lines. The second condition requires participants to connect four of the empty dots with four straight lines. The third condition involves a switching response that requires the individual to alternate between connecting filled and empty dots. The internal consistency for the switching condition on the California Design Fluency Test were reported to range between .48 and .63 and standard error of measure ranged from 1.69 to 2.15.

The California Stroop Test is another classic executive function paradigm that evaluates the ability to inhibit an over-learned verbal response (reading words of colors) in order to name the dissonant ink color. Many Stroop tests have three conditions: a baseline naming condition, a baseline reading condition, and an interference condition. The California Stroop Test adds a fourth condition in which the interference condition is repeated, only half of the stimulus words are encased in a box. The subject names the dissonant ink color except for the boxed words, in which case the subject must switch sets and read the word itself (and not name the dissonant ink color); this is called the "switching condition." The D-KEFS manual reports internal consistency ranged between .75 and .82 and the standard error of measure ranged between 1.29 and 1.59 for the California Stroop's interference task.

The Tower of California Test is a planning task that is a modification of the Tower of Hanoi (Borys,

Spitz, & Dorans, 1982) and Tower of London (Morris, Ahmed, Syed, & Toone, 1993; Shallice, 1982) tests. The Tower of California assesses spatial planning and cognitive flexibility (Delis *et al.*, 2001). The internal consistency of the Tower of California ranged between .60 and .72 depending on the age of the study's participant and the standard error of measure ranged between 1.59 and 1.89.

The California Verbal Fluency Test consists of six conditions, one of which is a switching condition. The California Verbal Fluency Test requires the individual to generate as many novel words to the letters F, A, and S. There are also two semantic categories where participants are asked to generate names of animal species and boys' names. The final condition is a switching condition where participants alternate between naming fruits and pieces of furniture. In each trial, the individual is given 60 seconds to say as many words as possible. The technical manual for the D-KEFS reported internal consistency ranged between .80 and .90 depending on the age of the study's participant and the standard error of measure ranged between 1.69 and 2.15 for the California Verbal Fluency Test's switching task.

Wisconsin Card Sorting Test

The Wisconsin Card Sorting Test (WCST) is a test of basic concept formation and perseverative thinking. It was originally developed to assess abstract reasoning in normal individuals (Grant & Berg, 1948). This card sorting test requires individuals to sort stimulus cards based on three principles (i.e., color, form, and number). As described in the instrument's technical manual (Heaton *et al.*, 1993), individuals are not told the correct sorting principle, but instead must ascertain the correct principle based on feedback provided by the examiner about their card sorting choices. The four main variables derived from the WCST are the number of correct responses, number of perseverations, number of categories completed, and number of failures to maintain set. The most sensitive variable to brain damage, however, is the number of perseverative responses (Heaton, 1981).

Since the study's research questions hinge on participants' performances on the neuropsychological tests (the D-KEFS subtests, the WCST, and the letter/number sequencing from the WAIS-III), these tests were counterbalanced and administered first to avoid the effects of fatigue. The remainder of the WAIS-III was then administered according to standardized

procedures. Although this neuropsychological battery was administered in one test session, the individuals were provided with ample rest and breaks. The primary investigator scored all of the intellectual and neuropsychological tests to ensure uniformity; however, a research assistant randomly selected 10 neuropsychological protocols to verify the scoring.

Data analysis

Due to profound expressive delays, two participants were unable to complete the letter–number sequencing test from the WAIS-III's. Five individuals received cognitive testing less than one year prior to participating in the current study. These individuals were given the WAIS-III's letter–number sequencing task with the other EF tasks, but the intellectual testing was not repeated.

Restricted, Repetitive Behavior Composite Variable

The ADOS-G, ADI-R, GARS, and ABC-C were implemented to measure restricted, repetitive behaviors in the AD group. A primary caregiver or parent completed the GARS and ABC-C. The parents also reported on the participant's early developmental history to complete the ADI-R, and an examiner completed the ADOS-G with the autistic participants and completed an ABC-C based on the complete interaction with the participant. These measures were selected because of their diagnostic value, diversity of symptoms measured, objective scoring criteria, and reliance on multiple scores (parents/caregivers and examiners).

In an attempt to capture the complex nature and broad domain of restricted, repetitive behaviors, the restricted, repetitive domains of the ADOS-G, ADI-R, and GARS, along with the parents' and examiners' ratings on the ABC-C's Stereotypic Behavior, were converted into *z*-scores and averaged to form the "restricted, repetitive behavior composite" variable. Table I displays the intercorrelations between the restricted, repetitive behavior composite variable and each measure of restricted, repetitive behavior. Although the scores from the restricted, repetitive measures that were based on parental report were not significantly correlated with the scores from restricted, repetitive measures derived from the examiners' observations, all the measures were retained to ensure the entire domain was adequately measured and to reduce the effects of shared method variance in the correlational analyses. Cronbach's Alpha for the restricted, repetitive behavioral composite variable was .81.

Cognitive Flexibility Composite Variable

Two variables were combined to form the "cognitive flexibility composite" variable (i.e., the WCST's number of perseverative responses and the time to complete the letter–number switch task from the California Trails Test). These variables were selected because they theoretically measure the same construct and because of their high intercorrelations (i.e., WCST's perseverative responses, $r = .92$, $p < .001$, and California Trail Making Test's letter–number switching, $r = .67$, $p < .001$). The cognitive flexibility composite score was calculated by averaging *z*-scores across these two measures. For the cognitive flexibility composite score, Cronbach's Alpha was calculated to be .80.

Working Memory Variable

The WAIS-III's Working Memory Index and total number correct from the letter–number sequencing were utilized as measures of working memory abilities for the between group comparisons; however, the working memory composite variable was utilized in the correlational analyses. The WAIS-III's letter–number sequencing was the only variable used to comprise the Working Memory variable. The internal consistency (Cronbach's Alpha) for the letter–number sequencing total score ranged from .75

Table I. Intercorrelations between Diagnostic Measures and the Restricted, Repetitive Symptom Composite Variable ($n = 17$)

	1	2	3	4	5	Mean (SD)
1. Restricted, Repetitive Behavior Composite	–					
2. ADOS-G Restricted, Repetitive Behavior	.80**	–				2.53 (1.6)
3. ADI-R Restricted, Repetitive Behavior	.56*	.23	–			9.65 (4.2)
4. GARS Restricted, Repetitive Behavior	.42	.02	.16	–		7.06 (2.5)
5. ABC-C Examiner Stereotypic Behavior	.72**	.63**	.18	.03	–	3.82 (2.1)
6. ABC-C Parent Stereotypic Behavior	.77**	.74**	.29	.05	.42	3.19 (2.6)

Note: Autism Diagnostic Observation Schedule-Generic (ADOS-G), $n = 17$; Autism Diagnostic Interview-Revised (ADI-R), $n = 17$; Gilliam Autism Rating Scales (GARS), $n = 16$; Aberrant Behavior Checklist-Community (ABC-C) for the examiner, $n = 17$; Aberrant Behavior Checklist-Community (ABC-C) for the parent, $n = 16$.

* $p < .01$.

** $p < .001$.

to .88 depending on the groups' age (Psychological Corporation, 1998).

Fluency Composite Variable

Three variables were converted to *z*-scores and averaged together to formulate the fluency composite variable. The variables utilized to develop the fluency composite variable were the total number of animals ($r = -.89, p < .001$) and boys' names ($r = .90, p < .001$) generated on the verbal fluency task and the total number of unique designs produced from all three trials of the design fluency task ($r = .78, p < .001$), all these variables which are from the D-KEFs. Cronbach's Alpha for the fluency composite variable was .74.

In examination of the statistical analyses of the present study, one will find that the variables employed in the between group comparisons are not always the same variables utilized in the correlational analyses. To that point, one will see that the restricted, repetitive behavior, cognitive flexibility, working memory, and fluency composite scores were only used in the correlational analyses. Additional variables were selected for replication purposes and because they are thought to measure specific executive processes reflecting an individual's frontal lobe functioning. For example, the WCST's perseverative responses, the Heaton corrected T-scores from the WCST, and the California Trail Making Test's letter-number switching were employed as measures of cognitive flexibility. The total number of puzzles solved from the Tower of California was utilized to measure planning abilities. This planning variable was utilized for both the between group comparisons and the correlational analyses. The WAIS-III's Working Memory Index and total number correct from the letter-number sequencing were utilized as measures of working memory abilities for the between group comparisons; however, the working memory construct was utilized in the correlational analyses. Response inhibition was measured by the amount of time it took participants to complete the California Stroop's interference task. This sole measure of response inhibition was utilized for both the between group comparisons and the correlational analyses.

Finally, several measures of fluency were utilized in the between group comparisons, but the only fluency composite variable was utilized in the correlational analyses. The number of perseverations and the number of unique designs generated from the California Design Fluency Test, a measure of non-

verbal fluency, were utilized in the between group comparisons. In addition, the verbal fluency variables employed from the California Verbal Fluency Test for the between group comparisons were total number of words generated on F, A, and S trials and by the total number of words generated on the two semantic topic trials.

Analysis of assumptions for the variables derived from the California Stroop and California Trail Making Test revealed significant skewness, kurtosis, and violation of sphericity. A logarithmic transformation and the Greenhouse-Geisser F-test were used to correct for these violations for the California Trail Making Test. However, the California Stroop remained significantly skewed despite transforming the data. Therefore, a more conservative alpha level (.025) was employed in the California Stroop analyses.

RESULTS

Descriptive Statistics

Participants were matched on age ($M_{AD} = 29.1$ - years, $SD_{AD} = 8.0$; $M_{NC} = 29.4$, $SD_{NC} = 11.4$), $t(32) = .12, p = .90$, and Performance IQ ($M_{AD} = 84.1$, $SD_{AD} = 12.2$; $M_{NC} = 87.6$, $SD_{NC} = 11.7$), $t(32) = .86, p = .40$. Despite utilizing a between group matching strategy to control for Performance IQ, the control group's Verbal IQ ($M = 92, SD = 15$) and Full Scale IQ ($M = 89, SD = 13$) were significantly higher than the autistic group's Verbal IQ ($M = 73, SD = 16$), $t(31) = 3.42, p < .002$, and Full Scale IQ ($M = 77, SD = 15$), $t(31) = 2.5, p < .01$. Although there were three more females in the normal control group (with 11 males and 6 females) than in the autistic group (with 14 males and 3 females), the groups did not differ significantly on gender, $t(34) = .63, p = .53$. The descriptive statistics for the diagnostic evaluation can be found in Table II.

Replication of Previous Findings

Cognitive Flexibility

The current study replicated the cognitive flexibility deficits frequently observed in AD. Table II displays the means and standard deviations for the groups' performance on the WCST. The AD group completed significantly fewer categories, $t(32) = 2.5, p < .01$, made more errors, $t(32) = -2.0, p < .05$, and generated more perseverative responses, $t(32) = -2.06, p < .05$, than did the normal control

group on the WCST. Moreover, the AD group also produced significantly more perseverative responses than the normal control group even when gender, age, and education were controlled, $t(32) = 2.6$, $p < .01$. A similar trend was observed on the composite cognitive flexibility variable ($M_{AD} = .25$, $SD_{AD} = .82$; $M_{NC} = -.25$, $SD_{NC} = .60$), $t(32) = -2.0$, $p < .05$.

A 2×5 (Group \times Trial) mixed factorial ANOVA was utilized on the data gathered from the California Trail Making Test. A significant Interaction, $F(1,31) = 5.11$, $p = .008$, main effect for Group, $F(1, 31) = 8.27$, $p = .007$, and main effect for Trial, $F(1,31) = 11.24$, $p < .001$, were detected. One planned comparison found that the groups did not differ on the time that it took to complete the letter-number switching task, $F(1,32) = 1.45$, $p = .24$. Bonferroni-corrected *post-hoc* analyses found that the autistic group performed similarly to the nonautistic group on the other four Trail Making tasks (i.e., visual scanning, numbers, letters, and motor speed). However, the autistic group completed the scanning task more quickly than the motor speed task, and the nonautistic group completed these two tasks in comparable times.

Planning

The autism literature on planning was the foundation for the second replication hypothesis, which stated that the autistic group would perform significantly worse than the nonautistic control group on a measure of planning. The data from the Tower of California indicated individuals with AD constructed fewer towers ($M = 3.4$, $SD = 1.97$) than the normal control group ($M = 6.1$, $SD = 2.7$), $t(32) = 3.2$, $p < .01$, which replicates previous studies.

Response Inhibition

The result of a 2×3 (Group \times Trial) mixed factorial ANOVA on the total time to complete the task did not detect a significant interaction, $F(3, 96) = 1.72$, $p = .17$ but did identify significant main effects for group, $F(1,31) = 5.47$, $p < .025$, and trial, $F(1,31) = 98.23$, $p < .001$. A planned comparison revealed that the autistic group did not differ from the normal control group on the interference task, $F(1, 31) = 2.25$, $p = .14$ (see Table III for descriptive statistics from the California Stroop).

Similarly, Bonferroni-corrected *post-hoc* analyses revealed that the groups did not differ on the

Table II. Descriptive Statistic for the Autistic and Nonautistic Groups on the Wisconsin Card Sorting Test and California Stroop ($n = 17$)

	Autistic Group		Normal Control Group	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
<i>Wisconsin Card Sorting Test</i>				
Number of Categories Completed	2.76	2.39	4.65	1.93
Number of Correct Responses	58.76	18.66	66.59	13.16
Number of Errors	56.88	32.46	35.65	29.34
Number of Perseverative Response	60.94	50.55	30.47	34.13
Number of Perseverative Errors	47.23	36.82	25.12	26.13
Failures to Maintain Set	.76	1.03	.71	.99
Heaton T-scores for Perseverative Responses	34.12	26.18	54.53	18.66
<i>California Stroop</i>				
Color Patch Naming	55.1	29.6	32.5	5.3
Time (seconds)	.65	1.3	.47	1.1
Errors				
Word Reading	42.6	22.3	29.5	25.1
Time (seconds)	.23	.56	.11	.49
Errors				
Interference	80.2	41.6	62.2	25.4
Time (seconds)	1.4	2.5	1.3	2.1
Error				
Switching	95.1	44.6	74.3	28.1
Time (seconds)	4.7	9.3	1.3	1.6
Errors				

Table III. Descriptive Statistics for the California Verbal Fluency test and California Design Fluency Test ($n = 17$)

	Autistic Disorder Group		Normal Control Group	
	<i>M</i>	<i>SD</i>	<i>M</i>	<i>SD</i>
<i>California Verbal Fluency</i>				
F, A, S	26.7	12.9	32.9	12.5
Animals	15.1	6.1	19.4	5.8
Boys Names	13.5	6.2	19.2	5.8
Switching	11.1	4.1	13.8	4.1
<i>California Design Fluency</i>				
Filled Dots				
Number of Perseverations	6.12	5.07	2.59	3.81
Percent of Unique Designs	.48	.36	.79	.26
Empty Dots				
Number of Perseverations	5.41	4.46	2.65	2.91
Percent of Unique Designs	.53	.36	.78	.24
Switching Between Filled and Empty Dots				
Number of Perseverations	3.06	5.0	.47	.78
Percent of Unique Designs	.66	.34	.82	.15

Note: The means and standard deviations for the F, A, S trial were summed across all three trials, whereas, the three semantic trials are the sum for one trials.

word-reading task, the color patch-naming task, or on the switching task. In addition, the post hoc analyses indicated that the interference task, $p = .001$, and switching task, $p = .001$, were more difficult than the word reading task and the color patch-naming tasks.

Similar to the total time to complete the California Stroop, a Group \times Trial mixed factorial ANOVA was employed to examine the number of errors generated. The interaction, $F(1,31) = .11$, $p = .95$, and main effect for group, $F(1,31) = .18$, $p < .68$, were not significant, but the main effect for trial, $F(1,31) = 7.58$, $p < .001$, was found to be significant. Bonferroni-corrected *post-hoc* analyses revealed both groups made more errors on the interference task than the color patch-naming task, $p = .03$.

Working Memory

As predicted, the groups did not differ on a measure of working memory ($M_{AD} = 5.9$, $SD_{AD} = 5.1$; $M_{NC} = 7.3$, $SD_{NC} = 3.2$), $t(31) = .97$, $p = .34$. However, the current study lacked sufficient power to detect a medium sized effect ($R^2 = .33$).

Verbal Fluency

Results indicated that the normal control group did not produce significantly more words across the F, A, and S trials ($M = 32.9$, $SD = 12.5$) than did

the autistic group ($M = 26.7$, $SD = 12.9$), $t(30) = 1.38$, $p = .17$ (see Table IV for the descriptive statistics on the California Verbal Fluency Test). Despite separating the semantic trials from the F, A, and S trials, a 2×3 (Group \times Trial) mixed factorial ANOVA indicated that neither the interaction, $F(1,30) = 1.29$, $p = .28$, nor the trial main effect, $F(1,30) = .003$, $p = .95$, or the group main effect, $F(1,30) = 3.97$, $p = .07$ (see Table III for descriptive statistics) were significant. Although a between group effect was not detected, a substantial effect size was observed ($R^2 = .50$). The large effect size reported here suggests the study lacked sufficient power to detect the group differences.

Nonverbal Fluency

Two 2×3 (Group \times Trial) mixed factorial ANOVAs were employed to explore the performance of the groups on the California Design Fluency tasks (see Table III for descriptive statistics). First we examined the number of perseverative designs, which yielded significant main effects for group, $F(1,31) = 7.2$, $p < .01$, and trial, $F(1,31) = 8.39$, $p < .001$, but did not yield a significant interaction, $F(1,31) = .26$, $p = .72$. Bonferroni-corrected *post-hoc* analyses revealed the autistic group generated significantly more perseverative responses on the filled dot, $p = .001$, and the empty dot trials, $p = .008$, than on the switching trial. This finding

Table IV. Correlations between Restricted, Repetitive Symptoms, Executive Functions, and Measures of Intelligence

	1	2	3	4	5	6	7	8
1. Restricted, Repetitive Symptoms	–							
2. Cognitive Flexibility	.63**	–						
3. Planning	-.08	-.59	–					
4. Working Memory	-.56*	-.74	.36	–				
5. Response Inhibition	.54*	.55	-.41	-.49	–			
6. Fluency	-.45	-.65	.40	.44	-.42	–		
7. Verbal IQ	-.45	-.81	.65	.76	-.57	.65	–	
8. Performance IQ	-.40	-.82	.48	.73	-.44	.49	.74	–
9. Full Scale IQ	-.46	-.87	.63	.79	-.56	.64	.97	.88

Note: Correlations between the composite restricted, repetitive variable and executive functions and intelligence quotients have $n = 17$ (i.e., column 1). An adjusted Bonferroni-correction procedure was utilized on the correlations between restrictive repetitive composite score and measures of intelligence. The correlations between executive processes and intelligence quotients have an $n = 34$ and are for descriptive purposes only. The sample size for working memory was less one subject because he was unable to complete the task.

* $p < .05$.

** $p < .01$.

suggests that the alternating task was more difficult than the other same color dot tasks for the autistic group, but the control group found all three tasks equally difficult.

Next, we examined the percentage of unique designs generated on the California Design Fluency Test. In the 2×3 (Group \times Trial) mixed factorial ANOVA there was not a significant interaction, $F(1,31) = 1.29$, $p = .28$, or main effect for trial, $F(1,31) = 2.41$, $p = .11$, but there was a main effect for group, $F(1,31) = 8.27$, $p = .007$. Through Bonferroni-corrected *post-hoc* analyses, we found that although the autistic group generated fewer novel designs on all three tasks, the groups did not significantly differ across all three tasks and that a similar number of novel designs were generated across all three tasks.

Statistically Controlling for Verbal Abilities

Given that the groups were not matched on verbal intellectual abilities and that we observed a large correlation between executive functions and VIQ, we reanalyzed the between group comparisons by using the executive function composite variables while statistically controlling for verbal abilities. The Verbal Comprehension IQ (VCIQ) score from the WAIS-III, which is a factor of the overall Verbal IQ, was used as the covariate because our measure of working memory is utilized in calculating the Verbal IQ on the WAIS-III but it is not used to calculate the VCIQ. This interdependence of a covariate on a DV (i.e., letter-number sequencing is on the same scale as

the Verbal IQ in the WAIS-III) was the primary reason we selected the VCIQ over the VIQ as the covariate and because such interdependence can create considerable ambiguity when trying to interpret the findings (Tabachnick & Fidell, 1996).

In using an ANCOVA to control for the verbal differences between the groups, we found that the groups did not differ on any of the executive processes. The group with autism performed similarly on measures of cognitive flexibility, $F(1,30) = 1.82$, $p = .19$, and planning, $F(1,30) = .002$, $p = .95$, to the nonautistic control group, which was contradictory to the mixed factorial ANOVA findings. In contrast, the ANCOVA findings were similar to the mixed factorial analyses, in that the groups did not differ on working memory, $F(1,30) = 2.43$, $p = .13$, fluency, $F(1,30) = 1.79$, $p = .19$, and response inhibition abilities, $F(1,30) = .19$, $p = .67$.

Correlations between Executive Functions and Restricted, Repetitive Behaviors

The bivariate correlations between the restricted, repetitive behavior composite variable and measures of executive functions are displayed in Table IV. As hypothesized, we found a strong positive correlation between cognitive flexibility and restrictive, repetitive behaviors, $r = .63$, $p = .007$. This finding provides preliminary evidence that the tendency to perseverate is related to stereotyped behaviors. Perhaps the most surprising findings of the study were that planning, $r = -.09$, $p = .74$ and fluency, $r = -.45$, $p = .07$,

abilities were not significantly correlated with restricted, repetitive behaviors. Equally unexpected, we found robust correlations between restrictive, repetitive behaviors and working memory, $r = -.56$, $p = .02$, and response inhibition, $r = .58$, $p = .02$. These findings suggest that relatively preserved executive processes influence restrictive, repetitive symptoms of autism and that executive strengths and deficits are related to restrictive, repetitive behaviors.

In addition to the *a priori* correlations, we also conducted several exploratory correlational analyses. Three correlations were also calculated on the relationship between restrictive, repetitive behaviors and the measures of intelligence. Given these correlations were exploratory analyses, we utilized an adjusted Bonferroni correction on the alpha levels. After the correction procedures, none of the correlations between the restrictive, repetitive behaviors and measures of intelligence were found to be significant. Table IV also displays the intercorrelations between the executive function process and the correlations between the executive process and intellectual abilities. These correlations were for descriptive purposes only, therefore, significance levels were not reported.

Several standard multiple regressions were employed to determine which executive processes predicted a uniquely significant portion of variance in restricted, repetitive symptoms. As seen in Table V, it was found that measures of intact, $r^2 = .51$, and impaired, $r^2 = .44$, executive function account for a

similar amount of variability in restricted, repetitive behaviors. Based on this analysis we found that cognitive flexibility was the only impaired executive process to uniquely predict restricted, repetitive behaviors. Similarly, we found that neither of the intact executive processes (i.e., working memory and response inhibition) predicted a uniquely significant portion of the variance in restricted, repetitive behaviors.

In an attempt to further investigate which executive processes are uniquely predictive of restrictive, repetitive behaviors, an additional *post-hoc* analysis was conducted using the cognitive flexibility, working memory and response inhibition to predict restricted repetitive behaviors. Each analysis is summarized in Table V. We found that all of these exploratory analyses accounted for a significant portion of variance in restricted, repetitive behaviors. When cognitive flexibility was entered into separate regression models with working memory and response inhibition, then none of the constructs predicted a unique portion of variance. In total, these findings suggest that intact executive processes predict a similar amount of variance as impaired executive processes and that cognitive flexibility does not mediate the relationship between intact (i.e., working memory and response inhibition) and restrictive, repetitive symptoms of autism. Moreover, these findings would suggest that an executive deficit model is insufficient to solely account for restrictive, repetitive symptoms.

Table V. Multiple Regression Analyses with Executive Functions Predicting Restricted, Repetitive Symptoms

Model	Name of Construct	R ² for Each Model	B	Beta	Semi-partial	t	p-value
1.	Cognitive Flexibility	.47	.49	.71	.34	2.56	.02
	Planning		.10	.31	.12	1.34	.20
	Fluency-		-.09	-.12	.02	-.46	.65
2.	Response Inhibition	.44	1.6	.43	.13	1.7	.11
	Working Memory		-.04	-.32	.07	-1.3	.23
3.	PIQ	.21	-.01	-.35	.05	-.95	.36
	VIQ		-.07	-.14	.008	-.38	.71
4.	Cognitive Flexibility	.52	.28	.40	.13	1.34	.20
	Working Memory		.24	.37	.006	1.58	.14
	Response Inhibition		-.04	-.09	.17	-.29	.77
5.	Cognitive Flexibility	.44	.69	.98	.30	2.26	.04
	PIQ		.02	.34	.05	.82	.43
	VIQ		.003	.08	.003	.19	.85

Note: Each of the above constructs was measured by the following variables: planning was measured by the total number of puzzles solved, cognitive flexibility variable was measured by the composite cognitive flexibility variable, California Stroop was measured by the time to complete the interference task of the California Stroop test, non-verbal fluency was measured by the number of unique designs generated across the three trials, working memory was measured by the letter-number sequencing task of the WAIS-III, and the verbal fluency task was measured by the number of words generated on the semantic trials of the California Verbal Fluency Test.

Two *post-hoc* standard multiple regression analyses were also conducted to examine the predictive differences between IQ and executive functions. Measures of intellectual abilities accounted for 21% of the variance in restricted, repetitive behaviors, which accounted for substantially less variance than regression equations that utilized measures of intact and impaired executive functions. Interestingly, when cognitive flexibility was entered into the same regression equation as PIQ and VIQ to predict restricted, repetitive symptoms, we found that cognitive flexibility predicts a uniquely significant portion of variance, but neither measure of intelligence predicted a significant portion of variance in restricted, repetitive behaviors. This finding suggests that executive abilities are more related and more predictive of restricted, repetitive symptoms than intellectual functioning.

DISCUSSION

The primary aim of the present study was to investigate how cognitive abilities in individuals with Autistic Disorder (AD) are related to the behavioral symptoms of the disorder. In order to achieve this goal, the current study initially set out to replicate the executive function findings reported in the autism literature. Utilizing a newly developed executive function battery and frequently utilized neuropsychological tests, we were able to replicate previously reported deficits in cognitive flexibility (Bennetto *et al.*, 1996; Pascualvaca *et al.*, 1998; Ozonoff, 1995; Ozonoff & McEvoy, 1994; Ozonoff *et al.*, 1994; Ozonoff *et al.*, 1991; Prior & Hoffmann, 1990; Rumsey & Hamburger, 1988, 1990; Szatmari *et al.*, 1990) and planning (Bennetto *et al.*, 1996; Hughes *et al.*, 1994; Ozonoff & McEvoy, 1994; Ozonoff *et al.*, 1991; Prior & Hoffmann, 1990). In addition, the study found that the autistic group performed similarly to the control group on measures of working memory and response inhibition, which is consistent with previous findings (see Bryson, 1983; Eskes *et al.*, 1990; Hughes *et al.*, 1994; Ozonoff & Strayer, 1997; Ozonoff & Jensen, 1999 for working memory, and see Russell *et al.* 1996; Ozonoff & Strayer, 2001 for findings on response inhibition). The findings of the present study in conjunction with previous findings provide strong support for an executive profile of relative strengths (i.e., working memory and response inhibition) and deficits (cognitive flexibility and planning).

The diversity of previous findings in the literature combined with the findings from the current study creates a complicated picture in determining how fluency abilities are related to this executive profile. Although the study did not find group differences on measures of fluency, our group of individuals with autism presented a marked tendency to perseverate on the nonverbal fluency tasks, which is similar to Turner's (1999) observations. In addition, a caveat needs to be addressed when thinking of the current study's fluency findings. This caveat centers on the medium-sized effect of .50 (Cohen, 1988) reported in our analyses. This medium effect size suggests that the verbal fluency results of the present study, and possibly previous studies, could be due to a lack of sufficient statistical power. Therefore, it will be important for future research conducted on fluency abilities within individuals with AD to have an ample number of research subjects to ensure an effect size of .50 can be detected. Such research could significantly help us to interpret the convoluted findings reported in the autism literature on verbal fluency abilities.

A caveat to the aforementioned findings is that the group with autism did not differ from the nonautistic group on any of the executive function abilities when we statistically controlled for verbal intellectual abilities. Given that the autism literature has reported robust effect sizes for cognitive flexibility and planning deficit in individuals with autism, the lack of group differences in cognitive flexibility, fluency and planning after statistically controlling for verbal intellectual abilities is thought to be a result of losing a degree of freedom in the statistical analyses, and that this loss of a degree of freedom was not countered by a substantial increase in statistical power. Hence, the lack of group differences after controlling for verbal abilities appears to be due to a statistical procedure as opposed to a real world representation. The working memory and response inhibition finds are consistent with the autism literature as a whole, and the verbal deficits associated with autism do not appear to interfere with relatively preserved executive abilities observed in individuals with autism.

After replicating previously reported findings, the study then turned to examine the relationship between executive abilities observed in individuals with autism and the restrictive, repetitive symptoms of AD. As predicted, we found a positive relationship between cognitive flexibility and restricted, repetitive behaviors. Contrary to our prediction, however, we

did find a significant relationship between restrictive, repetitive symptoms and working memory and response inhibition, but we did not find a significant association between restrictive, repetitive behaviors and planning or fluency. Although these simple correlations between executive functions and restrictive, repetitive behaviors are interesting in and of themselves, they do not depict the entire scope of the relationship between the cognitive abilities and core symptoms of AD.

Further examination of the relationship between executive processes and restricted, repetitive symptoms revealed that cognitive flexibility was the sole executive deficit to uniquely predict restrictive, repetitive behaviors. However, an exploratory analysis revealed that when cognitive flexibility was entered into the same regression model with working memory and response inhibition, none of the cognitive abilities uniquely predicted the restricted, repetitive behaviors. Moreover, the model predicted a significant portion of the variance in restricted, repetitive symptoms. When considered in total, the correlational findings provide preliminary evidence of an association between abnormal functioning in the prefrontal cortex and restricted, repetitive symptoms of AD. Moreover, the executive dysfunction theory of autism, being a deficit model, would not have predicted that relative executive strengths would contribute to the prediction of restrictive, repetitive symptoms. The current study also provides evidence that the executive function theory of autism needs to be reconsidered in a way that examines the entire executive profile of AD. Similarly, the study's findings propose an executive model of relative strengths and deficits to account for the restricted, repetitive symptoms of autism.

Although researchers tend to reify these executive processes and relate sole cognitive deficits to symptomatology, the data obtained in the present study provides additional evidence that these cognitive abilities are highly interrelated but that no single executive process can fully account for the restricted, repetitive symptoms of AD. In a similar vein, one could extrapolate that the entire executive function profile needs to be considered when researching the relationship between the executive phenotype of AD and social and communication symptoms of AD.

The study utilized Ridley's (1994) and Turner's (1999) theories to conceptualize the relationship between abnormal functioning in the prefrontal cortex with core symptoms of AD. We did find preliminary evidence to suggest that the tendency to

perseverate (i.e., inability to shift mental set or the executive process called cognitive inflexibility) is related to restrictive, repetitive behaviors. However, the study provides two lines of correlational evidence to suggest that impaired generativity does not appear to be related to the restricted, repetitive symptoms of AD. First, we did not find a significant relationship between measures of fluency and the restricted, repetitive symptoms of AD. The second line of evidence is that a measure of fluency did not predict a significantly unique portion of variance in restricted, repetitive symptoms. Turner (1999) postulates that these high rates of perseverative responding on nonverbal fluency tasks are indicative of generativity impairments. However, contrary to Turner's theory, we have provided evidence that the tendency for individuals with AD to perseverate on fluency measures might be moderated by deficits in cognitive flexibility. These two findings suggest deficits in generativity do not play a major role in the manifestation of restricted, repetitive symptoms of AD.

Although these findings provide evidence that generativity does not play a significant role in the production of restricted, repetitive symptoms, we must consider the findings provisional given our inability to analyze all of the executive function component processes in one multiple regression. Moreover, the generativity findings presented here need to be considered provisional due to the magnitude of the intercorrelations between executive function measures. Further research is needed to determine if the perseverative responses by individuals with AD on measures of fluency are a result of impairments in generativity, other executive function impairments, or a third unexplored possibility. The current model also needs to be replicated in a younger population before any theory is dismissed.

Although the current sample of individuals with AD displayed significant planning deficits, the planning impairments were not significantly correlated with and did not significantly predict restricted, repetitive behaviors. Although this finding was not surprising given the multidimensional nature of executive processes, we suspect that planning deficits were unrelated to autistic symptoms because planning skills are more similar to meta-representation. Ridley (1994) describes meta-representations as executive processes in one part of the brain about representations in another part of the brain. Similar to executive functions, meta-representations are also generated and manipulated in the frontal lobes (Baron-Cohen *et al.*, 1994). A difference between

executive processes and meta-representations is that meta-representations do not have any theoretical implications for stereotyped or repetitive behavior. Meta-representations, however, are thought to be highly related to the social deficits associated with autism (Baron-Cohen, 1988; Frith, 1989; Happé, 1993; McEvoy *et al.*, 1993; Tager-Flusberg, 1993) because meta-representations are the primary mechanism in developing a Theory of Mind (Frith, 1989). Therefore, conceptualization of autism as a failure to use varying levels of representation in mental processing provides a vehicle to possibly account for a variety of AD's hallmark symptoms, deficits in theory of the mind, and the observed executive function deficits.

Our correlational data also supports researchers' arguments that executive functions and general intellectual abilities measure similar cognitive capacities (Ardila, Galeano, & Rosselli, 1998; Basso, Nasrallah, Olson, & Bornstein, 1998; Pennington *et al.*, 1997). However, we have presented evidence to suggest executive functions and intellectual abilities are slightly different constructs. The present study did not identify a significant relationship between intellectual abilities and restricted, repetitive symptoms; whereas, a strong relationship exists between restricted, repetitive symptoms and multiple executive processes. Similarly, a model of general intellectual abilities did not predict a significant portion of variance in restricted, repetitive symptoms, but an executive function model did predict a significant portion of variance in restricted, repetitive symptoms. Given that we are presently unable to make any predictions as to the exact nature or cause of restricted, repetitive behaviors based on general intellectual theories (e.g., WAIS-III four factor structure or fluid-crystallized theory) (Cattell, 1971; Cattell & Horn, 1978; Horn, 1968; Horn & Cattell, 1966, 1967; Psychological Corporation, 1998), then a strong relationship between restricted, repetitive symptoms of AD and measures of intellectual abilities would have been unexpected.

Since the inferences drawn from the current study are largely based on correlational data, the results do not indicate a causal relationship between executive processes and the restricted, repetitive symptoms of AD. The inherent nature of correlational data always allows for the possibility of equally valid alternative explanations of the current findings. The present data provides preliminary evidence for a link between prefrontal dysfunction, cognitive abilities and specific types of AD symptoms. It is possible

that the constellation of restricted, repetitive symptoms in individuals with AD could be caused by a unitary neurobiological abnormality that also results in multiple cognitive and neuropsychological strengths and impairments. Yet, the present findings clearly suggest that a unitary deficit model of executive functions is insufficient to account for all of the variance observed in restricted, repetitive behaviors. An alternative possibility is that multiple neurobiological abnormalities exist independently (e.g., neurochemical, neurophysical, or neurofunctional) and could simply manifest as variability in the EF profile, the intellectual impairments, and the triad of symptoms associated with AD (Bailey, Phillips, & Rutter, 1996; Goodman, 1989). In addition to alternative neurobiological explanations, it is important to note that restricted, repetitive behaviors are a complex behavior influenced by one's age, developmental level, intellectual functioning, and reactions to environmental stimuli (Bove, Di Sarno, D'Addio, Chiapparo, & Bove, 2003; Militerni, Bravaccio, Falco, Fico, & Palermo, 2002). Similarly, the current results could provide evidence of cognitive mechanisms that maintain the restricted, repetitive symptoms of AD, in which case the correlational results would not speak to the etiology of symptoms. In any case, given this body of research is in its infancy, it is premature to dispel any one theory when attempting to study the associations between cognitive functions and restricted, repetitive symptoms of AD.

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