

Neurocognitive Features of the Frontal Lobe in Parents of Autistic Children

Burak BAYKARA, Özlem GENCER, Zeynep İLKİN, Süha MİRAL

Abstract

Objective: The aim of this study was to examine the neurocognitive functions of the frontal lobe in parents of autistic children.

Method: The study group included 64 parents of children (aged 4-18 years) diagnosed with autism, according to DSM-IV criteria, that were followed-up at the child and adolescent psychiatry outpatient clinic. Parents of children with Down syndrome (n = 60) were selected as the control group. We administered the Wisconsin Card Sorting Test (WCST), Stroop Test, and Wechsler Adult Intelligence Test (WAIS) to both groups in order to evaluate executive functions, attention, inhibition, and intelligence.

Results: Mothers of children with autism performed better than the control group mothers on the executive function measures of WCST. There were no group differences in Stroop Test measures of attention and inhibition, or in the verbal and performance intelligence subtests of WAIS. Fathers of children with severe autistic symptoms performed better on some WAIS subtests compared to other; however, there were no significant differences in IQ between the parents in both groups.

Conclusion: The results suggest that parents of autistic children could display different cognitive styles, but we did not observe any distinctive cognitive profile pertaining to frontal lobe functions. The cognitive ability of parents of autistic children and its neurobiological basis should be further investigated.

Key Words: Autism, parent, neurocognitive test, executive function, intelligence

INTRODUCTION

According to the Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV), autism disorder (AD) classified under the heading of pervasive developmental disorders (PDD), and is characterized by delay and impairment in social interaction and communication (American Psychiatry Association, 1994). AD is a neurobehavioral syndrome with a genetic component; however, researchers point out that there are multiple factors involved in its etiology and argue that AD patients are not a homogenous group (Bailey et al., 1998). In a study of the first-degree relatives of children with AD, researchers found that 25% of family members had AD-like symptoms. These symptoms involved recurrent and stereotyped behavior patterns, impairment in social

and communication skills, and they were said to exhibit a broad autism phenotype (Bolton et al., 1994; Volkmar and Klin, 2000). To what extent this broad autism phenotype influences individual families and societies has not yet to be understood (Klin et al., 2002; Micali et al., 2004).

The prevalence rate of AD increases gradually, which points to a significant impact of the broad autism phenotype on families and societies (Bryson, 1997). Researchers argue that in addition to problems with social and verbal communication skills, cognitive and executive dysfunction might be involved in the broad autism phenotype. Nevertheless, the extent of cognitive and executive dysfunction in AD is not clearly understood (Bailey et al., 1998). The neurocognitive profile of the

Received: 10.09.2006 – Accepted: 14.03.2007

Burak Baykara MD., e-mail: burak.baykara@deu.edu.tr

broad autism phenotype could be different for male and female relatives of individuals with AD. In a study conducted with relatives of individuals with AD, male relatives and siblings had showed more autism phenotype features compared to female relatives and parents (Bailey et al., 1998). Moreover, individuals with severe autistic symptoms might have a different etiology than those with mild symptoms (Beglinger and Smith 2001; Griffith et al., 1999). The probability of the presence of the autistic phenotype in other family members increases with the severity of autistic symptoms of diagnosed AD patients (Bailey et al., 1998).

There are several theories that attempt to identify the core dysfunctional area in AD. The most striking among them points to impairment in executive functions (EF). Executive functions include organized working memory, the ability to stop or delay inappropriate stimuli, cognitive flexibility, and the ability to organize and plan goal oriented activities (Gazzaniga et al., 1998). The prefrontal cortex and, in particular, the dorsolateral prefrontal cortex are responsible for executive functions (Goussé et al., 2002; Klin et al., 2002). Researchers found that although executive dysfunction is observed in individuals with AD (Hughes et al., 1994; McEvoy et al., 1993; Ozonoff et al., 1991; Ozonoff et al., 1994) and their relatives (Goussé et al., 2002), it does not seem to play a major role in AD (Griffith et al., 1999; Klin et al., 2002).

Planning, cognitive flexibility, and set-shifting ability are the executive functions most affected in relatives of AD patients (Klin et al., 2002). When researchers investigated the possible effects of executive dysfunction on the core impairments in autism they found that the inability to engage in creative play was more related to recurrent and stereotyped behaviors (McEvoy et al., 1993). Executive dysfunction alone does not account for recurrent and stereotyped behaviors. It is also related to problems with social functioning and communication skills (Goussé et al., 2002). Furthermore, impairment in other cognitive areas, such as theory of mind and shared attention, are linked to executive dysfunction (Griffith et al., 1999).

The present study aimed to investigate frontal lobe cognitive ability in parents of autistic children. We examined EF, attention, inhibition, and intelligence profiles of the parents. We then compared these parents with parents of children with Down syndrome (DS) (control group) in order to identify any differences in EF, attention, inhibition and intelligence. Moreover, we

looked at the differences between the parents of children with severe AD and those with mild-moderate AD, and the control group.

METHOD

The study group consisted of parents of children with AD and the control group consisted of parents of children with DS. The children of the parents in the study and control groups attended special education programs.

Sample

Study Group

We obtained the records of 241 children diagnosed with AD, according to DSM-IV criteria, that were followed-up at the child and adolescent psychiatry outpatient clinic. Age range of these children was 4-18 years. Selected families were phoned and provided information about our research project. In all, 37 families agreed to participate in the study. During the evaluation period, 5 families were excluded from the study; in 1 of the families the parents scored < 70 on the Wechsler Adult Intelligence Scale (WAIS), in another family the father was not a biological parent, in 1 family the autistic child did not meet the diagnostic criteria for AD when reevaluated according to DSM-IV, and the last family was excluded because the autistic child died at the age 4 years due to heart failure and therefore the diagnosis could not be reevaluate. Our study group consisted of 64 parents (32 mothers and 32 fathers).

The main selection criterion for the study group was the absence of any neurological disorder in the children diagnosed with AD that can cause AD. The selection criteria for both the research and control groups were as following: Parents that (1) had at least a primary education, (2) were aged between 25 and 55 years, (3) scored ≥ 70 on WAIS, (4) did not meet the criteria for a major depressive episode, a manic episode, or a psychotic disorder based on the Structured Clinical Interview for DSM-IV Axis-I Disorders, Clinical Version (SCID-I, clinical version) (Çorapçioğlu et al., 1999), and (5) did not have a history of illness or trauma that could affect cognitive ability.

Control Group

We selected parents of children with DS as the control group for 2 reasons: (1) Past studies of parents of children with DS have not identified any genetic risk

Table 1. Comparison of WCST, Stroop Test, and WAIS IQ scores of the mothers in the study and control groups.

Measures	Study group (n = 32)	Control group (n = 30)	t	P
Age (years)	38.50 ± 6.30	39.33 ± 6.24	-0.522	ns
Education level (years)	10.34 ± 3.72	10.13 ± 3.98	0.215	ns
WCST Results (mean ± SD)				ns
Total correct responses	90.25 ± 19.10	82.56 ± 23.13	1.430	ns
Total wrong responses	37.75 ± 19.10	45.43 ± 23.13	-1.430	ns
Perseverative response	21.62 ± 13.34	32.03 ± 21.85	-2.280	0.026
Non-perseverative error	19.81 ± 14.16	18.50 ± 11.80	0.395	ns
Perseverative error	19.37 ± 10.32	27.06 ± 16.48	-2.217	0.030
Stroop Test Results (mean ± SD)				
Section 1 (sec)	40.65 ± 8.73	40.89 ± 9.09	-0.104	ns
Section 2 (sec)	29.78 ± 7.31	32.41 ± 8.59	-1.304	ns
Section 3 (sec)	81.23 ± 36.13	78.29 ± 20.53	0.391	ns
Duration difference between sections 2 and 3 (sec)	51.45 ± 34.86	45.87 ± 15.14	0.808	ns
WAIS (mean ± SD)				ns
Verbal IQ	98.06 ± 11.62	95.06 ± 15.10	0.872	ns
Performance IQ	92.81 ± 13.28	90.82 ± 13.99	0.568	ns
Total IQ	95.34 ± 12.07	92.86 ± 14.71	0.723	ns

t-tests

factors for social relationship or communication problems, or stereotypic behaviour (Piven et al., 1997; Piven & Palmer, 1997); (2) Providing care to children with DS might possibly have emotional and social influences on the parents that are similar to those that affect the parents of AD children (Goussé et al., 2002).

In order to recruit parents of children with DS we contacted with the Society of Special Education Centers (Özel Eğitim Merkezi Derneği, ÖZEK) and a supportive association for people with DS (Down Sendromlular Yardımlaşma Derneği, DOSEYAD). We provided information about our research and made contact with 112 families that had children with DS aged 4-18 years. The study group and control group were matched for age and education level; age groups: 25-40 years and 41-55 years; education levels: primary school (5-8 years); high school (9-12 years); higher education (≥ 12 years). Parents were phoned and informed about the study. In total, 39 sets of parents initially agreed to participate; then, 6 sets of parents withdrew. Three sets of parents scored < 70 on WAIS and were therefore excluded from the study. In all, 60 parents (30 mothers and 30 fathers) of children with DS were included in the control group.

Measures

The mothers and fathers were simultaneously evaluated by 2 researchers at the outpatient clinic. Sociodemographic data on the children and parents were recorded. Children with AD were evaluated with the Childhood Autism Rating Scale (CARS). A clinical psychologist administered WAIS to the parents. Parents who scored > 70 were subsequently administered SCID-I, the Wisconsin Card Sorting Task (WCST), and Stroop Test.

Childhood Autism Rating Scale (CARS)

CARS measures social relatedness, imitation, emotional response, body and object use, adaptation to changes, visual-auditory response, anxiety, verbal and non-verbal communication. Professionals observe and evaluate the behaviors of children based on 15 items scored on a 7-point Likert-type scale. Scores range from 1 to 4. A score of 1 reflects normal behavior and 4 is indicative of abnormal inappropriate behavior. A total score ≥ 38 indicates severe autistic symptoms, whereas a total score < 38 denotes mild-moderate levels of AD (Schopler et al., 1980). The Turkish language version

Table II. Comparison of WCST, Stroop Test, and WAIS IQ scores of the fathers in the study and control groups.

Measures	Study group (n = 32)	Control group (n = 30)	t	P
Age (years)	42.21 ± 5.63	43.86 ± 7.31	-0.997	ns
Education level (years)	11.65 ± 3.18	11.30 ± 3.48	0.420	ns
WCST Results (mean ± SD)				
Total correct responses	81.93 ± 20.56	81.90 ± 20.49	0.007	ns
Total wrong responses	46.06 ± 20.56	46.10 ± 20.49	-0.007	ns
Perseverative response	32.50 ± 17.41	31.23 ± 19.10	0.273	ns
Non-perseverative error	18.43 ± 8.48	19.90 ± 8.96	-0.660	ns
Perseverative error	27.62 ± 14.04	26.20 ± 14.87	0.388	ns
Stroop Test Results (mean ± SD)				
Section 1 (sec)	39.64 ± 9.71	38.15 ± 7.44	0.671	ns
Section 2 (sec)	28.24 ± 6.92	29.74 ± 5.69	-0.929	ns
Section 3 (sec)	72.33 ± 23.01	69.87 ± 12.79	0.516	ns
Duration difference between sections 2 and 3 (sec)	45.40 ± 20.66	40.13 ± 10.63	1.251	ns
WAIS (mean ± SD)				
Verbal IQ	106.21 ± 12.55	103.86 ± 11.55	0.760	ns
Performance IQ	96.53 ± 10.58	95.96 ± 12.19	0.194	ns
Total IQ	102.53 ± 11.39	100.41 ± 11.15	0.732	ns

t-tests

of CARS has not been validated; however, previous AD studies in Turkey have used it (Aşan et al., 2006)

Wechsler Adult Intelligent Scale (WAIS)

David Wechsler developed WAIS in 1955 (Wechsler, 1955). It is administered individually to people ≥ 16 years of age. It consists of 11 subtests that measure verbal (6 subtests) and performance ability (5 subtests). The vocabulary subtest of the Turkish version of WAIS has not been standardized; therefore, we did not administer this subtest to the parents. We averaged the scores from the other verbal skills subtests and computed the mean as a test score of the vocabulary subtest (Şahin, 1996). Epir and Iskit validated the Turkish version of WAIS in 1972 (Epir & Iskit, 1972); nonetheless, a norm has not been established for the Turkish population.

Structured Clinical Interview for DSM-IV Axis I Disorders, Clinical Version (SCID-I)

SCID-I is a semi-structured clinical interview for making diagnoses of DSM-IV Axis-I disorders. First and his colleagues (1997) constructed the scale, which consists of a user handbook, scoring sheet, and guidelines

for interviewers. Professionals reach a diagnosis by evaluating the presence of symptoms, the absence of symptoms, or an insufficient level of symptoms with the help of codes and additional information on subjects. The reliability and validity of the Turkish version of SCID-I were reported by Çorapçıoğlu et al. (1999).

Wisconsin Card Sorting Test (WCST)

Berg developed WCST in order to measure abstraction and cognitive flexibility (Berg, 1948). Heaton (1981) standardized its application and measurement techniques. Individuals must use diverse cognitive skills, such as abstraction, cognitive flexibility, selective attention, and maintaining set and working memory, in order to score well (Ozonoff, 1995). Although these abilities cannot be measured separately with WCST (Ozonoff et al., 1994; Ozonoff, 1995), it is an effective instrument for measuring frontal lobe abilities in neurological and psychiatric patient populations (Heaton, 1981). The perseveration variables on WCST significantly increase in people with frontal lobe damage (Howieson and Lezak, 1992; Lishman, 1998). WCST is the most frequently used executive functions test in AD research (Ozonoff,

Table III. Comparison between the mothers of children with severe AD and mild-moderate AD, and the control group mothers in terms of WAIS subtest scores.

WAIS Subtests	Mothers of children with mild-moderate AD (n = 13)	Mothers of children with severe AD (n = 19)	Mothers of children with DS (n = 30)	χ^2	P
WAIS (50p median)					
Verbal IQ	98.00	102.00	94.00	0.985	ns
Information	10.00	10.00	8.00	1.829	ns
Comprehension	10.00	11.00	10.00	0.166	ns
Arithmetic	7.00	7.00	7.00	1.358	ns
Similarities	11.00	11.00	10.00	1.443	ns
Digit span	7.00	9.00	7.00	3.742	ns
Performance IQ	92.00	95.00	91.00	0.933	ns
Digit symbol	9.00	8.00	8.00	1.011	ns
Picture completion	7.00	9.00	8.00	5.218	ns
Block design	9.00	9.00	9.00	0.053	ns
Picture arrangement	8.00	7.00	8.00	0.200	ns
Object assembly	7.00	7.00	6.00	0.765	ns
Total IQ	96.00	98.00	89.00	1.158	ns
Kruskal-Wallis test χ^2 : Chi-square test					

1995). It was standardized for use in Turkey by Karakaş and Başar (1996).

Stroop Test

The Stroop Test was developed to measure the rate of information processing and the ability to ignore interference caused by automatic processes (Stroop 1935). According to MacLeod (1992) it is the gold standard of attention measures.

Dysfunction in the orbitofrontal cortex of the frontal cortex generally leads to disinhibition -the inability to focus attention on relevant stimuli and inhibit those that are irrelevant. The Stroop Test is used to measure these orbitofrontal cortex skills (Gazzaniga et al., 1998; Karakaş & Kafadar, 1999; Ozonoff et al., 1991). Karakaş et al. (1996) standardized its Turkish version.

Statistical Analysis

We used the SPSS v.11.0 computer package program for statistical analysis. The Kruskal-Wallis, Mann-Whitney U, and t-tests were used for continuous variables; the chi square test was used for categorical variables. The level of significance was accepted as $P < 0.05$.

RESULTS

There were 64 parents in study group and 60 parents in the control group. Age and education level of the parents are presented in Tables I and II. There were no statistically significance differences between the study and control groups in terms of age and education level. In the study group 18 mothers (56%) were housewives and 14 (44 %) worked outside the home. In control group, 18 mothers (60%) were housewives and 12 (40 %) had jobs. There were no group differences in the occupation characteristics of the mothers. In the control group 29 fathers (91%) and in the control group 25 fathers (83%) worked, whereas 3 fathers (9%) in the study group and 5 fathers (17%) in control group were retired. There were no group differences in the occupation characteristics of the fathers.

In all, 25 (78.1%) of the children with AD were male and 7 (21.9 %) were female; 22 children (73.3%) with DS were male and 8 (26.7%) were female. There were no statistically significant group differences based on gender. Mean age of the children with AD was 10.40 ± 4.18 years, whereas mean age of the children with DS was 8.51 ± 4.17 years. There were no significant group

Table IV. Comparison between the fathers of children with severe AD and mild-moderate AD, and the control group fathers in terms of WAIS subtest scores.

WAIS Subtests	Fathers of children with mild-moderate AD (n = 13)	Fathers of children with severe AD (n = 19)	Fathers of children with DS (n = 30)	χ^2	P
WAIS (50p median)					
Verbal IQ	107.00	106.00	104.00	0.299	ns
Information	13.00	13.00	13.00	0.976	ns
Comprehension	12.00	11.00	12.00	0.088	ns
Arithmetic	10.00	10.00	10.00	0.068	ns
Similarities	12.00	10.00	11.00	0.389	ns
Digit span	7.00	10.00	7.00	3.899	0.028
Performance IQ	96.00	95.00	97.00	0.131	ns
Digit symbol	9.00	9.00	9.00	1.064	ns
Picture completion	9.00	9.00	9.00	0.114	ns
Block design	10.00	9.00	9.00	0.702	ns
Picture arrangement	7.00	8.00	7.00	0.271	ns
Object assembly	8.00	6.00	7.00	1.578	ns
Total IQ	103.00	103.00	102.00	0.271	ns

Kruskal-Wallis test

χ^2 : Chi-square test

differences based on age. Children with AD had 2.12 ± 0.79 siblings and those with DS had 2.34 ± 0.85 ; there were no significant group differences based on the number of siblings. According to CARS scores, 19 (59.4%) of the children had severe AD and 13 (40.6%) had mild-moderate AD. The latter group (CARS score < 38) had a mean score of 33.92 ± 2.46 and former group's (CARS ≥ 38) mean score was 50.84 ± 6.68 .

WCST scores of the mothers in both groups are shown in Table I; a statistically significant difference in the perseverative response subtest scores ($t = -2.28$, $P = 0.026$) and perseverative error subtest scores ($t = -2.21$, $P = 0.030$) was observed between the mothers in both groups. Mothers in the study group had a significantly lower mean perseverative response score and perseverative error score. Fathers in both groups did not significantly differ on WCST performance (Table II).

Reaction times on the first, second, and third sections of the Stroop Test were not statistically different between the study and control groups. The 2 groups did not differ significantly in their reaction times on sections 2 and 3. Similarly, the 2 groups did not differ signifi-

cantly on WAIS verbal, performance, and total IQ scores (Tables I and II).

We divided the study group into 2 sub-groups according CARS score: the parents of children with severe AD and the parents of children with mild-moderate AD (Tables III and IV). We compared these 2 groups with the control group. Results indicated that there were statistically significant differences between the 3 groups of fathers based on WAIS verbal IQ digit span subtest score ($\chi^2 = 7.16$, $P = 0.028$) (Table IV). Bonferroni correction showed that the fathers of children with severe AD scored the highest on the digit span subtest, followed by the fathers of children with mild-moderate AD and the control group fathers. The fathers of children with severe AD and the control group fathers significantly differed in verbal IQ digit span subtest score ($z = -2.36$, $P = 0.019$).

DISCUSSION

In the present study we observed that the study group performed better in some cognitive domains than the

control group. These differences in cognitive performance were not related to the cognitive profile of frontal lobe functions and were not unique to AD. The most sensitive measure of executive dysfunction on WCST is the perseveration rate (Heaton, 1981; Ozonoff, 1995). We found that mothers in the study group had statistically significantly lower mean perseverative response and perseverative error scores than mothers in the control group. This finding showed that mothers in the study group performed better on cognitive flexibility skills, which contradicts previous findings on executive dysfunction among individuals with AD (Benetto et al., 1996; McEvoy et al., 1993; Rumsey, 1985; Ozonoff et al., 1991; Pennington and Ozonoff, 1996; Hughes et al., 1997). It also did not support the view that executive dysfunction was a feature of the broad autism phenotype (Hughes et al., 1999). Nevertheless, our findings support previous research that suggests the developmental processes observed in AD could result in some cognitive advantages for individuals (Baron-Cohen and Belmonte, 2005). According to Baron-Cohen and Belmonte (2005), despite the impairments associated with AD, individuals with AD can function superiorly in some cognitive domains and that this is explained with the empathizing-systemizing theory of autism. Accordingly, individuals with AD have an impaired ability to empathize. In other words, these individuals have specific difficulty understanding others' thoughts and with general social and communication skills. The theory suggests that despite this impairment, some individuals with AD could have normal or superior ability in regulation areas, such as islets of ability, obsession with systems, and repetitive behavior. Our finding that the mothers in the study group had superior EF performance does not directly point to an advantage in this area in AD; yet, it shows that these mothers might have a unique cognitive profile. We recommend further studies that utilize more extensive neurocognitive measurement techniques to investigate the neurocognitive profile of the family members of children with AD.

Unlike the mothers, the fathers in the study group did not have significantly different performance in cognitive flexibility task than the control fathers. This finding suggests possible gender differences in EF performance between mothers and fathers in the study group; however, to test this hypothesis researchers should study gender differences in the neurocognitive features of the frontal lobe.

We observed no group differences in Stroop Test scores, (Stroop Test measures attention and inhibition

ability), which supports the assumption that attention and inhibition ability are affected at different levels in AD. Previous findings showed that the Stroop Test performance of children and adolescents with AD was not impaired (Ozonoff and Jensen, 1999; Russel et al., 1999). In the current study, the study group did not have any impairment in attention and inhibition ability.

Both WCST and the Stroop Test measure frontal lobe functions, but specifically, WCST and Stroop Test are measures of dorsolateral prefrontal cortex and orbitofrontal cortex functions, respectively (Karakaş and Kafadar, 1999; Pennington and Ozonoff, 1996). Attention and inhibition ability, as measured with the Stroop Test, are closely related to EF ability (Ozonoff and Jensen, 1999). We found general differences in WCST and Stroop Test performance, which suggest differences in frontal lobe (particularly the orbitofrontal cortex and dorsolateral cortex) functioning of parents in the study group. Our study group and control group mothers differed in WCST performance, but not in Stroop Test performance. This result is in agreement with the finding of Carper and Courchesne (2005); they observed prominent enlargement in dorsolateral convexity of the frontal cortex, yet no significant change in the orbital cortex in individuals with AD. In other words, these 2 frontal regions are separately affected in AD. The neurocognitive features of the frontal lobe of our study group mothers were in accord with the structural difference observed in the study by Carper and Courchesne (2005). Future studies should focus on the relationship between the broad autism phenotype and neurocognitive differences in the frontal lobe. Moreover, social and verbal communication skills of the family members of individuals with AD should be evaluated with respect to the broad autism phenotype.

In the present study the fathers of children with severe AD performed significantly better than the fathers of children with mild-moderate AD and control group fathers on the WAIS verbal IQ digit span subtest (which measures short-term memory and attention ability). Mothers of children with severe AD performed marginally better on the WAIS performance IQ picture arrangement subtest (which measures visual-motor perception and attention skills). The literature did not show pathognomonic profile of broad autism phenotype in Wechsler Intelligence Test (Siegel et al., 1996). Similarly, researchers argue that Wechsler Intelligence tests are not adequate for showing the genetic liability of AD (Bailey et al., 1998; Palmer, 1997). In our study, isolated advantages on WAIS subtests were not reflected in total

intelligence scores. We think that the possible differences in WAIS subtest performance did not adequately reveal group differences; however, isolated advantages on WAIS that the study group had might indicate diversity of cognitive structure and organization in these individuals.

Our results indicate that the control group performed well on WCST and WAIS subtests. This finding is not a reflection of an AD-specific condition. Hughes et al. (1997) did not observe AD-specific executive dysfunction, but observed executive dysfunction in parents of individuals with AD, which contradicts our finding that there was no EF impairment in the study group. A subgroup of parents of children with AD might have EF impairment (Hughes et al., 1997). We found differences in WCST performance among mothers in the control group and this pointed to a possible subgroup of mothers with better EF ability. Future studies need to look at such subgroups that differ in various EF tasks by using larger samples.

Our study sample was solely composed of parents of children with AD—we did not include other pervasive developmental disorder groups in our study. Furthermore, we analyzed the scores of each parent. These conditions increased the generalizability and sensitivity of our results. Nevertheless, we could not identify a prominent cognitive profile of the parents of children with AD. One reason was related to our measurements; data obtained from tests that we used were very complicated and included no particular elements (Ozonoff et al., 1993; Ozonoff 1995). The sensitivity of the tests was low because various cognitive domains, such as attention and perception, play a role specifically in EF test results (Hill, 2004; Ozonoff, 1995; Ozonoff et al., 1993). Another reason is the interaction of multiple genes in the etiology of AD. Different genes and the level of interaction among genes may differ from one individual with AD to another. That is why it is difficult to find a cognitive profile specific to AD and the broad autism phenotype (Bailey et al., 1998; Lauritsen and Ewald, 2001; Maestrini et al., 1998; Spence, 2001).

Our sample consisted of individuals from third step health center, which may not be representative of the general population; however, this type of sample is appropriate for studies on clinical groups with a low prevalence rate (Fombonne et al., 1997). Moreover, the interviewers were not blind to previous diagnoses of the children and the main hypothesis of our study; yet, despite this weakness possible measurement biases were low because we used standardized tests.

Another limitation of the present study is that we could not explain the relationship between adaptation to living with an autistic child and the isolated instances of high scores of the mothers on EF tasks and of the fathers on WAIS subtests. We selected parents of children with DS as a control group because the literature suggests the existence of similar communication and adaptation problems across families of children with mental development problems (Goussé et al., 2002). AD, however, is associated with more severe social and communication problems in the family than other developmental disorders. The effect of caring for a child with AD on the social and cognitive difference in parents was not studied in the past and we recommend that researchers consider the environmental effects on the cognitive profile of parents in future studies.

In conclusion, although we did not find any specific neurocognitive features, we found isolated instances of high cognitive performance scores related to frontal lobe functions among the study group. We did not directly evaluate the social and verbal communication skills of these parents within the broad autism phenotype, but we thought that the parents' isolated neurocognitive performances might have had a relationship to the broad autism phenotype. We highly recommend that researchers study the neurocognitive profiles of the parents of individuals with AD, and the relationship between these profiles and specific neurobiological structures, as well as the broad autism phenotype in general.

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