

# The Influence of Parental and Offspring Autism Spectrum Disorder (ASD) and Attention-Deficit/Hyperactivity Disorder (ADHD) Symptoms on Family Climate

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**Abstract** There is a lack of knowledge of the influence of parental and offspring autism spectrum disorder (ASD) and/or attention-deficit/hyperactivity disorder (ADHD) symptoms on the quality of family climate. The number of affected children may play an important moderating role. 103 Families were recruited with at least one child with an ASD(+ADHD) diagnosis, one or more biological affected or unaffected siblings, and two participating biological parents. Parents and children were carefully screened for the presence of ASD and ADHD symptoms. Family climate (relationship and structure) was measured with the Dutch Family Environment Scale (FES). No overall differences were found between ASD, ASD + ADHD families and a norm group for family relationship and family structure. However, families with one affected child reported higher family relationship scores than the norm, whereas fathers and mothers of families with two or three affected children reported similar scores as the norm. Regarding the family structure index, fathers and mothers of families with one, two or three affected children reported similar scores as the norm data of the FES. As reported by both fathers and mothers, paternal and maternal ASD symptoms and paternal ADHD symptoms had a negative effect on family

relationships, whereas predominately maternal ADHD had a negative impact on family structure. However, families with two or more affected children and families in which parents have increased ASD/ADHD symptoms may have more difficulties in maintaining a positive family climate.

**Keywords** Autism spectrum disorder · Attention-deficit/hyperactivity disorder · Family climate · Parental symptoms · Multiple affected children

## Introduction

Children with autism spectrum disorders (ASDs), characterized by impaired communication, and social interaction skills, as well as repetitive and restricted behavior and interest (APA 2000), and/or attention deficit hyperactivity disorders (ADHDs), characterized by severe inattention, hyperactivity and impulsivity (APA 2000) put a larger strain on parenting skills than typically developing children (Herring et al. 2006; Rutgers et al. 2007). In previous literature, child symptoms were associated with a suboptimal parenting environment, a less optimal parenting style, and more parenting stress (Johnston et al. 2012). Raising an ASD/ADHD affected child might also interfere with the family climate (Biederman et al. 2002a, b; Higgins et al. 2005). The family climate involves among others the quality of the interpersonal relationships, organization, structure and social activities and is responsible for the transmission of values and skills that are important for the social behavior of the child (Jansma and Coole 1996). The quality of the family climate is important because it may play a significant role in the severity, comorbidity and prognosis of a child's disorder (Jansma and Coole 1996). Evidence has been found for a suboptimal family climate,

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lower cohesion and more family conflict in families with ASD and/or ADHD affected children (Biederman et al. 2002a, b; Higgins et al. 2005; Johnston and Mash 2001; Kelly et al. 2008; Pressman et al. 2006; Schroeder and Kelley 2009). However, several important factors have not been examined yet that may explain these findings, such as (1) the role of ASD–ADHD comorbidity in the offspring on family climate, (2) the role of family size and the number of affected children, and (3) the role of parental symptoms (in combination with those of the offspring) that may create the suboptimal family climate. These factors will be discussed in detail below.

ASD and ADHD are disorders that frequently co-occur together within the same patient (Rommelse et al. 2010; Ronald et al. 2008; Rowlandson and Smith 2009) and within the same families (Freitag 2007; Pamploma et al. 2009). However, due to the fact that the diagnostic guidelines in the DSM-IV have so far prevented making a comorbid diagnosis, and ASD and ADHD have been studied over the past decades in isolation from each other, there is a lack of evidence for a combined effect of ASD and ADHD on family climate. It remains unclear whether there are differences in family climate in families with ASD and comorbid ADHD pathology. It is possible that the presence of comorbid ADHD pathology in families with ASD pathology may have a different effect on family climate than the presence of ASD only pathology in families. Families with offspring with only ASD may be less demanding and challenging for parents, since parents must deal with only one disorder in contrast to families dealing with the sometimes opposite upbringing demands of offspring having both ASD and ADHD pathology. Consequently, a more positive family climate may be found in families with ASD only pathology compared to families with both ASD and ADHD pathology.

Another possibility is that the family climate may be influenced by raising multiple affected children within one family. Although, this hypothesis has rarely been studied in relation to family climate before, it is not hard to imagine that it might be more difficult for parents to maintain a positive family climate when multiple children are affected compared to the situation in which only one child is affected. Parents of multiple affected children may be more stressed because they must switch between the demands of raising two or more affected children with each of their own problem behavior and needs. This has been reported before in a study of Orsmond et al. (2007) who found higher levels of depressive symptoms and anxiety and lower family adaptability and cohesion in mothers who were parenting another child with a disability in addition to a child with ASD compared with mothers with only one child with ASD. Importantly, family size may be relevant to consider in this context, since it may adversely affect family climate (Hart and Risley 1992; Jenkins et al. 2003).

However, in previous literature mixed results were found, with one study reporting no effect of family size on parenting or temperament developmental processes (Bridgett et al. 2009), but several other studies reporting negative effects of increased family size parent–child interactions (Hart and Risley 1992; Jenkins et al. 2003).

Family climate may also be influenced by parental pathology. Since both disorders are highly heritable, children with ASD and/or ADHD may have one or two parents that display subthreshold or above threshold symptoms (Constatino and Todd 2005; Faraone et al. 2009). In previous literature negative effects of parental symptoms were reported. For example, parental ADHD was associated with higher levels of family conflict and lower levels of family cohesion compared to families without parental ADHD (see for a review Johnston et al. 2012). However, to what extent parental ASD symptoms may affect family climate is largely unknown. This may be an unexpected consequence of the in 1940s launched hypothesis of Bettelheim (1967) that the cause of autism was attributed to a lack of maternal warmth. Although, it is now universally accepted that this hypothesis is flawed, the influence of parental ASD symptoms on family climate for long remained a very sensitive and neglected area of research. Therefore, it is of great interest is to examine the possible effects of parental ASD and ADHD symptoms on family climate.

The current study sets out to examine family climate in 103 families with at least one ASD affected (with/without ADHD) and at least one sibling and two biological parents. Family climate data (operationalized as family relationship and family structure) were obtained from both fathers and mothers, as were data on their own ASD and ADHD symptoms. These data allowed us to examine (1) if the family climate differs between families with ASD only affected children and families with ASD + ADHD affected children and the norm group, (2) if the family climate is influenced by the number of (affected) children, and (3) if the family climate is influenced by paternal and maternal ASD and ADHD symptoms. In addition, we explore the effect of age and comorbid problems such as anxiety, oppositional behavior and lower cognitive functioning, since these factors may have also an effect on family climate (Hudson and Rapee 2001; Maes et al. 2003; Theule et al. 2013).

## Method

### Participants

### Children

A subsample of 103 families was recruited as part of a large family-genetics study (Biological Origins of Autism

[*BOA*]). Families were included in the current study if (1) they had at least one child (2–20 years) with a clinical diagnosis of ASD (with or without ADHD); diagnosis mostly based on Autism Diagnostic Interview-Revised (ADI-R) and Autism Diagnostic Observation Scale (ADOS) assessment), and (2) at least one biological sibling (regardless of possible ASD or ADHD-status), and, (3) both biological parents willing to participate. All families were of European Caucasian descent. Participants were excluded if they were nonverbal, had an  $IQ \leq 60$ , specific learning or language disorders, a diagnosis of epilepsy, brain disorders or known genetic disorder such as Down-syndrome, Fragile-X-syndrome or Rett's syndrome. Comorbid *DSM-IV* disorders were not excluded.

Both the children already clinically diagnosed with ASD and/or ADHD and their siblings were carefully screened for the presence of ASD symptoms with the Social Communication Questionnaire (SCQ) (Rutter et al. 2003). Children with a score above 10 on the parent version (to avoid the exclusion of children with milder ASD symptoms e.g. false negatives) were included. To confirm the diagnosis of ASD, the Autism Diagnostic Interview Revised (ADI-R) (Le Couteur et al. 2003) was administered for the children scoring above clinical cut-off of the SCQ. Children were given a diagnosis of ASD if scoring above clinical cut-off on the ADI-R.

Furthermore, all children were carefully assessed for the presence of ADHD symptoms using the Conners Parent Rating Scales-Revised long version for parents (CPRS-R:L) and the Conners Teacher Rating Scales Revised long version for teachers (CTRS-R:L) (Conners 1997). For all children scoring above cut-off ( $T \geq 63$ ) on any of the parent or teacher Conners' DSM-IV Inattention, Hyperactivity-Impulsivity or Combined scales, the Parental Account for Childhood Symptoms (PACSs) (Taylor et al. 1991), a standardized semi-structured interview, was administered by a certified clinician. In this research the section of the PACS that covers the DSM-IV symptoms of ADHD were used. Thereafter, the items of the teacher rated CRS-R that were scored 2 [pretty much true] or 3 [very much true] were combined with the item scores on the PACS to confirm a formal diagnosis of ADHD (Rommelse et al. 2007a).

We were able to include 47 families ( $N = 114$  children) that had one or more children (probands and siblings) with an ASD diagnosis *without* clinical ADHD symptoms (ASD families) and 56 families ( $N = 148$  children) that had one or more children with an ASD diagnosis and/or clinical ADHD symptoms (probands and siblings) (ASD + ADHD families), see Tables 1 and 2 for sample characteristics. Of the 103 families, 68 families has one affected child, 31 families two affected children and 3 families three affected children.

Behavioral measures of parents (ASD and ADHD symptoms) were compared with control families ( $N = 73$  fathers and 74 mothers) recruited in the same way as ASD families of the *BOA*-study.

Full scale IQ of the children was estimated by using four subtest (Similarities, Block Design, Vocabulary and Picture Completion) of the Dutch version of the Wechsler Preschool and Primary Scale of Intelligence (WPSI-III: 2; 6–7; 11 years), Wechsler Intelligence Scale of Children (WISC-III: 6–16 years), or the Wechsler Adult Intelligence scale (WAIS-III: 16–84; 11 years) (Wechsler 1989, 2000, 2002).

### Parents

All parents were assessed for the presence of ASD symptoms with the self report Autism Spectrum Quotient (AQ; Baron-Cohen et al. 2001). The AQ consist of 50 items rated on a 4-point rating scale. The total score was used as indication of ASD symptom severity (Hoekstra et al. 2007, 2008). Self reported ADHD symptoms of the parents were assessed with the DSM raw total score for ADHD on a 4-points scale of the Conners Adult Rating Scales-Self-report: Long version (CAARS-S:L; Conners et al. 1998, 1999). In total, self reported ASD and ADHD data were available for 99/103 fathers and 103/103 mothers.

To assess the family environment, the Dutch version of the Family Environment Scale (FES) (Jansma and Coole 1996) was used. The FES consists of 77 yes/no questions (for example, 'we fight a lot in our family', 'family members often keep their feelings to themselves') related to seven subscales: (1) cohesion (family commitment and support), (2) expressiveness (expression of feelings), (3) conflict (expression of anger and aggression), (4) organization (structure and planning of family life), (5) control (rules used in family life), (6) family values (opinion about norms and behavior) and (7) social orientation (involvement in the social environment). Each subscale consists of 11 questions and scores range from 0 to 11, higher scores indicating a more positive environment, (vice versa for the conflict scale). In this study two index scores were used: the Family Relationships Index (FRI), based on three subscales (i.e. cohesion, expressiveness and conflict) and the Family Structure Index (FSI), based on two subscales (i.e. organization and control). Higher scores indicate better family relationships and parental structure (Gazendam-Donofrio et al. 2007). Both FES indices and subscales have shown good test-retest reliability and adequate internal consistency ( $>.60$ ) and has been extensively tested by normal controls, families with/without behavioral problems and families with/without parenting problems (Jansma and Coole 1996). The two subscales which are not linked to the two indices i.e. family values and social orientation, are not included in this study. Both mothers and fathers completed

**Table 1** Characteristics children

	ASD only families (N= 47)		ASD+ADHD families (N= 56)		Within comparisons Contrast	Norms (n)	Contrast		
	M (SD)	Within comparisons Contrast	M (SD)	Within comparisons Contrast				M (SD)	Contrast
N children per family	2.5 (.82)		2.8 (.97)			ns			
N affected children per family	1.2 (.41)		1.6 (.72)			asd < asd + adhd			
Relative N of affected children (Ratio <sup>a</sup> )	.52 (.18)		.60 (.21)			ns			
Male (%)	85.1/41.8	Probands (N=47)/siblings (N=67)	83.9/55.4	Probands (N=56)/siblings (N=92)		Between probands	Between siblings		
Age (years)	12.1(4.0)/12.2 (4.9)		11.3 (3.7)/10.3 (4.6)			ns	asd < asd + adhd		
Estimated full scale IQ	97.7 (16.2)/104.42 (14.1)	prob < sibs <sup>b</sup>	97.2 (15.9)/103.7 (13.7)	prob < sibs		ns	asd > asd + adhd		
SCQ <sup>c</sup> (ASD symptoms)	20.0 (7.7)/5.3 (5.9)	prob > sibs	20.5 (6.6)/7.0 (7.6)	prob > sibs		ns	ns		
CRS-R <sup>d</sup> (ADHD symptoms)	58.6 (9.2)/53.0 (9.2)	prob > sibs	67.1 (9.2)/54.9 (10.6)	prob > sibs	4.7 (5.0) <sup>c</sup>	asd = asd + adhd > n	asd + adhd > n		
CRS-R: oppositional scale	57.2 (11.7)/54.1 (12.7)	prob > sibs	63.3 (12.1)/ 53.8 (10.0)	prob > sibs	50 (10)	asd < asd + adhd > n	asd = asd + adhd > n		
CRS-R anxiety scale	65.8 (14.2)/54.9 (12.5)	prob > sibs	66.2 (14.5)/56.3 (15.2)	prob > sibs	50 (10)	asd = asd + adhd > n	asd = asd + adhd > n		
ADI-R social interaction scale	18.4 (5.1)/8.6 (7.3) <sup>e</sup>	prob > sibs	19.0 (5.7)/11.1 (8.0) <sup>f</sup>	prob > sibs	10	asd = asd + adhd > n	ns		
ADI-R communication scale	14.4 (4.2)/7.1 (6.2) <sup>e</sup>	prob > sibs	14.8 (4.6)/8.8 (6.5) <sup>f</sup>	prob > sibs	8	asd = asd + adhd > n	ns		
ADI-R restricted behavior and interest scale	4.4 (3.0)/1.4 (1.5) <sup>e</sup>	prob > sibs	4.3 (2.8)/2.4 (2.5) <sup>f</sup>	prob = sibs	3	asd = asd + adhd > n	asd + adhd > asd > n		
ADI-R impairments before 36 months scale	2.9 (1.5)/.9 (1.2) <sup>e</sup>	prob = sibs	2.7 (1.6)/1.5 (1.7) <sup>f</sup>	prob = sibs	1	asd = asd + adhd > n	asd + adhd > asd > n		
% ASD (ADI) <sup>g</sup>	100/13.4		100/13.0						
% ADHD (PACS) <sup>h</sup>	Not applicable		Not applicable <sup>i</sup> /13.0						
% ASD+ADHD (ADI and PACS)	Not applicable		80.4/8.7						

<sup>a</sup> Ratio = amount of children per family/affected children per family

<sup>b</sup> prob and sibs = probands and siblings

<sup>c</sup> Social Communication Questionnaire, norms based on population cohort (N = 247) (Chandler et al. 2007)

<sup>d</sup> Conners long version Rating Scales Revised

<sup>e</sup> Scores of siblings were based on N = 21 affected children

<sup>f</sup> Scores of siblings were based on N = 37 affected children

<sup>g</sup> Autism Diagnostic Interview (ADI-R)

<sup>h</sup> Parental Account for Childhood Symptoms (PACSs)

<sup>i</sup> In this study no pure ADHD group was included

**Table 2** Characteristics parents

	ASD only families (N = 47)		ASD + ADHD families (N = 56)		Norms (n)	Contrast Between fathers	Contrast Between mothers
	Within comparisons		Within comparisons				
	M (SD)	Contrast	M (SD)	Contrast			
Education	4.8 (1.0)/4.4 (.88) <sup>a</sup>	Fath > moth <sup>b</sup>	4.5 (.99)/4.4 (.89) <sup>c</sup>	ns	4.3 <sup>c</sup>	asd > n	ns
Autism Quotient (ASD symptoms)	104.4 (17.4)/95.2 (18.4)	Fath > moth	107.8 (20.7)/96.4 (21.0)	Fath > moth	95.4 (16.3)/84.1 (12.9) <sup>d</sup>	asd = asd + adhd > n	asd = asd + adhd > n
ADHD DSM-IV raw inatt scores <sup>e</sup>	4.2 (4.0)/3.9 (4.3)	ns	6.9 (5.0)/5.2 (6.3)	Fath > moth	4.4 (3.1)/3.3 (2.5) <sup>e</sup>	asd + adhd > n	asd + adhd > n
ADHD DSM-IV raw hyp-imp scores <sup>e</sup>	5.0 (3.9)/5.1 (3.8)	ns	5.6 (3.7)/5.7 (4.9)	ns	4.9 (3.2)/4.4 (2.4) <sup>e</sup>	ns	asd + adhd > n

<sup>a</sup> 1 = nursery school 2 = primary education 3 = secondary education, first phase 4 = secondary education, second phase 5 = higher education, first phase 6 = higher education, second phase 7 = higher education, third phase  
<sup>b</sup> Fath and moth = father and mother  
<sup>c</sup> Central Bureau of statistics (CBS 2009)  
<sup>d</sup> Scores were based on N = 73 fathers and N = 74 mothers of control families of the BOA-study  
<sup>e</sup> ADHD DSM-IV raw inattentive scores and raw hyperactive-impulsive symptoms, scores were based on N = 73 fathers and N = 74 mothers of control families of the BOA-study

the FES under supervision of the researchers. Data was available for N = 97 fathers and N = 102 mothers regarding the family relation scale and N = 97 fathers and N = 102 mothers regarding the family structure scale.

No FES-data was obtained from the control families recruited at the BOA-study. However, norm data of the FES were available for the two indices based on N = 1,707 Dutch families without psychopathology (Jansma and Coole 1996) with secondary education or higher education level comparable with the BOA-families. No differences were found between scores of fathers and mothers regarding both indices (Jansma and Coole 1996).

**Procedure**

Eligible ASD (with/without ADHD) families registered at an outpatient clinic specialized in ASD and ADHD pathology (Karakter Child and Adolescent Psychiatric University Center) and members of the Dutch Autism Association (NVA) received a brochure containing information about the BOA study and were requested to return a pre-stamped response card. A short telephone screening and, subsequently, screening questionnaires were used to verify if families were eligible to participate. These families were invited to visit Karakter Child and Adolescent Psychiatry Nijmegen, where a researcher conducted the diagnostic interview(s) (ADI-R and/or the PACS). Children who scored above clinical cut-off on the SCQ and CRS-R received both the ADI-R and PACS interviews. All researchers were extensively trained in the ADI-R and PACS by a certified trainer. The ADI-R and PACS training consisted of watching and coding several videotapes of interviews and discussions about coding and administration. The trained researchers achieved at least 80 % agreement in coding with the certified trainer. As part of a larger study, additional data was collected, including neuropsychological data of the children. The study was approved by the local medical ethics board and parents and children (above 12 years of age) signed for informed consent.

**Data-Analyses**

Analyses were performed with SPSS 20. The Expectation Maximization (EM) algorithm (Tabachnick and Fidell 2001) was used to impute the missing values (<5 %). Correction for multiple testing using the 95 % CI, was performed with the False Discovery Rate procedure (Benjamini 2010). One sample T tests were performed for comparisons with the norm data of the FES. To examine the effect of family type (ASD or ASD + ADHD) on the family relationship and family structure, two separate (regarding the two indices) repeated measure ANOVAs were used. Variables included into the model were (1) family type (ASD and ASD + ADHD), and (2) parent (father/mother) as repeated

measure (to account for within family measurements), and (3) the two-way interaction effect (parent by family type). To examine the effect of raising multiple affected children or family size, repeated measure ANOVAs were used with raising multiple affected children or family size and family type (ASD vs. ASD + ADHD) as factors, and parent (father/mother) as repeated measure.

Further, to examine to what extent own and spouse ASD and ADHD symptoms were related to family climate, ANOVAs were used. Dependent variable was the family relationship index or the family structure index of the FES. Variables included in the model were (1) paternal and maternal ASD symptoms (total AQ score), (2) paternal and maternal ADHD symptoms (DSM-IV ADHD raw subscales scores), (3) family type (ASD vs. ASD + ADHD) and (4) all two- and three-way interactions. The two- and three-way interactions were dropped from the model in case of non-significance and the model was rerun. We repeated the above analyses with the subcomponents of the two indices (cohesion, expressiveness, conflict, organization and control) to explore which component was especially impacted by own and spouse ASD and ADHD symptoms.

## Results

### Does Raising Multiple Affected Children Within One Family Negatively Influence Family Climate?

Comparisons with the norm data of the FES revealed that both fathers and mothers of families with one affected child

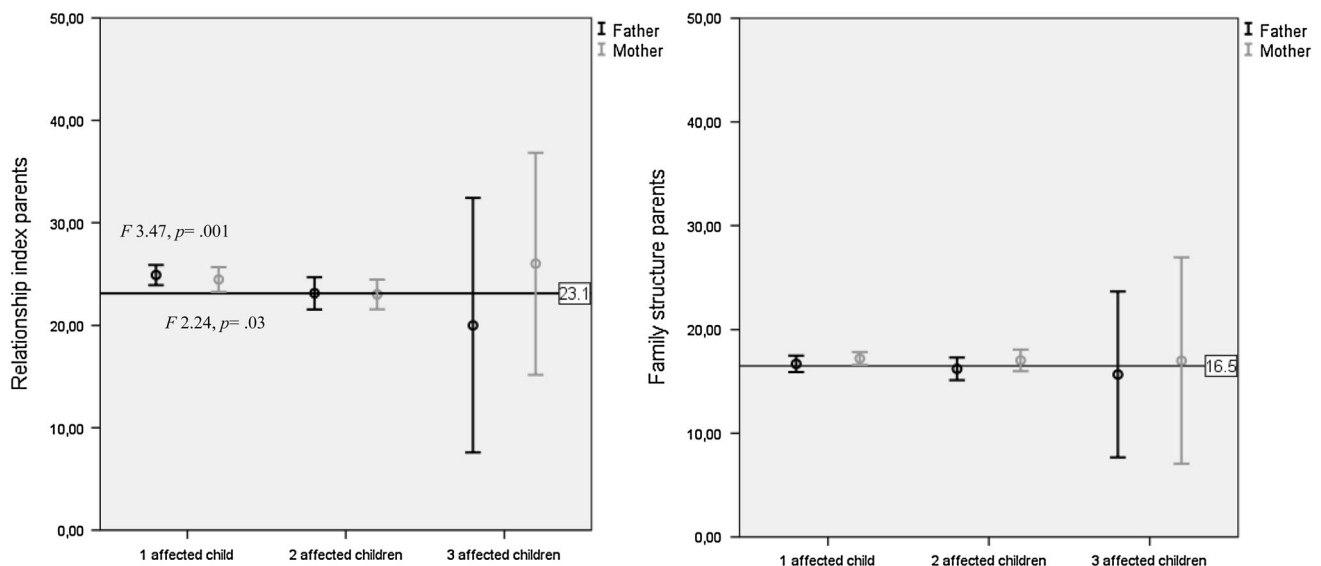
reported higher *family relationship scores* than the norm, whereas fathers and mothers of families with at least two or three affected children reported similar scores as the norm (see Fig. 1). Regarding the *family structure index*, fathers and mothers of families with one, two or three affected children reported similar scores as the norm data of the FES. ANOVA's with raising multiple affected children in a family revealed overall no significant effects of this factor on the family relationship and structure scale except that fathers reported lower family relationship scores when raising two or more affected children.

### Does Family Size Influence Family Climate?

No significant main effect of family size was found regarding both the family relationship ( $F(1,99) = 1.60, p = .21$ ) and the family structure index ( $F(1,99) = .24, p = .62$ ). In addition, regarding both the family relationship and family structure index, no three-way ( $F(1,99) = .44, p = .51$  and  $F(1,99) = .10, p = .76$ , respectively) or two-way interaction effects (all  $p$ 's were between .21 and .96) were found between family type, family size and parent. These results suggest family size (ranging from 1 to 4 children) does not impact upon family climate.

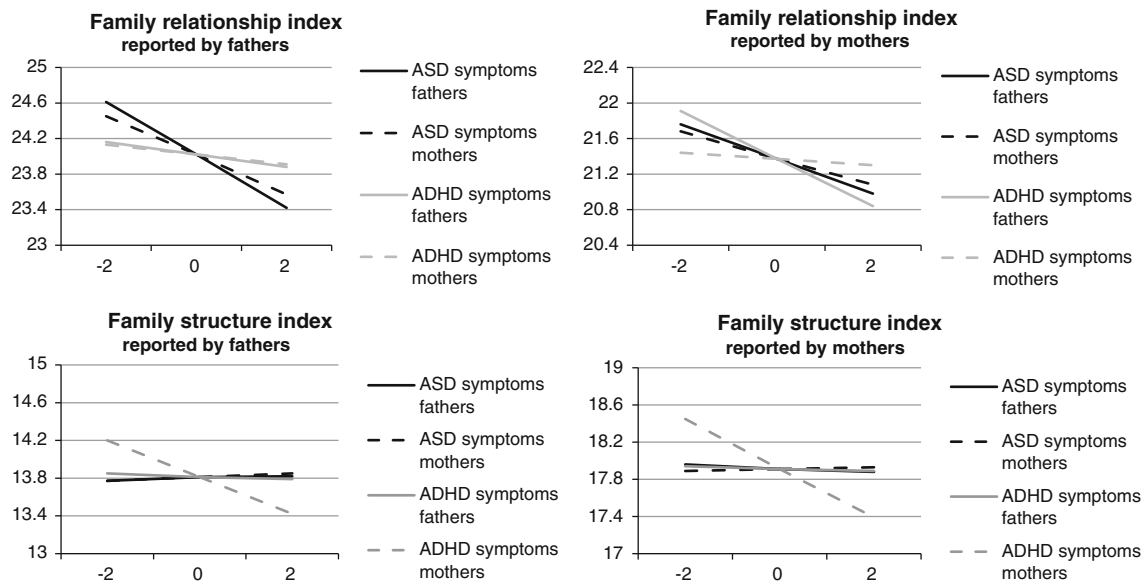
### Do Parental ASD and ADHD Symptoms (Together) Act Upon Family Climate?

ANOVAs with paternal and maternal ASD symptoms, paternal and maternal ADHD symptoms, family type and all two- and three-way interaction effects between these



**Fig. 1** Comparison of the Dutch version of the Family Environment Scale (FES) index scores between and within families with one affected child ( $N = 68$ ), two affected children ( $N = 31$ ) and three affected children ( $N = 3$ ). Norms relationship scale 23.1 (5.2) and

family structure 16.5 (3.8) for both fathers and mothers (Jansma and Coole 1996). The error bars represented the 95 % confidence interval (CI) using one-sample  $T$  test in comparison to the population norm value



**Fig. 2** Family relationship and structure index measured with the Family Environment Scale (FES) in relation to parent ASD and ADHD symptoms (N = 103). Family relationship and structure as a function of paternal and maternal symptoms of ASD and ADHD.

Higher Z-scores on the X-axis indicate more parental or maternal symptoms. Higher scores on the Y-axis indicate a higher score on the family relationship and structure index

variables were used to examine the effect of parental symptoms on family climate (family relationship and structure as reported by fathers and mothers). The most parsimonious model for the *family relationship index* for both fathers and mothers was a model with one two-way interaction between paternal and maternal ASD symptoms (as reported by fathers:  $F(1,102) = 5.98, p = .02 [\eta^2 = .06]$  and mothers:  $F(1,102) = 4.74, p = .03 [\eta^2 = .05]$ ), main effects of paternal ASD as reported by fathers:  $F(1,102) = 8.40 [\eta^2 = .08], p = .01$  and mothers:  $F(1, 102) = 5.14, p = .03 [\eta^2 = .05]$  and maternal ASD symptoms (as reported by fathers:  $F(1, 102) = 6.04, p = .02 [\eta^2 = .06]$  and mothers:  $F(1, 102) = 4.70, p = .03 [\eta^2 = .05]$ ), and paternal ADHD symptoms (only reported by mothers:  $F(1, 102) = 7.72, p = .01 [\eta^2 = .07]$ ) (see Fig. 2). The main effects of paternal and maternal ASD and paternal ADHD symptoms indicated that these symptoms were negatively related with the relationship index. The two-way interaction indicated that the effect of paternal ASD on the family relationship index was stronger than the effect of maternal ASD. The most parsimonious model for the family structure index for both fathers and mothers was a model with maternal ADHD (as reported by fathers:  $F(1,102) = 5.07, p = .03 [\eta^2 = .05]$  and mothers:  $F(1,102) = 5.81, p = .02 [\eta^2 = .06]$ ), but not paternal ADHD. This indicated that only maternal ADHD was negatively related with the family structure index.

The Effects of Possible Confounders

Overall no effects of age, IQ, anxiety and oppositional behavior were found (see supplement Table 2) regarding the family relationship and family structure index and as consequence these factors were omitted from further analyses to reduce the complexity of the results. Only two exceptions were found. First, a significant effect of age was found on the family relationship and structure index as reported by mothers, indicating improved family climate in families with older children. Second, a significant effect of oppositional behavior on the family relationship index as reported by fathers and mothers indicating increased oppositional behavior was related to poorer family relationship score. Even though age and oppositional behavior had effects on family relationships and structure (see supplementary Fig. 3), results remained identical when effects of age, IQ, anxiety or oppositional behavior were implemented in the analyses. These factors were therefore omitted from further analyses in the main analysis section to reduce the complexity of the results.

Discussion

The main aim of this study was to examine the possible influence of offspring and parental ASD and ADHD symptoms, as well as the possible effect of raising multiple

affected children, on family climate. We found no evidence that in general, compared with the Dutch norm data of the FES, family climate in families with offspring with ASD or ASD + ADHD was more negative. Raising multiple affected children negatively impacted the family climate which was not explained by family size per se. However, within ASD and ASD + ADHD families negative effects of both paternal ASD, maternal ASD and paternal ADHD symptoms on family relationships were found, as well as negative effects of maternal (but not paternal) ADHD symptoms on family structure.

The most intriguing finding of this study is that there was no evidence for a suboptimal family climate per se in families with ASD/ADHD affected offspring. This is remarkable, given that previous studies (Higgins et al. 2005; Johnston and Mash 2001; Kelly et al. 2008; Pressman et al. 2006; Schroeder and Kelley 2009) found a lower cohesion and more family conflict in families with ASD and/or ADHD affected children. However, other studies are in line with the current study, documenting on a similar positive family climate in families of children with ASD on a measure of emotional closeness (Hoffman et al. 2009). These authors suggested that the diagnosis of ASD itself may serve as a protective factor in the parent–child relationship, in that parents view the child as less responsible for his or her behavior. This view was supported by two other studies that have found that parents of children with ASD were less likely to be angry with their child—despite reporting that they were bothered by their child’s behavior—and attributed most of their child’s misbehavior to ASD symptoms, rather than their child’s personality or temperament (Montes and Halterman 2007; Whittingham et al. 2008). As suggested by Weiss et al. (2012) parent’s psychological acceptance of the child diagnosis may serve as a partial mediator between problematic child behavior and parent mental health problems. Nevertheless, a negative effect of raising multiple affected children on the family climate (family relationship index) was found, which could not be explained by family size per se. Parents of families with at least two affected children reported lower family relationship scores than families with one affected child, but not lower than Dutch norm data, suggesting that even in these families an adequate family climate appears to exist.

This study provides evidence for negative effects of parental ASD and ADHD symptoms. Both fathers and mothers reported that paternal ASD and maternal ASD symptoms were negatively related with the relationship index, aspects of the family climate that are related to parent–child relationships. In contrast, parental ASD symptoms did not influence family structure, and these contrasting effects are comparable for fathers and mothers. In contrast, paternal ADHD symptoms negatively

influenced family *relationships*, whereas maternal ADHD symptoms negatively influenced family *structure*. This may possibly be explained by the different roles and functioning in family life fulfilled by fathers and mothers: although there are fathers as primary caregivers, mothers are usually more involved in organizing day-to-day activities (making sure all children arrive on time at school, and overview what groceries are needed to be bought) compared to fathers (Craig 2006) and ADHD symptoms characterized by disorganized, chaotic lifestyle may negatively affect these day-to-day activities; In contrast, fathers are usually more involved in play, talking and recreational activities and paternal ADHD may negatively affect these activities resulting in a more negative family relationship. In other words, it seems that ASD symptoms have an negative effect on family relationships for mothers and fathers alike, whereas the negative effect of ADHD on family functioning depends on which parent is affected.

Our findings should be interpreted in the context of several limitations. First, we relied on parents’ self ratings of family climate instead of objective measurements. Our findings may be the results of parents’ perception of the family climate and may not be an accurate reflection of the actual home situation. Nevertheless, because previous studies suggest that self reported family climate correlates highly with observation of family climate in the home situation (Hinshaw et al. 1992; McHale et al. 2000; Morrongiello and House 2004; Zaslów et al. 2006), and the results were quite consistent across reporter, it is unlikely that reporter bias explained our findings. However, repeating this study with more objective home climate assessments is needed to clarify this issue. Second, it is unclear to what extent results are equally applicable to children affected with an ADHD-only diagnosis. Future research should clarify whether the results found in this study were also present in ADHD only families. Third, it is possible that some of the lower ADHD behavioral ratings may reflect successful medication treatment of these symptoms. The lack of detrimental impact on the family of the child’s ADHD symptoms may—in fact—reflect successful treatment of these symptoms. Fourth, given the evidence that parental expressiveness and control differs across cultures (Deater-Deckard et al. 2011), it will be important to examine whether the outcomes of this study are also applicable to families of non-Caucasian descent. Fifth, based on the sample size, we had 80 % power to detect effects of moderate effect size ( $d = 0.7$ ) on family climate between families with one and two affected children. Apparently, if family climate differs between these types of families, the effect is only small.

In conclusion, this is one of the first studies that reports on the influence of parental and offspring ASD and ADHD symptoms on family climate. No evidence was found for a



more negative family climate in families with ASD (+ADHD) affected offspring. Maternal and particularly paternal ASD symptoms have a negative effect on family relationships. Further, maternal ADHD symptoms appear to be negatively related to family *structure* whereas paternal ADHD symptoms were negatively related to family *relationships*. Child diagnosis (ASD or ASD + ADHD affected) does not appear to influence family climate, but raising two or more affected children does have a negative effect on the family relationship compared to raising one affected child (albeit the family climate is still not worse compared to Dutch norms). The results suggest that, although parental ASD or ADHD and raising multiple affected children were negatively related to family climate, still a positive family climate were found in families with ASD (+ADHD) affected offspring.

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