Pediatric RESEARCH

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CLINICAL RESEARCH ARTICLE

An exploratory study of parent–child association in sensory modulation disorder involving ADHD-related symptoms

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BACKGROUND: Sensory modulation disorder (SMD) and attention deficit hyperactivity disorder (ADHD) can co-occur and have overlapping symptoms, thus challenging practitioners. This study aimed to phenotypically explore parent–child associations in SMD, and the interplay between SMD- and ADHD-related symptoms in children with SMD and their parents.

METHODS: A cross-sectional study examined 70 parents ($n = 35$ mothers; $n = 35$ fathers) and their 35 children with and without SMD, aged 4–6 years. Parents completed care-giver reports: The Short Sensory Profile (SSP) and the ADHD Rating Scale, and self-reports: The Sensory Responsiveness Questionnaire (SRQ) and the ADHD Self-Report Scale (ASRS).

RESULTS: In the entire sample, we found a mother–offspring correlation between SSP and SRQ-Aversive scores ($rs = -0.68$; $p < 0.001$), but no such father–offspring correlation. However, when testing the ADHD Rating Scale and ASRS scores, we found correlations between mothers and offspring ($rs = 0.54$, $p = 0.0008$), and between fathers and offspring ($rs = 0.34$, $p = 0.0494$). In the entire sample a high correlation was found between SSP and ADHD Rating Scale scores ($rs = -0.837$, $p < 0.001$). We further found a high correlation in mothers ($rs = 0.70$, $p < 0.001$), and a moderate correlation in fathers ($rs = 0.40$, $p = 0.019$) between SRQ-Aversive and ASRS scores.

CONCLUSIONS: Novel findings reveal that parents–offspring heritability patterns differ in both these related conditions. These may contribute to familial practice and research.

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INTRODUCTION

Attention deficit hyperactivity disorder (ADHD) is one of the most common childhood-onset neurodevelopmental disorders, impacting about 5% of school-age children worldwide.[1,2] The population affected is heterogeneous and shows considerable variation in the degree of symptoms, as well as in the frequent presence of associated comorbidities.[2,3] ADHD clinical guidelines[4] as well as recent findings emphasize the importance of assessing ADHD and related symptoms in preschoolers, given the early intervention benefits in this chronic disorder.[5]

Sensory modulation disorder (SMD), a type of sensory processing disorder, is a neurodevelopmental condition impacting single or multiple sensory systems, and affects the capacity to regulate responses to sensory input in a graded and adaptive manner.[6,7] Consequently, SMD interferes with quality of life and participation in daily activities and functions.[8,9] This disorder is characterized by sensory under-responsiveness (SUR) associated with decreased or delayed responses to stimulation, and sensory over-responsiveness (SOR) in which innocuous sensations are perceived as abnormally irritating, unpleasant,[6,7] or painful.[10,11] While SMD occurs in at least 70% of individuals with autism[12] and at a similar rate in ADHD,[13] the estimated probability in the otherwise healthy general pediatric and adult population is 5–16%.[11,14,15]

Both ADHD and SMD have been controversial diagnoses in the public and professional domains, partially due to the constraints of categorical behavioral diagnosis.[2] However, neuropsychological, neuroanatomical, and neurophysiological studies have clearly demonstrated that ADHD symptoms are the manifestation of abnormalities in large-scale brain networks,[16,17] while sensory processing disorders have not yet been anchored within a neuroanatomical framework. Clinically, hyperactivity, distractibility, and impulsivity are manifested in those with SMD,[18] yet these are also among the core symptoms of ADHD.[19] This overlap of symptoms makes it difficult to discriminate between these two often co-occurring clinical conditions.[20] Thus, differentiating the two is crucial for the understanding of the phenotype origin and for guiding best practice evaluation and treatment.[18]

In order to discriminate more clearly SMD from ADHD, we chose to explore SMD heritability. Only recently have SOR profile tests been conducted on parents and their adolescent offspring, revealing only weak associations.[21] Conversely, for ADHD, its heritability has been estimated to be approximately 77%,[22] and studies have demonstrated that either parent with ADHD is a main risk factor for offspring with ADHD.[23] When considering any heritable interaction between the SMD and ADHD, high incidents of SMD have been reported in children with ADHD,[13] which might enhance the behavioral difficulties and distress observed in a significant number of children.[18] However, to the best of our knowledge, there is no published study exploring the interplay between SMD- and ADHD-related symptoms in children with SMD

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and their parents. Therefore, the aim of the current study is to explore the parent–child associations in SMD, and to investigate the SMD-ADHD-related symptoms interplay between children with SMD and their parents.

METHODS

The experimental protocol was approved by the institutional Investigational Review Board; the study protocol conformed to the ethical guidelines of the 1975 Declaration of Helsinki. Informed consent was obtained from all participants. This was a cross-sectional study.

Participants

A convenience sample was recruited via child development centers under a public health service, and control participants were enrolled using the snowball sampling method. All participants were living in the same region. Parents' age, years of education, employment status, income level, and general health, as well as the offspring's age, sex, developmental history, and current health status were assessed. This study was conducted between March 2013 and November 2014. The study group comprised of children on a waiting list for occupational therapy treatment who scored lower than the cut-off for typical sensory modulation on the Short Sensory Profile (SSP) total score (<155). The control group included children who scored in the typical range for an SSP total score (>155).[24] Inclusion criteria for both groups stipulated that the children were living with their biological parents and enrolled in educational systems for typically developing children, and that both parents had no language barriers. Exclusion criteria for both groups included a diagnosis of ADHD; neurological, psychiatric, or genetic syndromes, or who had siblings with any such syndromes. Exclusion criteria for the control group included receiving current or past developmental therapy or having a sibling identified with SMD.

Instrumentation

The SSP[24] is a care-giver questionnaire aimed as a diagnostic tool for SMD in children aged 3–10 years. It consists of 38 statements, arranged into seven categories: tactile sensitivity, taste-smell sensitivity, under-responsive-sensation seeking, auditory filtering, visual-auditory sensitivity, low energy, and movement sensitivity. Care-givers respond on a five-point Likert scale (1 = always to 5 = never). A total score is calculated by adding the points assigned for each item. Total scores of <141 indicate a definite difference; 142–154 indicate a probable difference; and 155–190 indicate typical sensory modulation. Reliability has been demonstrated using Cronbach's α (range: 0.70–0.90). Construct validity has been established via the known-group procedure, factor analysis (range $r = 0.25$–0.76) and by using electrodermal response testing.

ADHD Rating Scale:[25] The ADHD diagnostic questionnaire is an 18-item scale with one item for each of the 18 symptoms contained in the Diagnostic and Statistical Manual of Mental Disorder (DSM) diagnosis of ADHD for children aged 4–17 years.[4] Each item is scored on a 0–3 scale: 0 = none (never or rarely); 1 = mild (sometimes); 2 = moderate (often); 3 = severe (very often). The total score is computed as the sum of the scores on each of the 18 items. To determine ADHD at least six items need to be assigned a score of 2 or 3.[26]

The Sensory Responsiveness Questionnaire-Intensity Scale (SRQ-IS)[27] is a self-report questionnaire assessing responses to daily sensations, and is used to clinically identify SMD in adults. The SRQ-IS presents a set of 58 items that represent typical scenarios encountered occasionally throughout daily life. Each scenario involves one sensory stimulus in one modality including auditory, visual, gustatory, olfactory, vestibular, and somatosensory stimuli, excluding pain. Items are worded in a manner that attributes a hedonic or aversive valence to the situation, yielding two scores: SRQ-Hedonic, assessing SUR (26 items) and SRQ-Aversive, assessing SOR (32 items). Each item is scored on a 5-point Likert scale: 1 (not at all) to 5 (very much). The two total scores are calculated for each scale separately by adding the points assigned for each item and then dividing the total sum by the number of responded items. For both scales, scores higher than the normal mean cut-off score of + 2 standard deviations (SD) indicate SOR (SRQ-Aversive 1.87 + 0.52) or SUR (SRQ-Hedonic 2.10 + 0.66). Two SD cut-offs were applied to ensure conservative group categorization. The SRQ has been demonstrated to have content, criterion, and construct validity, as well as test-retest reliability ($r = 0.71$–0.84; $p < 0.001$–0.005).

The WHO Adult ADHD Self-Report Scale (ASRS) Symptom Checklist[28] is a standardized self-report questionnaire, assessing symptoms described by the DSM-IV as being commonly observed in adult ADHD. The ASRS version 1.1 comprises 18 items reflecting DSM-IV-TR criteria to assess inattention (9 items) and hyperactivity-impulsivity (9 items). Each item is scored on a 5-point Likert scale from 0 (never) to 4 (very often). In this study, the short form which includes questions 1–6 (Part A) was used for analyses as this has demonstrated good properties of sensitivity (68.7%), specificity (99.5%), and symptom concordance (0.8). Good internal consistency (0.63–0.72) and test–retest reliability (Pearson's correlations $r = 0.58$–0.77) has been established. Using Part A, screening for ADHD requires at least four items to be rated 3 or 4 (but they can also be rated 2 if scored for items 1–3).[28]

Procedure

Negating an ADHD diagnosis was carried out before enrolling in this study by a pediatrician specializing in Developmental Neurology. A routine clinical interview and evaluation was conducted with the child and parents on admission, before referring the child to occupational therapy. Recruitment for the study group followed an initial screening of children on a waiting list for occupational therapy treatment, who matched the inclusion criteria and were suspected of having SMD following the pediatrician's referral. Parents were initially approached by phone, and if eligible, received a mailed letter explaining the purpose of the study and its procedures, an informed consent form, and the SSP, ADHD symptoms, SRQ, ASRS, and medical and demographic questionnaires, in a return-addressed and stamped envelope. Initially the SSP was scored and children scoring a total score below <154 were included in the study group after verifying their health condition through a medical questionnaire and a short telephone interview.

The control group was similarly recruited using a snowball sampling of children living in the same region as those in the study group. The control group required an SSP total score of a typical sensory profile (≥155).

Data analysis

Statistical analyses were performed with SAS® V9.4 (SAS Institute, Cary, NC, USA). Study data is summarized with descriptive statistics presented as a mean, SD, median, and interquartile ranges (IQR) for continuous variables and as frequencies and percentages for categorical variables. Groups were compared using the Wilcoxon's two-sample test for continuous variables and Fisher's exact test for categorical variables (due to the small sample size in each group). Correlations between continuous variables were evaluated with Spearman's correlation coefficients (rs). The associations between SMD and ADHD conditions between the children and parent variables were evaluated with Fisher's exact test. Repeated-measures analysis of variances were used to compare SRQ scores between the groups within mothers and fathers (via the group by parent interaction term of the model), to take the within-family correlation into consideration. LSmean differences are compared between the groups within mothers and fathers. All statistical tests were two-sided tests at a 5% level of significance. Nominal p values are presented.

Table 1. Group differences (SMD vs. control) in demographic and clinical characteristics

Characteristic	SMD group ($n = 15$)	Control group ($n = 20$)	*P* value
Educational stage, *n* (%)			
Pre-kindergarten	8 (53.33%)	8 (40%)	
Kindergarten	7 (46.67%)	9 (45%)	0.32
Elementary school	0 (0%)	3 (15%)	
Birth order, *n* (%)			
Eldest	6 (40%)	9 (45%)	
Middle	6 (40%)	9 (45%)	0.83
Youngest	2 (13.3%)	2 (10%)	
Other	1 (6.67%)	0 (0%)	
Complications during pregnancy, *n* (%)			
Yes	4 (26.67%)	2 (10%)	0.37
No	11 (73.33%)	18 (90%)	
Labor week, *n* (%)			
Premature (before 37 weeks)	3 (20%)	1 (5.26%)	0.23
Mature	13 (80%)	18 (94.74%)	
Labor type, *n* (%)			
Natural	9 (60%)	18 (90%)	
Cesarean section	4 (26.67%)	1 (5%)	0.14
Vacuum, *n* (%)	2 (13.33%)	1 (5%)	
General health, *n* (%)			
Healthy	13 (86.87%)	19 (95%)	0.56
Not healthy	2 (13.33%)	1 (5%)	
Medication use on daily basis, *n* (%)			
Yes	0 (0%)	0 (0%)	0.18
No	15 (100%)	20 (100%)	

SMD sensory modulation disorder

RESULTS

Demographic characteristics

Seventy parents (35 mothers) and their 35 children participated in this study, 70% of the 50 families that were approached. The SMD group was comprised of 15 children [mean (SD) age: 4.8 (0.57) years, 10 boys], while the control group included 20 children [mean (SD) age: 5.3 (0.78) years, 11 boys]. A statistically significant group difference was found for the SSP scores ($p < 0.001$) [SMD vs. control: median (IQR): 126 (38.0); 176 (14.5), respectively]. No group differences were found for age and sex distribution ($p = 0.0619$ and 0.4857, respectively), nor for the children's developmental and medical history variables (e.g., educational stage, complications during pregnancy, pregnancy week in which labor occurred, labor type, use of daily medications, general health), see Table 1. Table 2 demonstrates no statistical group differences in most of the parents' demographic variables (e.g., age, education, employment status, and general health), except for lower income, which was more frequent among mothers of children with SMD (Table 2).

Associations between SMD and ADHD symptoms in children and parents

SSP and ADHD rating scale scores were found highly correlated in the entire sample among children ($N = 35$) (rs $= -0.837$, $p < 0.001$; Fig. 1a). Additionally, in the study group of SMD children, ADHD rating scale scores were significantly higher [median (IQR): 27.0 (25.0)] than the scores of the control group children [median (IQR): 5.0 (8.0); $p = 0.0001$]. Mothers demonstrated a statistically significant high correlation between the SRQ-Aversive and ASRS scores (rs $= 0.70$, $p < 0.001$; Fig. 1c), but not between the SRQ-Hedonic and ASRS scores (rs $= 0.23$, $p = 0.18$; Fig. 1b). Fathers similarly demonstrated a statistically significant moderate correlation between SRQ-Aversive and ASRS scores (rs $= 0.40$, $p = 0.019$, Fig. 1c), but not between SRQ-Hedonic and ASRS scores (rs $= 0.17$, $p = 0.34$; Fig. 1b).

SMD among parents and their children

Table 1 displays the parents' descriptive statistics. Parent–offspring correlations between SRQ and SSP scores in the entire sample were found between mothers' SRQ-Aversive scores and their offspring's SSP scores (rs $= -0.68$; $p < 0.001$), with low-moderate correlations between mothers' SRQ-Hedonic scores and their offspring's SSP scores (rs $= -0.45$; $p = 0.007$) (Fig. 2a, b). No such SRQ-SSP correlations were found between fathers and their offspring (SRQ-Aversive, rs $= -0.08$; $p = 0.64$; SRQ-Hedonic, rs $= 0.08$; $p = 0.67$; Fig. 2a, b). A significant difference between groups (children with SMD vs. control children) in the mothers' SRQ-Aversive scores was found, but not in the fathers' SRQ scores: [SRQ-Aversive: mothers $t(33) = 0.6155$, $p = 0.0002$ cf. fathers $t(33) = 0.0989$, $p = 0.5011$; SRQ-Hedonic: mothers $t(33) = 2.45$, $p = 0.0197$ cf. fathers $t(33) = -1.17$, $p = 0.2493$] (Table 3). Furthermore, a significant group difference (children with SMD vs. control children) was found in the distribution of mothers' SMD condition (SOR, SUR vs. non-SMD) ($p = 0.0221$), indicating a higher frequency of mothers with SMD-SOR having children with SMD (77.8%), compared to SMD-SOR mothers having non-SMD children (22.2%). In other words, only 10.0% of non-SMD children (control group) had a mother with SMD-SOR vs. 46.7% of SMD children. Among fathers, no such difference was found ($p = 0.6446$).

ADHD symptoms among parents and their children

In the study group 66.7% of the children were found to have ADHD reported symptomology, while no children in the control group had ADHD symptomology ($p < 0.0001$). In the entire study sample of children, significant low-moderate correlations were found in the level of ADHD symptoms between children (ADHD Rating Scale) and their parents' ASRS scores: mother–offspring (rs $= 0.54$, $p = 0.0008$); father–offspring (rs $= 0.34$, $p = 0.0494$) (Fig. 3). No statistically significant differences were found between mother and father ADHD scores within offspring with ADHD symptoms vs. those without (child ADHD status by parent interaction term: $F(1,33) = 0.0$; $p = 1.00$). Furthermore, 14.3% ($n = 5$) of mothers and 11.3% ($n = 4$) of fathers were found to have ADHD out of the entire sample ($p = 1.00$). (These represent eight families as one child had both parents with ADHD.) Of note, all fathers with ADHD had children in the study group, as opposed to mothers with ADHD who were less likely to have a child in the study group (one out of five had a child in the study group). A statistically significant association was found between offspring SMD status and paternal ADHD status ($p = 0.0261$).

DISCUSSION

To the best of our knowledge, this is the first study to explore the interplay between SMD- and ADHD-related symptoms in children with SMD and their parents. Testing the associations between SMD- and ADHD-related symptoms within each group of subjects (i.e., children, mothers, fathers) found that both conditions are strongly correlated in children and mothers, and moderately correlated in children and fathers. Of note, in both parents, ADHD-related symptoms correlated with only the over-responsive type of SMD, but not with the under-responsive type of SMD. Furthermore, findings indicate that most children with SMD have mothers with SMD, but not fathers with SMD. Of note, this pattern of parent–child association differs from the one we found for the

Table 2. Group differences (children: SMD vs. control) in their parents' demographic and background characteristics

Characteristic	Mothers			Fathers		
	Child with SMD ($n = 15$)	Child without SMD ($n = 20$)	*P* value	Child with SMD ($n = 15$)	Child without SMD ($n = 20$)	*P* value
Age (years)						
Median (IQR)	33 (7)	33 (7.5)	0.54	36 (8)	35 (6.5)	0.42
Mean (SD)	33.6 (4.16)	34.5 (4.05)		37.3 (5.7)	36 (3.68)	
Education (years)						
Median (IQR)	16 (5)	16 (3.5)	0.18	15 (5)	15 (2)	0.60
Mean (SD)	15.7 (2.66)	16.8 (2.07)		14.9 (2.43)	15.4 (2.83)	
Income, *n* (%)						
A lot above average	0 (0%)	3 (15%)	**0.034**	1 (7.69%)	2 (10%)	0.43
Beyond average	0 (0%)	3 (15%)		5 (38.46%)	12 (60%)	
Average	7 (46.67%)	11 (55%)		4 (30.77%)	5 (25%)	
Below average	8 (53.33%)	3 (15%)		3 (23.08%)	1 (20%)	
Employment status, *n* (%)						
Working	15 (100%)	19 (95%)	1.00	15 (100%)	19 (95%)	1.00
Not working	0 (0%)	1 (5%)		0 (0%)	1 (5%)	
General health, *n* (%)						
Healthy	14 (93.33%)	20 (100%)	0.43	15 (100%)	20 (100%)	0.63
Not healthy	1 (6.67%)	0 (0%)		0 (0%)	0 (0%)	
Diagnosis of Medical illness, *n* (%)						
Yes	4 (26.67%)	2 (10%)	0.37	3 (20%	2 (10%)	0.63
No	11 (73.33%)	18 (90%)		12 (80%)	18 (90%)	

SMD sensory modulation disorder, *IQR* interquartile range
Bold values denote statistically significant group differences

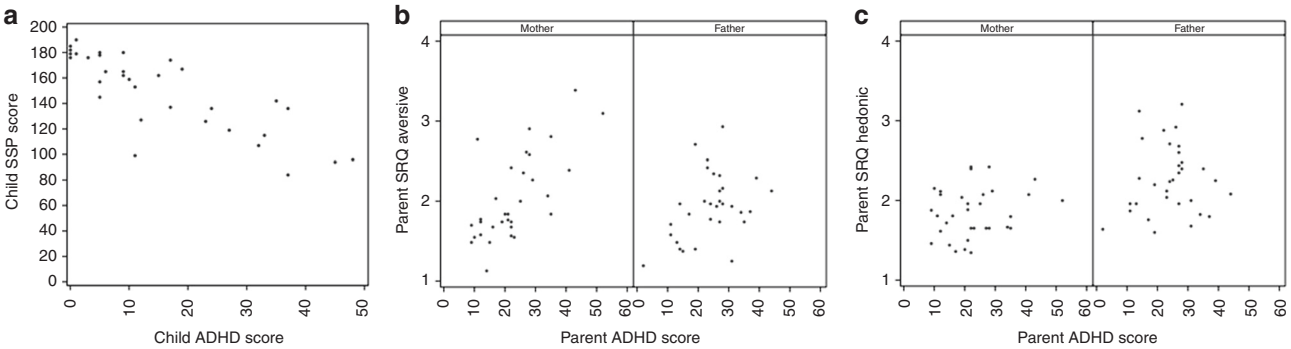

Fig. 1 **a–c** The relation between sensory modulation disorder (SMD) and attention deficit hyperactivity disorder (ADHD) symptoms in children (**a**) and their parents (**b**, **c**) *SMD, sensory modulation disorder; Children ADHD score was obtained using the ADHD symptoms scale; Father/Mother ADHD score was obtained using ASRS—Adult ADHD Self-Report Scale. Father/mother SMD score was obtained using SRQ—Sensory Responsiveness Questionnaire; children SMD score was obtained using the SSP— Short Sensory Profile

ADHD-related symptomology. Namely, no difference was found in the ADHD distribution of both parents between the children's groups (parents of children with SMD vs. parents of control children).

Moderate to strong relationships were found between the SMD scores in mothers and their children, in both SRQ-Aversive and SRQ-Hedonic scores, but not in fathers. Conversely, when testing the relationship between each parent and offspring on the ADHD related symptom level, we found low to moderate correlations in both parents. Importantly, our findings demonstrate that SMD- and ADHD-related symptoms co-occur in children with SMD, with each condition constituting a different parent–child association pattern. This alludes to possible separate endophenotypes for each condition that nevertheless may be related.

Since there are no previous studies reporting on parent–offspring relationships in SMD, we can only examine our findings against such reports on ADHD. Of note, children participating in this study did not have an ADHD diagnosis, but were tested for ADHD-related symptomology solely. This study found moderate correlations of ADHD-related symptoms in both parents and their children. Previous reports consistently demonstrate that both parents' ADHD symptoms similarly predict higher rates of offspring having internalizing and externalizing behaviors.[29] A meta-analysis of 22 studies found that genetic factors

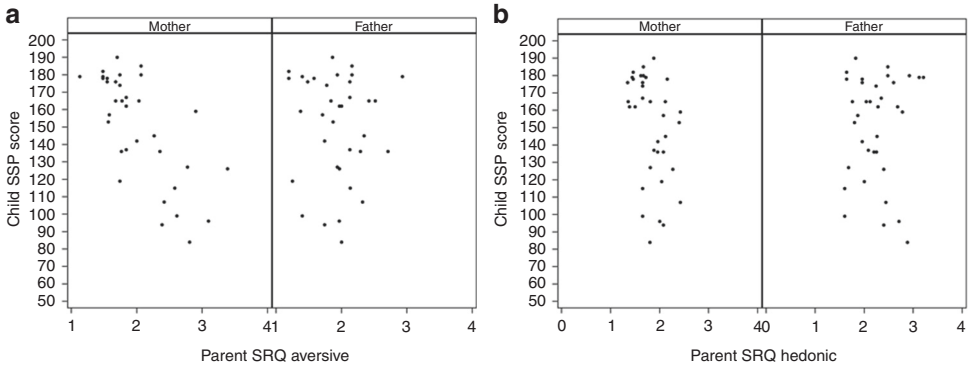

Fig. 2 **a**, **b** Parent–child associations in SMD scores. SMD, sensory modulation disorder; SRQ, Sensory Responsiveness Questionnaire. Father/mother SMD score was obtained using SRQ; children SMD score was obtained using the SSP—Short Sensory ProfileBold values denote statistically significant group differences

Table 3. SRQ-IS and ASRS scores for parents according to the children groups

Measures	Mothers ($N = 35$)				Fathers ($N = 35$)			
	Children with SMD ($N = 15$)		Children without SMD ($N = 20$)		Children with SMD ($N = 15$)		Children without SMD ($N = 20$)	
	Median	IQR	Median	IQR	Median	IQR	Median	IQR
SRQ-Hedonic	2.0	0.3	1.7	0.4	2.2	0.6	2.3	0.7
SRQ-Aversive	2.4	0.9	1.7	0.3	2.0	0.5	1.9	0.6
ASRS	26.0	14.0	16.5	10.5	27.5	11.0	23.0	13.5

SMD sensory modulation disorder, *IQR* interquartile range, *SRQ* sensory responsiveness questionnaire, *ASRS* Adult ADHD Self-Report Scale

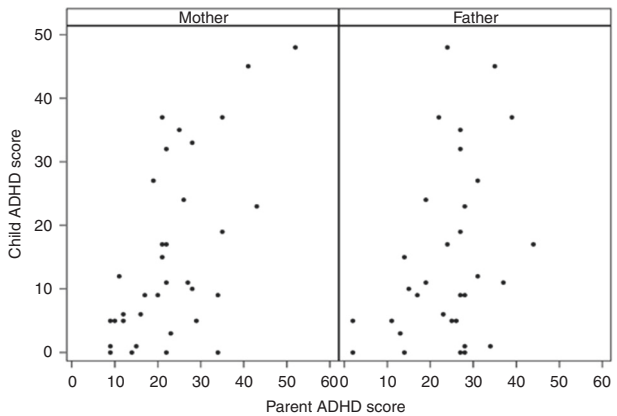

Fig. 3 The relation between attention deficit hyperactivity disorder (ADHD) symptoms in children and their parents. Children ADHD score was obtained using the ADHD symptoms scale; Father/Mother ADHD score was obtained using ASRS—Adult ADHD Self-Report Scale

accounted for 73 and 71% of the variance in hyperactivity and inattention, respectively.[30] However, there is some inconsistency in the reported rates of ADHD between parents and their children,[31] meaning that the ADHD risk transmission from parent to offspring is still not clearly understood.[32,33]

This study reveals that parents–offspring phenotypic associations differs between SMD- and ADHD-related symptoms. These findings could potentially support the differential diagnosis necessary for a more personalized preventive and therapeutic intervention as well as lead to future genetic research. Parent-of-origin (PEO) refers to conditions wherein the offspring phenotype is not equally contributed by fathers and mothers.[34] It could be suggested that genomic imprinting, an epigenetic PEO effect in which specific genes are active only when inherited from the father or the mother, may play an important role in SMD. The PEO hypothesis may offer a new direction for gaining insight into the molecular basis of SMD, a field that has not yet been explored. Other genetic mechanisms that may account for this differential influence between parents, though beyond the scope of this exploratory study, may include: (i) mitochondrial genome and sex chromosomes; (ii) genomic imprinting; and (iii) effects of the maternal genome on intrauterine environment and fetus (maternal effects).[35]

CONCLUSIONS

This is the first study to report on the SMD parent–child heritability pattern as well as on the interplay between SMD- and ADHD-related symptoms in parents and their children with SMD, compared to controls. Given the similar phenotypic presentation of both ADHD and SMD, findings may provide new directions for research and practice, both in SMD and in ADHD, and contribute to ADHD-SMD differential diagnosis. Further studies are required to confirm this study's novel results.

This study has some limitations; as an exploratory study, findings warrant further validation extending this design to a larger sample. Future studies may further investigate parent–offspring associations when considering children with either SMD or ADHD.

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AUTHOR CONTRIBUTIONS

M.K.-A.—Substantial contributions to conception and design, and acquisition of data. I.B—Drafting the article, revising it critically for important intellectual content, and final approval of the version to be published. A.R.—Analysis and interpretation of data. T.B.-S.—Substantial contributions to conception and design, analysis, and interpretation of data; drafting the article, revising it critically for important intellectual content, and final approval of the version to be published.

ADDITIONAL INFORMATION

Competing interests: The authors declare no competing interests.

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