Is there a correlation between ADHD symptom expression between parents and children?

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Abstract

BACKGROUND: Attention-deficit hyperactivity disorder (ADHD) is one of the most common mental health disorders in childhood; symptoms persist into adulthood in a majority of patients. It is among the most heritable of psychiatric disorders with a high risk for familial aggregation and has been linked in adulthood with impairment across a variety of domains, including parenting. Parental gender, ADHD status and symptom expression could be related to the severity of ADHD symptoms in the child.

METHODS: We used prospective, observational study of clinical group of 30 children with diagnosed ADHD and control group of 37 healthy subjects. Only children with both biological parents available were included. Data on ADHD symptomatology for all subjects was gathered by a set of clinical tools (CBCL1991, TRF1991, WURS, self-report scale modified from DSM IV). Under the assumption that ADHD is a dimensional disorder, raw scores from questionnaires were used as they display the complete range of values.

RESULTS: Clinical group showed higher values in all areas of children symptomatology, the same was observed for parental ADHD symptomatology. Significant correlation was found between children and paternal current ADHD symptomatology in the clinical group. This was not confirmed for mothers.

CONCLUSION: Our study stresses an importance of screening for ADHD symptoms in parents of clinically referred children with ADHD as the correlation between severity of paternal and child's ADHD symptoms was confirmed. Our results stress the importance of including the father into the clinical assessment.

Abbreviation:

ADHD	- Attention Deficit Hyperactivity Disorder
WISC	- Wechsler Intelligence Scale for Children
DSM	- Diagnostic and Statistical Manual of Mental Disorders
CBCL	- Child Behaviour Checklist
TRF	- Teacher Report Form
WURS	- Wender Utah Rating Scale
CBCL-AP	- CBCL - Attention Problem subscale
CBCL-AB	- CBCL - Aggressive Behaviour subscale
CBCL-DB	- CBCL - Delinquent Behaviour subscale
CBCL-EXT	- CBCL - Externalizing Problem subscale
TRF-AP	- TRF - Attention Problem subscale
TRF-AB	- TRF - Aggressive Behaviour subscale
TRF-DB	- TRF - Delinquent Behaviour subscale
TRF-EXT	- TRF - Externalizing Problem subscale
ANCOVA	- Analysis of Covariance

ORIGINAL ARTICLE

INTRODUCTION

Attention-deficit hyperactivity disorder (ADHD) is one of the most common mental health disorder of childhood, affecting approximately three to seven percent of all school-age children (Swanson *et al.* 1998; Newcorn *et al.* 2001). ADHD symptoms persist into adulthood in a majority of patients (Biederman *et al.* 1998; Faraone *et al.* 2000), the prevalence of ADHD in adults was estimated to be four percent (Kessler *et al.* 2006). High levels of comorbidities were confirmed in many studies (Biederman *et al.* 2005; Goos *et al.* 2007; Spencer *et al.* 1999; Connor *et al.* 2003; Wilens *et al.* 2002).

ADHD is not a categorical disorder as symptoms can also be found in healthy subjects; disorder is best viewed as the extreme of a behaviour that varies genetically through the entire population rather than a disorder with discrete determinants (Levy *et al.* 1997; Larsson *et al.* 2012).

ADHD is among the most heritable of psychiatric disorders, a mean heritability estimate of 76% was reported by Faraone *et al.* (2005). Parents of the ADHD children have two- to eightfold increase in the risk for ADHD with similarly elevated risk among the siblings of ADHD subjects (Faraone 2004). A variety of genetic and non-genetic risk factors contribute to the disorder, but familial transmission is not well understood. Parents may contribute different ADHD relative quantity and quality risk factors due to the genetic and environmental factors and the combination of the two. Parenting responses to child's ADHD could contribute to maintenance of ADHD symptoms or even exacerbation to major disruptive disorder (Johnston & Jassy 2007). Co-occurrence of ADHD symptoms in a parent might interfere with parenting practices even more. Therefore, it is reasonable to assume that combined burden of genetic predisposition to ADHD and response of parents suffering from ADHD as well is likely to lead to increased expression of ADHD symptoms in the child.

AIM

Aim of the study was to investigate potential correlation between reported ADHD symptoms in children and their biological parents in clinical and control group of children. We speculated that parental symptom expression would confer risk for the severity of proband's ADHD presentation.

METHODS

Ethical approval

The study was approved by the The National Medical Ethics Committee of the Republic of Slovenia.

Participants

30 children and both biological parents were included in the clinical sample. Children were referred for clinical assessment to child and adolescent psychiatrists from different settings (parents, pediatrician, school workers, other child carer, etc.). All of the eligible children with diagnosed ADHD within the observed time period were included. Inclusion criteria were diagnosis of ADHD based on clinical criteria, age between 7 and 10 years, IQ > 75 (WISC III), pharmacologically naïve child and both biological parents available. Exclusion criteria included major medical diagnoses, neurological problems (cerebral palsy, epilepsy, etc.) and neuropsychiatric disorders (autism spectrum disorders, psychosis, bipolar disorders, major depressive disorder).

The control group consisted of 37 children enrolled from different primary schools by teachers. Inclusion criteria were no history of psychological or psychiatric assessment (including ADHD), age between 7 and 10 years, a child should be an average functioning subject without severe learning disabilities and both biological parents available.

Informed consent for participation in the study was obtained from both parents for themselves and for their children.

Assessments

Questionnaires

Children in the clinical sample were assessed by child and adolescent psychiatrist and clinical psychologist. All children met diagnostic criteria DSM IV for ADHD (DSM-IV 2000) and IQ >75 (WISC III). Parents were given the Child Behaviour Checklist (for ages 4–18) for parents (1991)(CBCL) (Achenbach 1991a) to assess the symptoms in the child and Wender Utah Rating Scale (WURS) questionnaire and DSM IV ADHD symptoms checklist to report their symptoms. Child's teacher was sent the Teacher Report Form (1991) for teachers (TRF) (Achenbach 1991b).

In the control group the child's teacher fulfilled the TRF for the pupil and provided both parents with CBCL for parents, WURS and DSM IV checklist.

Questionnaire evaluation

Parental ratings were obtained from Attention Problem (AP) subscale of the CBCL/4–18 (CBCL-AP). The scale includes 11 items of inattention, hyperactivity and impulsivity; behavioural problems were rated for occurrence during the last 6 months.

Teacher ratings were obtained from Attention Problem (AP) subscale of the TRF (TRF-AP). Teachers were instructed to rate the child's behaviour over the preceding 2 months. The AP subscale of the TRF consists of 20 items for the ADHD related symptoms.

Ratings were also obtained from CBCL and TRF subscales for Delinquent Behaviour (CBCL-DB and TRF-DB) and Aggressive Behaviour (CBCL-AB and TRF-AB). Finally, the sums of Attention Problem,

Delinquent and Aggressive Behaviour scores for both checklists were calculated as Externalizing Problem score (CBCL-EXT and TRF-EXT).

The diagnosis of ADHD in adults is challenging because symptoms of inattention, hyperactivity and impulsivity must have been present and causing impairments in childhood (officially before 7 years of age). Retrospective recall bias and elapsed time necessarily limit the accuracy of information on behaviour in childhood. This makes diagnosis in adults difficult. The important diagnostic tool are different rating scales. One of them is Wender-Utah Rating Scale, which retrospectively assesses ADHD-relevant childhood behaviours and symptoms in adults. This scale consists of 61 items reflecting signs and symptoms characteristic for ADHD. Ward *et al.* (1993) found that 25 items were shown to be specific for diagnosing adults with ADHD. Murphy and Schachar (2000) confirmed that adults can give a true account of their childhood and current symptoms of ADHD. Taylor *et al.* (2011) did a systematic review on the scales for the identification of adults with ADHD and concluded that WURS had robust psychometric statistics and content validity.

Current ADHD symptoms were assessed by self-report scale modified from the DSM IV (DSM-IV 2000, Weiss & Murray 2003). Scale contains 18 ADHD symptoms from DSM IV, each of which is rated on its frequency in the past 6 months using a 4-grade scale, from 0 (never or not at all) to 3 (very often or very much).

Under the assumption that ADHD is a dimensional disorder, raw scores from CBCL, TRF, WURS and DSM IV checklists were used as they display the complete range of values.

Statistical analysis

Numerical variables were described as means +/– standard deviations (SD). Univariate comparisons between groups were performed by one-way analysis of variance (ANOVA) and Chi-square test, as appropriate. Kolmogorov-Smirnov test was used to check for assumed normal data distribution prior to using parametric tests. Correlations between checklist scores/subscores of CBCL, TRF, WURS and DSM-IV were evaluated by Spearman rho correlation coefficient. To determine the relative effect of evaluated factors (age, gender, checklist scores/subscores) on expression of ADHD symp-

Tab. 1. Descriptive statistics of patient characteristics and evaluated parameters in the clinical and control groups.

	Clinical group		Control group		Sig. (2-tailed)	
	(n=30)	Males (n=24)	(n=37)	Males (n=18)	Clinical vs. control group	Clinical (males) vs. control (males) group
Age*	8.23±1.33	8.21±1.32	8.76±1.04	9.17±1.04	NS (0.08)	0.05
No. gender (%)						
M	24 (80.0)		18 (48.6)		0.008	
F	6 (20.0)		19 (41.4)			
CBCL subscale*						
CBCL-AP	9.87±3.59	10.33±3.17	2.19±2.15	2.11±1.61	<0.001	<0.001
CBCL-AB	12.70±6.20	14.33±5.56	4.14±3.47	3.39±3.35	<0.001	<0.001
CBCL-DB	2.67±1.95	2.83±2.12	1.22±1.90	1.50±2.50	<0.001	<0.001
CBCL-EXT	15.37±7.52	17.17±7.04	5.35±4.21	4.89±4.14	<0.001	<0.001
TRF subscale*						
TRF-AP	18.37±7.19	18.75±7.59	1.89±2.34	2.17±2.96	<0.001	<0.001
TRF-AB	18.17±12.71	19.58±12.55	2.46±4.25	2.50±3.54	<0.001	<0.001
TRF-DB	2.07±1.64	2.38±1.61	0.49±0.87	0.61±0.78	<0.001	<0.001
TRF-EXT	20.23±13.71	21.96±13.38	2.95±5.01	3.11±4.11	<0.001	<0.001
mother*						
WURS	18.43±13.99	19.83±15.05	12.41±9.58	12.56±10.22	0.04	NS (0.09)
DSM	13.17±10.55	14.00±11.56	10.24±6.48	10.72±5.87	NS (0.16)	NS (0.28)
father*						
WURS	21.60±14.76	21.00±15.36	13.76±11.01	12.33±10.81	0.015	0.05
DSM	17.83±13.60	18.75±14.97	11.19±6.69	10.11±6.49	0.011	0.03

*=Numbers represent mean values±SD

toms of the child, multiple regression analysis using linear model/analysis of covariance (ANCOVA) was performed. All statistical tests were two-tailed and used the 0.05 level of statistical significance. All data were entered and analyzed by using SPSS 17.0.

RESULTS

Descriptive statistics

Descriptive statistics are shown in Table 1. Patient groups did not differ significantly in patient age. However, there was a significant predominance of boys in the clinical group, in keeping with results from epidemiological studies.

Clinical and control group differed significantly in all evaluated subscores from CBCL and TRF checklists. All of the subscore values were significantly higher in the clinical group.

Tab. 2. Correlation between child and parental ADHD symptom scores, clinical and control groups.

group		CBCL-AP/WURS		CBCL-AP/DSM	
		Spearman rho	Sig. (2-tailed)	Spearman rho	Sig. (2-tailed)
father	clinical	0.16	NS (0.38)	0.46	0.01
	control	0.19	NS (0.26)	0.23	NS (0.16)
mother	clinical	0.06	NS (0.76)	–0.09	NS (0.62)
	control	0.23	NS (0.18)	0.27	NS (0.09)

Tab. 3. Correlation between reported parental childhood and current ADHD symptoms, clinical and control groups.

group		WURS/DSM	
		Spearman rho	Sig. (2-tailed)
father	clinical	0.71	<0.001
	control	0.75	<0.001
mother	clinical	0.66	<0.001
	control	0.71	<0.001

Tab. 4. Results of analysis of covariance (ANCOVA) for relative effects of evaluated factors on ADHD symptoms of the child, clinical group.

		estimate (β)	Std. error	Test stats.	Sig.
age		–0.7349	0.41494	–1.771	0.089
gender		0.82133	1.43215	0.573	0.571
father	WURS	–0.0858	0.05343	–1.605	0.122
	DSM	0.21846	0.06127	3.566	0.001
mother	WURS	0.10079	0.05182	1.945	0.064
	DSM	–0.1305	0.07407	–1.762	0.091

Apart from a single evaluated item (maternal DSM score), parents from clinical group demonstrated significantly higher childhood and current levels of inattention and hyperactivity/impulsivity symptoms based on their self-reports (WURS and DSM checklists) than their counterparts in the control group.

Due to high gender ratio with male predominance in the clinical group, subgroup analysis for boys was performed in both clinical and control groups. Relations between symptom subscores remained essentially unchanged, apart from maternal WURS score.

Parental and proband ADHD symptoms

Potential association between parental and proband ADHD symptoms was assessed. Childhood and current ADHD symptoms (WURS and DSM checklist scores) from both parents were correlated to current ADHD symptoms of the child (CBCL-AP subscore). The only significant correlation (Spearman rho) was found between current parental and child's ADHD symptoms (parental DSM score and CBCL-AP subscore of the child; Table 2).

To determine the relative effect of evaluated factors on ADHD symptom expression in the child, ANCOVA was used, which allowed us to control for confounding factors. In addition to parental ADHD symptom scores, patient gender and age (confounding factors) were included in the analysis. Current paternal ADHD symptoms (DSM score) remained the only significant predictor of ADHD symptom expression in the child (CBCL-AP subscore, Table 4; 95% CI for paternal DSM was calculated at 0.092–0.345 with adjusted R^2 of 0.344, signifying a large parameter effect size).

Parental childhood and adulthood ADHD symptoms

In both patient groups a highly significant correlation was found between childhood and current parental ADHD symptoms (Table 3, Spearman rho consistently above 0.65, $p<0.001$).

Reported ADHD symptoms from parents and teachers

In the clinical patient group, ADHD symptoms reported by parents (CBLC-AP) correlated well with those reported by teachers (TRF-AP, Spearman rho of 0.417, $p=0.022$), whereas no such level of correlation could be found in the control group (Spearman rho of 0.183, $p=0.279$).

DISCUSSION

This study is an attempt to investigate the relationships on the dimensional spectrum of ADHD symptomatology between children and their parents. Its application could be of benefit in everyday psychiatric clinical practice.

The groups did not differ in mean age, but differed strongly in gender ratio with strong prevalence of boys. Similar relationship is also reported in other studies

from 3:1 in community samples to between 6:1–9:1 in clinic-referred samples (Gaub & Carlson 1997), confirming the similarity of our clinical sample to those from recent studies.

Higher values of externalizing problems were observed in the clinical group, suggesting that even after exclusion of children with overt comorbid difficulties, children that are clinically referred have other difficulties beside ADHD as well, especially behavior problems. The convergence of CBCL scales with specific youth diagnoses was previously found between AP subscale and the ADHD diagnosis, between the DB and AB subscales and the diagnosis of Conduct disorder (Biederman *et al.* 2005; Biederman *et al.* 1993; Steingard *et al.* 1992). We considered the possibility that our findings of higher values for all symptom subscores in the clinical group could be driven by male predominance. However, subsequent analysis of male clinical and control subgroups did not prove this assumption.

Both parents reported significantly higher values of their own childhood and current ADHD symptoms in the clinical group, confirming the familial aggregation among family members (Takeda *et al.*, 2010). It is known that many parents of children with ADHD suffer from the same disorder. The data on adult ADHD is fairly new, so it is not common for parents to have a history of any clinical assessment regarding their difficulties. Recently, Manor *et al.* (2010) reported that ADHD symptoms in adults are under-reported indicating that subjects tend to under-estimate their own ADHD-related impairments, while Faraone *et al.* (1997) showed that number of symptoms reported by adults with ADHD did not differ between those who did and did not have children with ADHD, so that ADHD adults with ADHD children are not biased to over-report ADHD symptoms.

We used TRF-AP as a means of control on parental reporting of ADHD symptoms in a child. A good correlation was found between ADHD symptoms of the child reported by parents and teachers.

The results of the study showed that paternal ADHD symptom severity was significantly associated with global ADHD symptom severity in proband, whereas we did not confirm a significant relationship with maternal symptom severity. Our findings were discordant with the results of the study by Goos *et al.* (2007) in which greater levels of impairment were found in children with maternal ADHD history versus paternal history.

In concordance with findings from different studies claiming that persistence of ADHD symptoms into adulthood renders the disorder highly heritable (Faraone 2004; Faraone *et al.* 2000), our results showed a significant correlation between adult current ADHD symptoms and ADHD symptoms of the child, whereas the correlation between parental childhood ADHD symptoms and ADHD symptoms of the child was not significant. Moreover, in multivariate regression/ANCOVA, the paternal DSM score was the only significant predictor of ADHD symptoms of the child.

A correlation between parental childhood and current ADHD symptomatology confirms association of childhood and adulthood ADHD symptoms and showed robustness and stability of reported symptoms and are in concordance with the findings that in 60% of children ADHD symptoms persist either as a residual type or as a full clinical disorder. Thus, ADHD displays all characteristics of chronic disease (Mannuzza *et al.* 1993; Barkley 2002).

Finally, it might be useful for the clinician to explore the severity of the parents' ADHD symptomatology. ADHD in adults is associated with impairment across a variety of domains with a higher than average rate of psychiatric conditions (Pliszka 1998). The symptoms of ADHD are likely to influence parenting style, which could be even more important in the interaction with ADHD children because they tend to be more sensitive to ineffective parenting. Importantly they require consistency, patience and monitoring even more, which is, for parents with high levels of ADHD symptoms, difficult to achieve. Parents could benefit from additional parent training, but on the other hand merit a modified approach taking into account their difficulties. The results of our study stress the importance of involving the father into the clinical assessment, suggesting children with ADHD could benefit if their father participated in the therapeutic process.

Limitation to the study

We included only children with both available biologic parents. Many children that fulfilled inclusion criteria couldn't be included because one of the parents was not available; many of them were divorced, not in contact with the child, or in conflict with partner. Some parents gave the consent, but dropped out because they failed, repeatedly forgot to return the questionnaires or did not want to include the teacher into the study. We could speculate that parents with most severe forms of ADHD were not included in the study. We found these criteria constricted the number of children included, on the other hand it confirmed higher levels of interparental discord observed in other studies (Wymbs *et al.* 2008) and added important insight into these fragile pediatric population.

Children in control group were chosen by the teacher. The bias is that the sample is not representative of the general school population, since probably the most cooperative parents were included to ensure high probability of returned questionnaires.

Many of the symptoms of ADHD as described in questionnaires are subjective. Parental data derived from self-reports, which could be influenced by parental self-awareness, feelings of fear or guilt, comorbid psychopathology, etc. Our findings should be conformed in the study using larger samples.

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