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PSYCHIATRY AND PRECLINICAL PSYCHIATRIC STUDIES - ORIGINAL ARTICLE

Hair cortisol concentration in mothers and their children: roles of maternal sensitivity and child symptoms of attention-deficit/hyperactivity disorder

Susan Schloß[1] · Viola Müller[1] · Katja Becker[1] · Nadine Skoluda[2,3] · Urs M. Nater[2,3] · Ursula Pauli-Pott[1]

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Abstract
Associations between mothers' and children's cortisol secretion parameters are well established. According to the bio-behavioral synchrony model, these associations reflect influences of the mother–child relationship, the child's social adjustment, and might also reflect shared genetic dispositions. From the bio-behavioral synchrony model, we predicted a stronger mother–child hair cortisol concentration (HCC) link in mothers showing highly adequate (compared to those showing less adequate) parenting behaviors and in children showing low (compared to those showing high) ADHD symptoms. From a genetic perspective, no such moderator effects, or a stronger mother–child HCC link in children with high ADHD symptoms, can be expected. The study sample consisted of 111 4–5-year-old children (64 of whom screened positive for increased ADHD symptoms) and their mothers. ADHD symptoms were assessed by a clinical interview and parent and teacher questionnaires. Maternal sensitive/responsive parenting behavior was assessed by an at-home behavior observation procedure. In mothers and children, HCC in the most proximal 3-cm scalp hair segment was analyzed using luminescence immunoassay. Overall HCCs of mothers and their children correlated significantly. Maternal sensitivity/responsiveness and child ADHD symptoms proved to be significant moderator variables of this association: High maternal sensitivity/responsiveness and low ADHD symptoms of the child were associated with a stronger mother–child link in HCC. The findings are in line with the bio-behavioral synchrony model in the mother–child relationship, and are less compatible with a genetic perspective. The results might hint at environmental events influencing the development of stress axis functioning in subgroups of preschoolers with high ADHD symptoms.

Keywords ADHD · Hair cortisol concentration · Mother–child relationship · Bio-behavioral synchrony · Maternal sensitivity

Introduction

In recent years, research on the caregiver–child relationship has revealed that caregivers and children continuously exert mutually regulating influences during their communication. Accordingly, their behaviors (regarding aspects such as gaze, affect expression, vocalization, or touch) are often coordinated and synchronized (Tronick and Beeghly 2011). The phases of such behavioral synchrony are typically accompanied by synchronized physiological signaling (e.g., in heart rate, or brain oscillations in alpha and gamma rhythms) and endocrine responses (e.g., in oxytocin or cortisol secretion) (Feldman 2017; Palumbo et al. 2017). A higher magnitude of such synchronized behaviors and bio-signals (i.e., indications of the caregiver–child "bio-behavioral synchrony") have been found to be associated with more adequate, sensitive/responsive caregiving and a secure caregiver–child attachment relationship, which in turn foster the child's self-regulation and social adjustment development (Feldman 2012).

✉ Ursula Pauli-Pott
Ursula.pauli-pott@med.uni-marburg.de

[1] Department of Child and Adolescent Psychiatry, Psychosomatics and Psychotherapy, Philipps-University of Marburg, Hans Sachs Str. 6, 35039 Marburg, Germany

[2] Clinical Biopsychology, Department of Psychology, Philipps-University of Marburg, Gutenbergstraße 18, 35032 Marburg, Germany

[3] Clinical Psychology, Department of Psychology, University of Vienna, Liebiggasse 5, 1010 Vienna, Austria

Regarding the secretion of cortisol (i.e., responses of the hypothalamic–pituitary–adrenal (HPA) axis), in particular, recent studies demonstrated significant correlations between mothers' and their children's salivary cortisol responses to stress and to their dyadic play interactions. Such correlations have been found in infancy (Bright et al. 2012; Middlemiss et al. 2012), in toddlerhood (Atkinson et al. 2013), in preschool age (Ruttle et al. 2011), and even in adolescence (Papp et al. 2009; Saxbe et al. 2014). Further studies, moreover, revealed that mothers' basal salivary cortisol concentration (e.g., Hibel et al. 2014; Middlemiss et al. 2012; Ruttle et al. 2011) and their accumulated, long-term cortisol secretion [as captured by the hair cortisol concentrations (HCC)] are associated with those of their children (Flom et al. 2017; Liu et al. 2017; Ouellette et al. 2015).

Several studies have analyzed the question of whether the association of mother–child cortisol parameters is modulated by more adequate caregiving. In a community-based, low-risk sample, Atkinson et al. (2013) found that the link between mothers' and children's cortisol response to a separation episode was stronger in mothers who showed highly sensitive, responsive parenting than in mothers who showed less sensitive, responsive parenting. Likewise, Sethre-Hofstad, Stansbury, and Rice (2002) demonstrated that mothers' sensitivity/responsiveness toward their preschool children modulated the association between the children's cortisol response to a challenging episode and the mothers' own response to watching this episode on a monitor. The higher the mothers' sensitivity, the stronger was this link. Ruttle et al. (2011) however, found that the link between preschoolers' salivary cortisol secretion and that of their mothers, as measured by repeated saliva samples collected during two home visits, was not affected by maternal sensitivity/responsiveness. Hence, although the correlation between mothers' and children's cortisol responses to several challenging events has been found to depend on maternal sensitivity/responsiveness, it remains unclear whether this moderator effect can be generalized to the long-term secretion of cortisol in mothers and their children.

High maternal sensitivity/responsiveness has been found to be associated with a higher compliance of the child with maternal rules (Kochanska 1997), moral internalization (Feldman 2012), and may also lead to an easier adoption of the mother's attitudes and communicated affective meaning of events, situations, and objects (see, e.g., Baldwin and Moses 1996; Tronick and Beeghly 2011). Given these findings, and the well-established dependence of the cortisol response on appraisal and attribution processes of an event (Denson et al. 2009; Dickerson and Kemeny 2004), it seems very probable that there is a particular correlation between sensitive/responsive mothers' integrated everyday cortisol secretion over a long-term period and that of their children. However, such moderator effects by maternal responsiveness/sensitivity have not yet been analyzed.

As mentioned above, continuously high "bio-behavioral synchrony" has been thought to predict self-regulation and social adjustment development in childhood. A high self-regulation capacity facilitates the development of social adjustment, thus protecting the child from developing externalizing behavior problems and symptoms of attention-deficit/hyperactivity disorder (ADHD) (Moffitt et al. 2011). Based on this perspective, one can infer a lower synchrony between the long-term cortisol secretion in children showing such behavior problems and that of their mothers. The less sensitive/responsive parenting of mothers whose children show externalizing/ADHD symptoms (Chronis et al. 2007; Denham et al. 2000; Olsen et al. 2005; Pauli-Pott and Beckmann 2007; Pauli-Pott et al. 2017a) might further contribute to a lower mother–child cortisol correlation.

However, the mother–child cortisol correlation (or adrenocortical synchrony) might also reflect influences of genetic factors (Federenko et al. 2004; Wust et al. 2004). In children with ADHD, a lower basal salivary cortisol secretion (small effect size) (Scassellati et al. 2012) has been found, which has sometimes been thought to reflect a genetically based disposition (Isaksson et al. 2012). From a genetic perspective, one might expect to find no modulating effect of ADHD symptoms or, if ADHD and cortisol secretion share genetic factors, to find a substantially positive correlation between the long-term cortisol secretion of children with high ADHD symptoms and that of their biological mothers.

To our knowledge, no previous study has analyzed the mother–child correlation in long-term cortisol secretion in children with externalizing behavior problems or symptoms of ADHD. The present study therefore aims to contribute to this issue by addressing the following questions: Do maternal sensitivity/responsiveness toward her child and ADHD symptoms of the child affect the mother–child correlation in long-term cortisol secretion? In children with high ADHD symptoms, is this correlation less pronounced (in comparison to children with low symptoms), as predicted by the bio-behavioral synchrony model, or more pronounced, as predicted from a genetic perspective? From the model of bio-behavioral synchrony, we infer the following hypotheses: (a) maternal sensitivity/responsiveness and (b) ADHD symptoms of the child modulate (i.e., show a moderator effect on) the association between the mother's long-term cortisol secretion and that of her child. We expect this association to be stronger in mothers who show highly sensitive/responsive parenting (compared to those who show less sensitive/responsive parenting) and in children who show low ADHD symptoms (compared to children who show high ADHD symptoms).

Methods

Participants

The sample consisted of $n = 198$ 4–5-year-old children (115 boys; 58%) and their primary caregivers. The study draws on data from the first assessment wave of an ongoing longitudinal study from preschool age onwards (for publications of other data from this study see e.g., Pauli-Pott et al. 2017a, b). Participants were recruited from childcare facilities in Marburg and the surrounding area (middle-west Germany). After expressing their willingness to participate, parents filled in a screening questionnaire (ADHD rating scale by Döpfner et al. 2008). To enrich the sample with children showing more severe ADHD symptoms, 113 preschoolers (57%) who scored above the lower bound of the 95% confidence interval of the clinical cut-off score of this questionnaire, and 85 children scoring below this point were included. Exclusion criteria were: IQ < 80, motor disabilities, sensory handicaps, chronic diseases involving brain functions or the HPA-axis (e.g., Cushing's disease), indication of a trauma (serious physical maltreatment, life-threatening injury), any continuous pharmacological treatment, and insufficient German language skills of parents or child.

Of the original sample, 65 children and 41 mothers had to be excluded from the present analyses because they refused to participate in the hair collection part of the study or did not fulfill the criterion of minimum hair length of 3 cm. During hair processing, a further 11 children and five mothers had to be excluded because the hair sample was insufficient in terms of length or amount. In 111 cases, complete HCC data of primary caregivers[1] and their children were available. To examine selection effects, we compared those participants with available HCC data ($n = 111$) with those with missing data ($n = 87$). No significant differences emerged regarding maternal [$\chi^2(3) = 1.77$] and paternal education level (Chi2 $(3) = 6.52$), ADHD symptom screening [$\chi^2(1) = 0.36$], an ADHD symptom composite score ($t = 0.15$), and oppositional symptoms ($t = 0.19$) or anxiety/depressive symptoms ($t = 0.21$). However, due to the shorter haircuts of the boys, significantly more boys than girls were excluded from the analysis ($t = 15.3$, $p < 0.001$).

Table 1 presents descriptive statistics of the 111 cases analyzed below as well as comparisons between the 64 children with and 47 children without elevated ADHD symptoms according to the study variables. Caregivers provided written informed consent for their own and their child's participation. They received an expense allowance of 50 Euros. The Ethics Committee of the Medical Faculty, University of Marburg, approved this study.

Procedure

Data were collected within the scope of a "playroom" session at the childcare facilities (data from which will be published elsewhere), a home visit, and a telephone interview with the mother. The initial telephone call (scheduling of appointments), the home visit, the playroom session, and the telephone interview were conducted in that order within a maximum time frame of six weeks. During the home visit, the observation of the mother–child interaction, the intelligence test with the child, and the collection of the hair samples (including a questionnaire assessing hair characteristics and treatment) were conducted. A clinical interview on the child's ADHD symptoms was carried out by telephone. Parents and kindergarten teachers completed questionnaires on ADHD symptoms as well as oppositional and anxiety/depressive symptoms of the child.

Variables

Hair cortisol concentration

Several thin hair strands were cut as close as possible to the scalp from the posterior vertex region of the head. To determine hair cortisol concentration, the first scalp-near 3 cm segment was used, which is thought to reflect the cumulative cortisol secretion of the past 3 months (Wenning 2000). Hair-washing and cortisol extraction procedures were based on laboratory protocol by Stalder et al. (2012), with minor modifications. In brief, hair samples were washed twice for 3 min using 3 mL isopropanol. For cortisol extraction, 10.0 ± 0.5 mg whole, finely cut hair samples were incubated in 1.8 mL methanol for 18 h at room temperature. After incubation, 1.6 mL of the extract was transferred to another glass vial. The methanol was evaporated at 50 °C under a constant stream of nitrogen until the samples were completely dried. Finally, 150 µl high performance liquid chromatography (HPLC) water was added and vials were vortexed for 15 s. For cortisol determination, 50 µL was used for analysis with commercially available cortisol luminescence immunoassay (LIA; IBL, Hamburg, Germany).

As the HCC values of mothers and children showed skewed distributions, the HCC scores were log-transformed after the exclusion of outliers (i.e., regarding maternal HCC two cases, regarding child HCC two cases, which exceeded the mean ± 3 SD) to reach a more normal distribution. Potential influences of several confounders were checked. In the children, we found no significant

[1] One of the 111 primary caregivers (defined as the parent who shared the most amount of time with the child) was a father. Because an exclusion of this caregiver–child pair did not alter the results of the presented analyses, the case was left in the sample. In the following, however, we refer to this father as a "mother" for the sake of simplicity of wording.

Table 1 Description of sample, comparisons between ADHD symptom groups

	Low ADHD symptoms	High ADHD symptoms	Comparison between ADHD symptom groups	Total sample
n	47	64		111
Age in months m (sd)	52.8 (6.0)	52.3 (5.8)	$t=0.48$, $p=0.634$	52.5 (5.8)
Gender *n* (%)				
Male	24 (51)	27 (42.2)	χ^2 (1) = 0.86, $p=0.354$	51 (45.9)
Female	23 (49)	37 (57.8)		60 (54.1%)
Education level of mother *n* (%)				
No completed education	0 (0)	1 (1.6)		1 (0.9)
Basic education	2 (4.3)	6 (9.4)		8 (7.2)
Vocational qualification	13 (27.7)	28 (43.8)	χ^2 (4) = 6.26, $p=0.181$	41 (36.9)
High school	11 (23.4)	9 (14.1)		20 (18.0)
College	21 (44.7)	20 (31.2)		41 (36.9)
Education level of father *n* (%)				
Basic education	6 (12.8)	14 (21.9)		20 (18.5)
Vocational qualification	6 (12.8)	15 (23.4)	χ^2 (3) = 6.80, $p=0.079$	21 (19.4)
High school	18 (38.3)	12 (18.8)		30 (27.8)
College	17 (36.2)	20 (31.2)		41 (34.3)
(No reply)	(2)	(1)		(3)
Employment of mother *n* (%)				
Full time	6 (12.8)	14 (22.2)	χ^2 (2) = 1.77, $p=0.412$	20 (18.0)
Part time	26 (40.6)	33 (52.4)		59 (53.2)
None	15 (31.9)	16 (25.4)		31 (27.9)
(No reply)	(–)	(1)		(1)
Employment of father *n* (%)				
Full time	41 (87.2)	53 (84.1)	χ^2 (2) = 0.24, $p=0.889$	94 (85.5)
Part time	1 (2.1)	2 (3.2)		3 (2.7)
None	5 (10.6)	8 (12.7)		13 (11.8)
(no reply)	(–)	(1)		(1)
Symptom scores of the child				
ADHD quest. parent *m* (sd)	10.45 (4.91)	26.27 (8.28)	$t=-12.56$, $p<0.001$	19.57 (10.54)
ADHD quest. teacher *m* (sd)	9.86 (10.41)	18.13 (12.48)	$t=-3.80$, $p<0.001$	14.62 (12.31)
ADHD clinical interview *m* (sd)	3.80 (3.55)	7.78 (5.11)	$t=-4.82$, $p<0.001$	6.08 (4.90)
ODD/CD quest. parent *m* (sd)	6.19 (4.76)	10.16 (7.24)	$t=-3.26$, $p=0.002$	8.45 (6.57)
Anx./dep. quest. parent *m* (sd)	4.90 (4.49)	7.49 (5.09)	$t=-2.76$, $p=0.007$	6.38 (4.99)
Maternal Sensitivity/responsiveness *m* (sd)	24.47 (3.94)	23.32 (3.77)	$t=-1.56$, $p=0.122$	23.80 (3.86)

ADHD Attention-deficit/hyperactivity disorder, *ODD/CD* oppositional defiant disorder/conduct disorder, *quest* questionnaire, *anx./dep.* anxiety/depressive symptoms

associations between HCC and body mass index (Spearman's $\rho=-0.03$), hair color, curling, hair-washing frequency, or use of hair products (gel, spray) (*t* scores: 1.83–1.06), and number of cigarettes smoked by the mother (Spearman's $\rho=-0.06$). In the mothers, there were also no significant associations between HCC and body mass index (Spearman's $\rho=0.01$), number of cigarettes smoked per day (Spearman's $\rho=0.09$), hair color, curling, hair-washing frequency, hair coloring, or use of hair products (spray, conditioner) (*t* scores: 0.19–1.78).

Maternal sensitivity/responsiveness

Maternal responsiveness was assessed during two standardized interaction tasks conducted at home (Pauli-Pott et al. 2017a). In the first task, mother and child were instructed to work together on a puzzle (60 pieces, picture of cats), and in the second task, they were asked to build a figure (according to a template) with a set of toy building blocks. Each episode lasted for 6 min. Responsiveness was assessed according to the 4-point rating

scale "Responsiveness/Sensitivity/Appropriate Scaffolding" adapted for home visits from the Mannheim Rating Scale for the Assessment of Mother–Child Interaction (Pauli-Pott and Beckmann 2007; Pauli-Pott et al. 2004; Polowczyk et al. 2000). High responsiveness/sensitivity (scored with 3 = "often" or 4 = "all the time") is defined as being attentive to the child's behavior, interpreting his/her behavior accurately, and reacting adequately while respecting the intentions and rhythm of the child, such that the mother often follows the child's lead in the play interactions. Poor responsiveness/sensitivity (scored with 2 = "often" or 1 = "most of the time") is characterized by intrusive/overinvolved and/or non-responsive behavior of the mother. Assessments were conducted using a 30-s time-sampling procedure, i.e., maternal responsiveness was scored every 30 s across the two episodes. Inter-rater reliability was checked in 10% ($n = 13$) of cases (home visits conducted by two observers) and proved to be adequate ($\kappa = 0.74$). For further analyses, the scores of the 30-s intervals were summed up and split at the median of the distribution to distinguish mothers showing high vs. relatively low responsiveness/sensitivity.

ADHD symptoms of the child

ADHD symptoms were assessed by a structured clinical parent interview conducted with the mother and by parent and teacher questionnaires. The Parental Account of Childhood Symptoms (PACS) interview (Taylor et al. 1986) in the modified preschool version by Daley (2010) was used. The ADHD scale of this interview shows good test–retest reliability (0.78, 15-week interval) and discriminates significantly between children with ADHD and healthy controls (Sonuga-Barke et al. 2003). In addition to the interview, mothers and teachers filled in the preschool version of the ADHD rating scale (FBB-ADHS-V teacher) by Döpfner et al. (2008). The scale captures ADHD symptoms according to the ICD-10 and DSM-5. The parent version shows high internal consistency (Cronbach's alpha: 0.94) and validity (differentiation between children with and without an ADHD diagnosis) (Breuer and Döpfner 2008). The teacher questionnaire also shows high homogeneity (Cronbach's alpha: 0.93) and validity (Breuer and Döpfner 2008). In the present sample, the three ADHD measures intercorrelated significantly (PACS interview with parent questionnaire: $r = 0.61$, $p < 0.001$; PACS interview with teacher questionnaire: $r = 0.25$, $p = 0.009$; parent questionnaire with teacher questionnaire: $r = 0.40$, $p < 0.001$). Moreover, children who screened positive for high ADHD symptoms at study enrolment differed significantly from children who did not regarding all measures of ADHD symptoms (see Table 1).

Symptoms of oppositional defiant disorder and anxiety/depressive symptoms

For descriptive and control purposes, we assessed symptoms of oppositional defiant disorder (ODD) or conduct disorder (CD) and anxiety/depressive symptoms of the child. Mothers filled in the German FBB-SSV questionnaire, which measures ODD/CD symptoms of the child (Döpfner et al. 2008) according to the ICD-10. The scale shows high internal consistency (Cronbach's alpha = 0.91) and validly discriminates between children with ODD/CD and controls (Görtz-Dorten et al. 2014).

The Anxious/Depressed scale of the German version of the Child Behavior Checklist (CBCL4-18) by Döpfner et al. (1994) was employed. The scale shows significant associations with anxiety and emotional disorders, indicating good validity (Döpfner et al. 1994).

Maternal depressive and ADHD symptoms

For control purposes we moreover assessed maternal depressive and ADHD symptoms.

Maternal depressive symptoms

The German version of the Center for Epidemiological Studies Depression Scale (CES-D; Hautzinger et al. 2012) was applied to assess maternal depressive symptoms. For this version, good internal consistency (Cronbach's $\alpha = 0.89$) and validity (correlations with other depression questionnaires) have been established (Hautzinger et al. 2012).

Maternal ADHD symptoms

Mothers filled in the German version of the Conners Adult ADHD Rating Scale (CAARS; Christiansen et al. 2011) and were interviewed using the Wender-Reimherr interview for adults (Rösler et al. 2008). Internal consistency (Cronbach's alpha) of the interview scale is 0.82. The method validly differentiates between adults with and without an ADHD diagnosis (Rösler et al. 2008). The scores were summed up after z-transformation.

Statistical analyses

To analyze whether the association between maternal and child HCC is modulated by maternal responsiveness/sensitivity, i.e., whether responsiveness/sensitivity is a moderator variable of this link (first hypothesis), we conducted a hierarchical multiple regression analysis, with child HCC as the criterion variable and maternal HCC as the predictor variable. Following Aiken and West (Aiken and West 1991), the moderator effect of maternal sensitivity, i.e., the interaction

Table 2 Results of the multiple hierarchical regression analyses

Criterion: child HCC					
A. Analysis of the hypothesized moderator effect by maternal sensitivity/responsiveness					
Step	Variable(s) added	R	R^2_{change}	$F_{change\ (df)}$	p_{change}
1	Gender of child, paternal education level	0.30	0.09	5.35 (2,108)	0.006
2	Maternal sensitivity/responsiveness	0.30	0.00	0.11 (1,107)	0.736
3	Maternal HCC	0.49	0.15	20.47 (1,106)	0.000
4	Maternal sensitivity/respon. X maternal HCC	0.52	0.03	4.02 (1,105)	0.048
B. Analysis of the hypothesized moderator effect by ADHD symptom groups					
1	Gender of child, paternal education level	0.30	0.09	5.35 (2,108)	0.006
2	ADHD symptom groups	0.30	0.00	0.00 (1,107)	0.978
3	Maternal HCC	0.49	0.15	20.59 (1,106)	0.000
4	ADHD symptom groups X maternal HCC	0.55	0.06	9.33 (1,105)	0.003

ADHD attention-deficit/hyperactivity disorder, *HCC* hair cortisol concentration

effect between maternal sensitivity and maternal HCC, was modeled by the product of these variables. We controlled for gender of child, parental education level, and daily hours the child spends outside the home in daycare. If significantly associated with HCCs of mothers or children, the respective variable was introduced into the regression equation in a first step. In consecutive steps, maternal sensitivity/responsiveness, maternal HCC, and the product of maternal sensitivity/responsiveness and HCC were introduced. The change statistics after each step reflect the amount of variance which was explained in the criterion variable by the (set of) variable(s) introduced into the regression equation. By the change statistic of the final step (introduction of the interaction term), the first hypothesis is tested.

To analyze the second hypothesis, i.e., whether ADHD symptom groups (children who screened positive for high ADHD symptoms vs. those who did not, see Table 1) act as a moderator variable, we used the corresponding procedure: In a hierarchical regression analysis with the child's HCC as the criterion variable, we introduced those control variables which significantly correlated with the mother's or the child's HCC (i.e., gender of child, parental education level, and daily hours the child spends outside the home in daycare). In subsequent steps, maternal HCC and ADHD symptom groups, and the product of ADHD symptom groups and maternal HCC were introduced. Calculations were carried out using SPSS 20, IBM.

Results

In preliminary analyses assessing the associations of the control variables with the HCC, we found no associations between maternal HCC and maternal depressive and ADHD symptoms (i.e., clinical interview and questionnaire scores), parental education levels, gender of child, or daily hours the child spends in daycare (r's between -0.043 and 0.144). We also found no interaction effects between maternal HCC and these control variables on child HCC (F_{change} scores between 0.22 and 2.44). HCC of the child was significantly associated with gender of child (boys showed lower HCC than girls, $r = -0.20$, $p = 0.037$) and paternal education level ($r = 0.24$, $p = 0.012$), while no such associations emerged with maternal education level and hours of daycare (r's = 0.055 and 0.064). In the following, we therefore adjusted for gender of child and paternal education level in all analyses. Maternal responsiveness/sensitivity and ADHD symptoms of the child correlated significantly ($r = -0.19$, $p = 0.048$).

Table 2 shows the results of the multiple hierarchical regression analyses. As can be taken from the change statistics of the third step, maternal HCC significantly predicted the HCC of the child, indicating an overall substantial association between the mother's and the child's HCC ($R^2_{change} = 0.15$, $p < 0.001$). While maternal responsiveness/sensitivity did not predict the child's HCC (step 2 of the regression analysis), the interaction effect between responsiveness/sensitivity and maternal HCC on the child's HCC proved to be statistically significant ($R^2_{change} = 0.03$, $p = 0.048$). The significant interaction effect indicates that maternal responsiveness/sensitivity is a significant moderator of the mother–child HCC association (Table 2A): the association was stronger in highly sensitive mothers than in the less sensitive mothers (see Fig. 1).

Table 2 also shows the results of the regression analysis on the hypothesized moderator effect by the ADHD symptom groups. While ADHD symptoms per se were not associated with the child's HCC, the interaction effect between maternal HCC and ADHD symptom groups on the child's HCC proved to be statistically significant ($R^2_{change} = 0.06$, $p = 0.003$): in children with high ADHD symptoms, the mother–child HCC association was smaller than in the comparison group of children with low symptoms (Table 2b, Fig. 2).

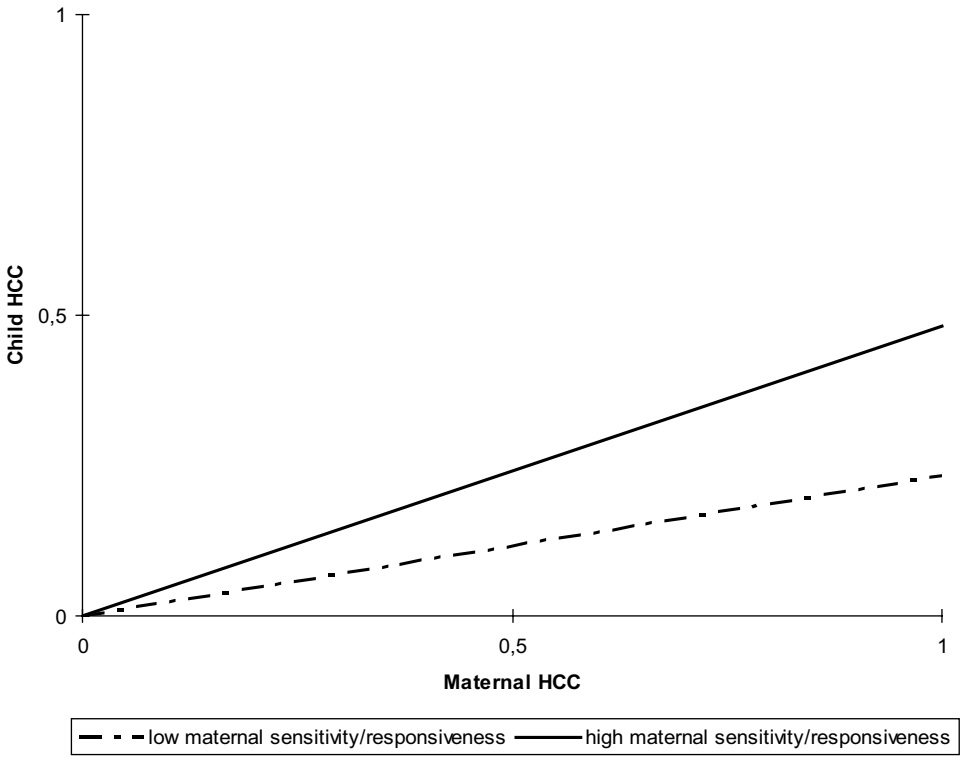

Fig. 1 Depiction of the significant moderator effect by maternal sensitivity/responsiveness. Regression lines of maternal HCC on child HCC in mothers showing low and high sensitivity/responsiveness after controlling for child gender and paternal education level, using standardized distributions. (Slopes of the regression lines were: β (CI95) = 0.20 (−0.11,.54); $p = 0.192$ for low maternal sensitivity/responsiveness; β (CI95) = 0.50 (0.34,.86); $p < 0.001$ for high maternal sensitivity/responsiveness). *HCC* hair cortisol concentration

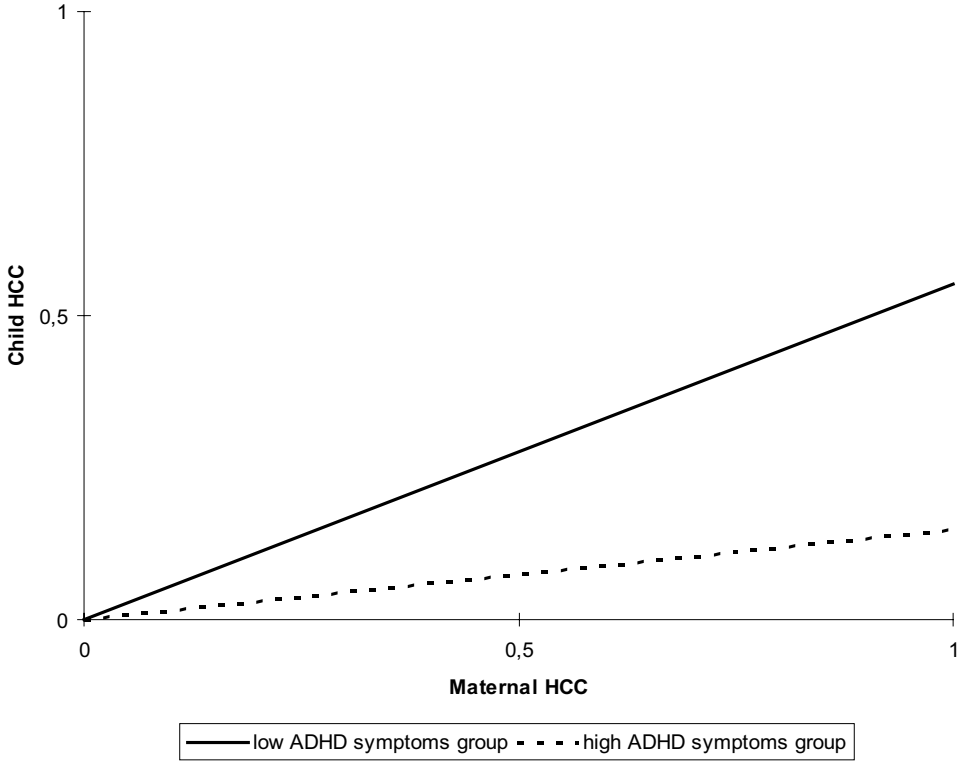

Fig. 2 Depiction of the significant moderator effect by ADHD symptom groups. Regression lines of maternal HCC on child HCC in children, showing low and high ADHD symptoms after controlling for child gender and paternal education level, using standardized distributions. (Slopes of the regression lines were: β (CI95) = 0.57 (0.42, 0.99); $p < 0.001$ for low ADHD symptoms group; β (CI95) = 0.15 (−0.10, 0.40); $p = 0.245$ for high ADHD symptoms group.) ADHD: attention-deficit/hyperactivity disorder; HCC: hair cortisol concentration

In a final step, we explored whether the two significant interaction effects explain common or independent variance in the child's HCC. The two interaction effects together explained 8% of the variance ($R^2_{\text{change}} = 0.08$, $F_{\text{change}} = 6.06$, $p = 0.003$). However, only the interaction between maternal HCC and ADHD symptom groups significantly contributed

to this prediction (maternal HCC-sensitivity: $\beta = 0.35$, $t = 1.65$, $p = 0.101$; maternal HCC-ADHD-symptom groups: $\beta = -0.55$, $t = -2.80$, $p = 0.006$). This indicates that the moderator effects by maternal sensitivity and ADHD symptoms of the child partially overlap, and that the moderator effect by ADHD symptoms explained significant independent variance over and above the effect of maternal sensitivity/responsiveness.

Discussion

Adrenocortical synchrony in caregiver–child pairs has been well established. Substantial caregiver–child correlations have been demonstrated in HPA axis reactivity and activity parameters such as cortisol secretion during dyadic play interactions, basal salivary cortisol concentration and long-term cortisol secretion captured via HCC. However, it is not known whether the strength of the mother–child HCC link depends on sensitive/responsive parenting, and it has not yet been analyzed whether and how ADHD symptoms of the child affect this link. Based on the bio-behavioral synchrony model by Feldman (Feldman 2012, 2017), we hypothesized a stronger association between mothers' and children's HCC in highly responsive/sensitive mothers (compared to less sensitive/responsive mothers) and a weaker association in children who show impaired social adjustment development, i.e., symptoms of ADHD (compared to children with low symptoms). Both hypotheses were confirmed: We found that high maternal sensitivity/responsiveness and low ADHD symptoms of the child were associated with a stronger mother–child link in HCC. The latter finding contradicts the expectation of strong genetic influences on HCC in children with high ADHD symptoms.

In line with the meta-analysis by Stalder et al. (2017), we found HCC to be significantly higher in boys than in girls. Further, we found HCC of the child to be positively associated with the father's education level. Gray et al. (2018) recently reviewed existing research on the association between parental education and the child's HCC, and found inconsistent results, i.e., sometimes positive, sometimes negative, and mostly no association. The link therefore seems to be rather weak in general. Future studies should analyze the conditions under which a positive or negative association emerges. In the present study, we controlled for these influences.

Adequately sensitive, responsive parenting has been found to lead to higher compliance with parental rules (Kochanska 1997) and probably an easier adoption of parental attitudes and internalization of communicated emotional meaning of situations and events (Feldman 2017). These shared attributions of meaning (Baldwin and Moses 1996; Tronick and Beeghly 2011) probably also affect cortisol responses to environmental conditions in everyday life. The stronger associations between the long-term cortisol secretion in the sensitive/responsive mothers and that of their children compared to less responsive mothers and their children may have resulted from these processes. We found that the moderator effect by maternal sensitivity/responsiveness partially overlapped with the moderator effect by ADHD symptoms. This overlap is likely due to the association between maternal responsiveness/sensitivity and ADHD symptoms ($r = -0.19$ in this sample, see, e.g., Pauli-Pott et al. 2017a for further details), which might reflect influences of parenting behavior on the ADHD symptoms and conversely influences of the ADHD symptoms on parenting. The moderator effect might therefore also be due to the processes discussed in the following.

In children with high ADHD symptoms, we found a significantly lower mother–child HCC association than in children with low symptoms. ADHD (see, e.g., Faraone et al. 2015) as well as HCC (Rietschel et al. 2017) show considerable heritability, and low HPA axis activity in ADHD has been attributed to genetic factors (e.g., Isaksson et al. 2012). From a genetic perspective, however, no moderator effect by ADHD symptoms (if HCC and ADHD share no genetic factors) or a stronger mother–child HCC link in children with high ADHD symptoms (if HCC and ADHD share genetic factors) had to be expected. The comparably weak mother–child HCC link that we found in children with high ADHD symptoms might reflect the less adequate mother–child relationship in these children. Additionally, the weak association might indicate non-genetic contributions to HCC in some of the children with high ADHD symptoms. For example, for some of these children, past environmental influences such as fetal programming effects associated with low birth weight or environmental adversity (including less adequate parenting) might have affected the HPA axis functioning of the children (Lupien et al. 2009) but not that of their mothers. Low birth weight and psychosocial family adversity are well-established risk factors of ADHD (e.g., Faraone et al. 2015). Our finding might therefore point to specific etiological subgroups. Some of the children with high ADHD symptoms may show dysregulated HPA axis functioning caused by these risk factors. Previous results from our study also pointed in this direction: We found that boys with increased ADHD symptoms who had been exposed to environmental adversity factors showed particularly low HCC (Pauli-Pott et al. 2017b). The present finding adds further support for the assumption that low HCC in some children with high ADHD symptoms are probably due to environmental effects. These effects might be pathogenically relevant. It would be worthwhile to analyze this hypothesis further in future research.

The current study has several strengths. We analyzed a comparably large, well-defined sample of caregiver–child

dyads using unbiased and validated methods such as HCC to capture the everyday mother–child cortisol coordination, an at-home behavioral observation of maternal sensitivity/responsiveness, and a multi-method approach to the assessment of ADHD symptoms. Such biological and behavioral data have rarely been combined in the given context. However, the study also has some limitations. First, we did not refer to ADHD diagnoses, but rather to high symptoms of ADHD. We used this procedure due to its higher sensitivity to the developing and often not yet full-blown symptoms in the preschool period (Sonuga-Barke et al. 2011) and its correspondence with the well-established dimensional distribution of ADHD symptoms in the general population (Coghill and Sonuga-Barke 2012). Second, based on current research and theorizing, we focused on the model of bio-behavioral synchrony and the mother–child link in HCC. This link might reflect diverse further influences, which were not analyzed in our study. We inferred specific modulating factors of this link and found significant effects, which add valuable insights into mechanisms underlying the link. Nevertheless, this does not rule out the possibility that other variables might also affect HCC in children and mothers and the mother–child link in HCC. In the present study, we analyzed moderator effects of sensitive/responsive parenting and ADHD symptoms on the mother–child long-term cortisol synchrony for the first time. The current results, as well as previous results regarding the link between HCC and ADHD symptoms (Pauli-Pott et al. 2017b), point to the relevance of HCC in the context of ADHD symptoms and associated environmental conditions.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

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