

Endocrinological and subjective stress responses in children with depressive, anxiety, or externalizing disorders

STEPHANIE STADELMANN, SONIA JAEGER, TINA MATUSCHEK, YOON JU BAE, KAI VON KLITZING, ANNETTE MARIA KLEIN, AND MIRKO DÖHNERT

University of Leipzig

Abstract

In this study, we used a stress test to investigate endocrinological and subjective stress responses of 8- to 14-year-old children with internalizing or externalizing disorders and healthy controls. The sample ($N = 170$) consisted of clinical and community children. Parents were given a diagnostic interview to diagnose their children's psychiatric condition. We measured saliva cortisol and subjectively experienced arousal in children before and after the Trier Social Stress Test for Children. Children also rated their performance immediately after the stress test, and 1 hr later they rated their positive and negative thoughts about this stressful event. Children with internalizing or externalizing disorders exhibited a blunted cortisol response compared to healthy controls. Depressed children rated their test performance lower and reported more negative thoughts after the test in comparison to healthy controls, anxious children reported more arousal before and after the task, and children with externalizing disorders reported more positive thoughts. In regression analyses, cortisol and subjective stress responses were both predictive of psychiatric disorders. The study extends previous work on the relation between psychiatric disorders and children's stress responses to an experimentally induced stress task by including a broad range of psychiatric disorders and by integrating endocrinological and subjective stress responses.

The ability to deal with stressful situations is an important developmental task for children (Compas, Connor-Smith, Saltzman, Harding Thomsen, & Wadsworth, 2001; Muris, Meesters, Merckelbach, Sermon, & Zwakhalen, 1998). In addition to normative (e.g., entering school) and negative life events (e.g., parental separation), dealing with daily hassles (e.g., interpersonal conflicts and taking an exam) is important and poses challenges to a child's development. An inadequate stress response can have an impact on the development and maintenance of psychiatric disorders (de Kloet, Joëls, & Holsboer, 2005; Susman, 2006). In this study, we analyzed the endocrinological and subjective stress responses of 8- to 14-year-old children with internalizing (i.e., depressive disorders and anxiety disorders) compared with externalizing disorders (i.e., oppositional defiant disorders and conduct disorders) and healthy control children after a social stress test.

Endocrinological Stress Response

One of the biological systems that has been in the focus of stress-related research is the hypothalamus–pituitary–adrenal

(HPA) axis, with cortisol as its primary hormonal product. The HPA axis is part of the body's interconnected set of physiological systems for managing physical, cognitive, and psychosocial stress (see Gunnar & Quevedo, 2007, for a detailed description of the neurobiological stress reaction). To cope effectively with these challenges, one must have the ability to flexibly adjust one's regulatory activity to the arousal set point that is most advantageous for a certain context (Hastings et al., 2011). Repeated stress over time, however, might lead to less flexible physiological systems with a consistently high or low arousal level. This so-called allostatic load leaves the individual vulnerable to pathology (Juster, McEwen, & Lupien, 2010; McEwen, 1998) and less able to adapt his or her physiological functions to new or different environmental challenges.

Endocrinological Stress Response in Individuals With Psychiatric Disorders

Individuals with internalizing or externalizing disorders often display dysregulated cortisol responses, such as cortisol levels that show a smaller increase in response to a stressor or that show a slower recovery after the removal of a stressor (Ayer et al., 2013; Burke, Davis, Otte, & Mohr, 2005; Chida & Hamer, 2008; Chrousos, 2009). In studies on the endocrinological stress processes of individuals with internalizing disorders, the hyperreactivity of cortisol (elevated and prolonged cortisol secretion) has received the vast majority of research attention. There is a great deal of evidence for the hyperreactivity of cortisol to acute psychological stress in

This publication was supported by the Leipzig Research Center for Civilization Diseases, University of Leipzig, which is funded by the European Union, the European Regional Development Fund, and the Free State of Saxony within the framework of the Excellence Initiative. The last two authors have a combined last authorship.

Address correspondence and reprint requests to: Stephanie Stadelmann, Department of Child and Adolescent Psychiatry, Psychotherapy, and Psychosomatics, University of Leipzig, Liebigstraße 20a, D-04103 Leipzig, Germany; E-mail: Stephanie.stadelmann@medizin.uni-leipzig.de.

depressed individuals (see meta-analyses by Lopez-Duran, Kovacs, & George, 2009; Stetler & Miller, 2011). In a study by Rao, Hammen, Ortiz, Chen, and Poland (2008), for example, depressed adolescents showed more elevated and prolonged cortisol secretion in response to the Trier Social Stress Test for Children (TSST-C; Buske-Kirschbaum et al., 1997) compared with healthy control subjects. Furthermore, similar findings of an elevated cortisol response to a psychosocial stressor were shown in a study on prepubertal children with social phobia as compared with healthy controls (van West, Claes, Sulon, & Deboutte, 2008). Overall, these findings indicate that this form of dysregulation of the HPA axis may be a phenomenon that is present in different internalizing disorders. However, the findings have been inconsistent. More recent studies with adults as well as with adolescents have also found that a reduced cortisol response (hyporeactivity) following a psychosocial stressor was associated with depressive and anxiety symptoms (Booij, Bouma, de Jonge, Ormel, & Oldehinkel, 2013; de Rooij, 2013; Keenan et al., 2013).

For children with externalizing disorders, most studies have found a blunted cortisol response following stress compared with healthy children (e.g., Fairchild et al., 2008; Hartman, Hermanns, de Jong, & Ormel, 2013; Maldonado, Trianes, Cortés, Moreno, & Escobar, 2009; Randazzo, Dockray, & Susman, 2008; van Goozen, Matthys, Cohen-Kettenis, Buitelaar, & van Engeland, 2000; van Goozen et al., 1998). However, in a meta-analysis on the stress-related cortisol responses of children with externalizing disorders, no robust effect was found (Alink et al., 2008). It is interesting to note that in this meta-analysis, which used a strong stressor that included outcome uncontrollability and a social-evaluative threat, the relation between cortisol reactivity and externalizing behavior approached the level of significance. This indicates that a relation between externalizing behavior and cortisol reactivity may be found when the stressor is strong enough to elicit a stress response in the total group. In the underarousal theory, Raine (2002) suggested a certain mechanism that underlies the inverse relation between cortisol levels and externalizing disorders. According to this theory, children with externalizing disorders are less sensitive to stress and are less easily physiologically aroused than other children. As a result, they have low levels of anxiety and more often engage in externalizing behavior.

Developmental Influences on the Endocrinological Stress Response and Its Relationship to Psychiatric Disorders

The reactivity of the HPA axis is influenced by developmental changes during the transition from childhood to adolescence. Studies provide evidence that basal cortisol levels increase with age and sexual maturation during the adolescent years (Gunnar, Wewerka, Frenn, Long, & Griggs, 2009). Developmental effects were also found in stress responses of the HPA axis (Gunnar, Wewerka, et al., 2009; Kudielka, Buske-Kirschbaum, Hellhammer, & Kirschbaum, 2004; Stroud

et al., 2009), although results are inconsistent. In their study of normative developmental changes in the HPA axis in 9- to 15-year-olds, Gunnar, Wewerka, et al. (2009) found that cortisol stress reactivity showed only a slight positive correlation with sexual maturation. Besides, the authors found gender-specific effects around age 13, with a more pronounced increase in cortisol stress response in girls than in boys.

Heightened physiological stress responses in typically developing adolescents may facilitate their adaptation to the new challenges of adolescence and adulthood (Gunnar, Wewerka, et al., 2009). However, puberty is also a key period for the onset of mood and anxiety psychopathology, and in vulnerable adolescents, pubertal changes may partially account for enhanced susceptibility to psychopathology during this phase (Angold, Costello, & Worthman, 1998; Costello, Copeland, & Angold, 2016; Reardon, Leen-Feldner, & Hayward 2009). Among individuals with depression, cortisol levels seem to increase with age, with the largest rate of increase between childhood/adolescence (mean age in studies = 12.7 years, range = 8.9–17.9 years) and adulthood (see meta-analysis by Stetler & Miller, 2011). As a consequence, differences in cortisol release between depressed and healthy subjects were found to be significantly smaller during childhood/adolescence compared with differences in cortisol release during middle or older adulthood (Stetler & Miller, 2011). An association between depression and elevated cortisol levels has been found in both (adult) men and women. Moreover, blunted cortisol reactivity to a stressor was reported for prepubertal dysphoric youth, contrary to exaggerated cortisol reactivity to a stressor in postpubertal dysphoric adolescents (Hankin, Badanes, Abela, & Watamura, 2010). Colich, Kircanski, Foland-Ross, and Gotlib (2015) also found that pubertal stage moderated the effects of cortisol stress reactivity on the development of major depressive disorder (MDD) in girls. Specifically, the onset of MDD was predicted by cortisol hyporeactivity in girls who were in an earlier pubertal stage (Tanner stage ≤ 2), but by cortisol hyperreactivity in girls who were in a later pubertal stage (Tanner stage ≥ 3.5). Among individuals with externalizing problems, Alink et al. (2008) found that the association between stress-related cortisol response and externalizing disorders did not differ according to age.

It is also known that the stress response of the HPA axis is influenced by some other factors, including the severity and persistence of psychopathology (Burke et al., 2005; Chida & Hamer, 2008; Kudielka, Hellhammer, & Wüst, 2009). For example, Booij et al. (2013) reported that the stress response changed from hyper- to hyporeactivity of cortisol when the depressive problems lasted for a longer period of time. This finding is also supported by Ayer et al. (2013), who found a relation between blunted cortisol responses to stress and a persistent dysregulation profile in youth. This profile consists of clinically elevated scores on the anxious-depressed and aggressive behavior, as well as attention problems scales of the Child Behavior Checklist (Achenbach, 1991). Blunted stress reactivity has also been proposed to

be a result of exposure to chronic stress (Fries, Hesse, Hellhammer, & Hellhammer, 2005).

Subjective Stress Response

Thus far, studies on experimentally induced stress have focused primarily on indicators of the biological stress reaction (i.e., measured by heart rate, blood pressure, and cortisol levels) and have largely neglected stress-related cognitive–emotional responses (Brosschot, Gerin, & Thayer, 2006). Two of the rare studies that included multiple stress response levels found a heightened self-reported anxiety in children and adults with social phobia compared with healthy controls before, during, and after the TSST-C, but no significant difference in their cortisol response (adults: Klumbies, Braeuer, Hoyer, & Kirschbaum, 2014; children: Krämer et al., 2012). In addition, another study on a sample of 8- to 12-year-old children from the general population found that the relation between perceived arousal and anxiety symptoms was stronger than the relation between perceived arousal and depressive problems. By contrast, only depressive problems but not anxiety problems were significantly related to the cortisol measures. Thereby, children with higher rates of depressive problems showed a flattened cortisol response to the stress task (Dieleman, van der Ende, Verhulst, & Huizink, 2010). In male adolescents with early-onset conduct disorders, Fairchild et al. (2008) found lower levels of reported fear and worry under psychosocial stress compared to healthy controls. Perceived arousal during TSST-C does not seem to be age dependent (Stroud et al., 2009; Gunnar, Wewerka, et al., 2009).

There is controversy in the literature about the association between subjective and physiological stress response measures (Quas, Hong, Alkon, & Boyce, 2000). Gunnar, Wewerka, et al. (2009) found a concordance between perceived arousal and cortisol reactivity in response to the TSST, whereas others have documented a discrepancy between the two (Fairchild et al., 2008; van Goozen et al., 2000) or even no association (Dielemann et al., 2010). Fairchild et al. (2008), as well as researchers from other fields (Gunnar, Brodersen, Nachmias, Buss, & Rigatuso, 1996; Kircanski, Waugh, Camacho, & Gotlib, 2016), suggested a weaker coordination between subjectively experienced and physiological arousal in individuals with psychiatric disorders.

A further important aspect of the stress-related cognitive–emotional response involves perseverative cognitions, which have been defined as “the repeated or chronic activation of the cognitive representation of one or more psychological stressors” (Brosschot et al., 2006, p. 114; see also the review by Watkins, 2008). Perseverative cognitions are hypothesized to prolong the immediate psychological and biological responses to life events and daily stressors. Thus, the body’s systems that are associated with stress (e.g., the cardiovascular, HPA, and immune systems) become chronically activated and more susceptible to the development of disease (the perseverative cognition hypothesis; Brosschot et al., 2006;

Schwartz et al., 2003). Perseverative cognitions seem to increase with higher age and pubertal status (Jose & Brown, 2008; Rood, Roelofs, Bögels, & Meesters, 2010) and are primarily implicated in a vulnerability to depression and anxiety (Watkins, 2008). Schmitz, Krämer, Blechert, and Tuschen-Caffier (2010) reported that children with social phobia reported more negative thoughts than healthy controls 2.5 hr after the TSST-C. A strong positive relation between perseverative cognitions (stress-reactive rumination) and depressive symptoms was found by Rood, Roelofs, Bögels, and Meesters (2012) in a large nonclinical sample of children and adolescents. Studies in adults using social-evaluative stressor tasks showed that stress-related state rumination predicted greater cortisol reactivity or delayed recovery (see review of Zoccola & Dickerson, 2012). In a study investigating the association between rumination and cortisol levels after a laboratory stressor in a clinical adolescent sample, Stewart, Mazurka, Bond, Wynne-Edwards, and Harkness (2013) reported that trait rumination in response to depressed mood was associated with prolonged cortisol secretion only in depressed adolescents. No such relation was found in the healthy control group. In contrast, Rudolph, Troop-Gordon, and Granger (2011) found no association between stress-related state rumination in 9-year-old children and their cortisol reactivity in response to a laboratory-based social challenge task. In sum, existing studies have rarely addressed both endocrinological and cognitive–emotional stress responses in the context of a stress task, and therefore they present an inconclusive picture of children with depressive and anxiety disorders.

The Current Study

In the present study, we investigated stress-induced cortisol and subjective responses (subjectively experienced arousal, and immediate and delayed thoughts) in preadolescents and young adolescents (8 to 14 years old) with internalizing disorders (without externalizing comorbidity) in comparison to those with externalizing disorders (without internalizing comorbidity) and healthy controls.

Because our main study interest was in internalizing disorders, and since data about endocrinological and cognitive–emotional stress responses in children with internalizing disorders are sparse and inconclusive, we focused on this spectrum of disorders and analyzed children with a depressive disorder separately from children with pure anxiety disorders (i.e., pure anxiety disorders without depression) in order to better understand the impact on the stress response of each type of disorder. Children with depressive disorders often suffer from high levels of anxiety symptoms or disorders (Kessler, Chiu, Demler, Merikangas, & Walters, 2005). Empirical findings and theory that discuss anxiety disorders as precursors of depressive disorders suggest that anxiety symptoms could be part of a depressive syndrome in children (Crick & Zahn-Waxler, 2003). A study by von Klitzing et al. (2014) also showed that comorbid depression and anxiety

symptom clusters lead to far higher levels of impairment compared to pure anxiety disorders. In composing our group of children with a depressive disorder, we decided to group together children with a pure depressive disorder and those with co-occurring depressive and anxiety disorders.

We expected a dysregulation of the HPA axis in response to the TSST-C in children with depressive and pure anxiety disorders compared with healthy controls. Due to the inconsistent cortisol-related results on internalizing disorders, we did not formulate specific hypotheses. In line with Beck's (1967) depression model and the empirical findings of Rood et al. (2012), we hypothesized that depressed children would rate their own performance more poorly immediately after the TSST-C and report more stressor-related negative thoughts 1 hr after the stress test than would healthy controls. Subjectively experienced arousal might also be higher in these children than in healthy controls; however, the literature is inconsistent here. In line with findings by Dieleman et al. (2010) and Krämer et al. (2012), we hypothesized that subjectively experienced arousal would be higher in children with pure anxiety disorders than in healthy controls. In accordance with Schmitz et al. (2010) we also expected more stress-related negative thoughts in the pure anxiety group than in the healthy control group, but we think that this is a more prominent feature in children with depressive disorders.

In children with an externalizing disorder, we expected a blunted cortisol reaction following the TSST-C. According to the theoretical implications of Raine (2002) and the work of Fairchild et al. (2008), we expected a lower level of subjectively experienced arousal in children with externalizing disorders than in healthy controls. As there is no literature on TSST-C-related cognitive responses in externalizing children, we did not formulate specific hypotheses on these aspects for this group of children.

In line with Fairchild et al. (2008) and Kircanski, Waugh, et al. (2016), we expected to see a discrepancy between subjectively experienced arousal and cortisol reactivity in children with psychiatric disorders, and a concordance between the two in the healthy control group. Based on the perseverative cognition hypothesis (Brosschot et al., 2006; Zoccola &

Dickerson, 2012), we expected to observe a general association between negative thoughts and cortisol.

Finally, we explored whether cortisol reactions or subjective stress responses would specifically predict the assignment to each of the diagnostic groups and whether interactions between cortisol and subjective stress responses would serve as predictors of psychiatric disorders. Due to our cross-sectional design, the statistical predictions do not imply any direction of effect.

We tested associations of study variables with age, pubertal status, and gender, and controlled for these variables in case of significant associations.

Method

Participants

Our sample consisted of 170 children (44.7% female; age: $M = 11.18$ years; $SD = 1.94$) and their parents, who participated in a longitudinal cohort study on depressive disorders from childhood to adulthood (Quante et al., 2012). In the majority of cases, mothers were the informants on symptoms and diagnoses ($n = 146$, 86%). Other informants were the biological or social father ($n = 14$, 8%), both parents ($n = 8$, 5%) and the grandparents ($n = 2$, 1%). The selection criteria for this study were the presence of a depressive disorder (DEPR group: pure depressive disorder [$n = 16$] or depressive disorder and anxiety disorder [$n = 10$], but without a comorbid externalizing disorder; $n_{\text{DEPR}} = 26$), at least one anxiety disorder (ANX group: anxiety disorders without a comorbid externalizing disorder, $n_{\text{ANX}} = 32$), or an externalizing disorder (EXT group: conduct disorder [$n = 7$] or oppositional defiant disorder [$n = 29$] without a comorbid internalizing disorder, $n_{\text{EXT}} = 36$). With respect to the externalizing spectrum, we focused on children with an oppositional defiant disorder or conduct disorder. We grouped both types of disorders together, because of the very small sample size of children with conduct disorder (see Table 1 for frequencies of comorbid psychiatric disorders within each group). A healthy control group ($n = 76$) was also recruited. Children in the healthy control group showed

Table 1. Frequencies of comorbid psychiatric disorders with at least one present diagnosis in the relevant category (% within subsamples)

Diagnosis	DEPR ($n = 26$)	ANX ($n = 32$)	EXT ($n = 36$)
At least one comorbid Axis I disorder	17 (65.4)	11 (34.4)	22 (61.1)
Anxiety disorder	10 (38.5)	0 (0.0)	0 (0.0)
ADHD	3 (11.5)	1 (3.1)	19 (52.8)
Adjustment disorder	3 (11.5)	2 (6.2)	0 (0.0)
Eating disorder	1 (3.8)	0 (0.0)	0 (0.0)
Enuresis/encopresis	1 (3.8)	5 (15.6)	5 (13.9)
Tic disorder	0 (0.0)	4 (12.5)	2 (5.6)

Note: DEPR, depression group; ANX, anxiety group; EXT, externalizing disorder group; ADHD, attention-deficit/hyperactivity disorder.

no psychiatric disorders and had a total difficulties score (Strengths and Difficulties Questionnaire; Goodman, 1997) that was within the normal range according to parent and child ratings. Within our control group, we matched boys and girls by age and parental socioeconomic status (SES). Further inclusion criteria for all participants were fluency in German, an IQ higher than 80 confirmed by the Culture Fair Intelligence Test—Revised Version (Weiss, 2006), and no concurrent endocrine diseases or concomitant administration of glucocorticoid medications. The study protocol was reviewed and approved by the Ethics Committee of the University Hospital of Leipzig. Informed consent was obtained from the parents, and assent was obtained from the children after the procedure had been explained. The investigation was carried out in accordance with the latest version of the Declaration of Helsinki. Sample characteristics and group differences in age, gender, pubertal status, and parental SES are shown in Table 2. To measure parental SES, we used a multidimensional index score (Lange et al., 2007). The score is the sum of three components: education and occupational qualification, occupational status, and net income. According to cut-offs presented by the KiGGS study group (Lange et al., 2007), SES was divided into three categories: low, intermediate, and high. The highest score of the two parents was used as the index for the SES of the family.

Procedure

We invited the parents and children to attend two sessions at our research center. During their first testing session, the children and parents completed different questionnaires, for ex-

ample, on anamnestic data (only parents) or children's symptoms and pubertal status (only children). Children were also tested for their IQ. Trained undergraduate and graduate psychologists, residents in training for child and adolescent psychiatry, and child psychiatrists conducted a diagnostic interview with at least one parent (or legal guardian) to obtain a child's categorical diagnosis. Children meeting the above-mentioned inclusion criteria were invited to take part in a second testing session. We administered the TSST-C embedded in a 3.5-hr afternoon session, which lasted from 2:00 p.m. to 5:30 p.m. for all participants. The session consisted of different questionnaires, calm play, and resting time before and after the TSST-C (which took place from 3:35 p.m. to approximately 3:55 p.m.) as well as a debriefing and positive feedback at the end of the session. Figure 1 provides an overview of the testing procedure.

Measures

Categorical assessment of psychiatric disorders. The German Version of the Kiddie Schedule for Affective Disorders and Schizophrenia for School Aged Children—Present and Lifetime Version (K-SADS-PL; Delmo, Weiffenbich, Gabriel, Stadler, & Poustka, 2001; based on the K-SADS-PL by Kaufman, Birmaher, Brent, Rao, & Ryan; 1996; and revised according to the K-SADS-PL 2009 Working Draft, Axelson et al., 2009) was administered to at least one parent. The K-SADS-PL is an internationally well-known and widely used semistructured diagnostic interview to assess current and lifetime psychopathology in children and adolescents ac-

Table 2. Demographic and psychometric measures for the diagnostic groups

Measure	DEPR (<i>n</i> = 26)	ANX (<i>n</i> = 32)	EXT (<i>n</i> = 36)	HC (<i>n</i> = 76)	Test Statistic
	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	<i>M</i> (<i>SD</i>)	
Age (years)	12.80 _a (1.35)	10.79 _b (2.00)	11.22 _b (1.79)	10.80 _b (1.90)	$F(3, 166) = 8.08$, $p < .001$; $\eta^2 = 0.13$
	<i>n</i> (%)	<i>n</i> (%)	<i>n</i> (%)	<i>n</i> (%)	
Gender (female)	14 (53.8)	13 (40.6)	11 (30.6)	38 (50.0)	$\chi^2(3, 170) = 4.87$, $p = .181$; Cramer $V = 0.17$
Pubertal status					
Pre/early	4 (15.4)	19 (63.3)	18 (52.9)	46 (63.9)	$\chi^2(3, 162) = 19.49$, $p < .001$; Cramer $V = 0.35$
Middle/late ^{a,b}	22 (84.6) _a	11 (36.7) _b	16 (47.1) _b	26 (36.1) _b	
SES parents					
Low	10 (41.7)	16 (51.6) _a	18 (52.9) _a	4 (5.6) _b	$\chi^2(6, 161) = 44.81$, $p < .001$; Cramer $V = 0.37$
Middle	11 (45.8)	9 (29.0)	12 (35.3)	32 (44.4)	
High ^c	3 (12.5)	6 (19.4)	4 (11.8) _a	36 (50.0) _b	

Note: DEPR, depression group; ANX, anxiety group; EXT, externalizing disorder group; HC, healthy control group; *n*, frequency; SES, socioeconomic status. Different subscript letters indicate number/frequency of significant group differences found in post hoc analyses (Games-Howell).

^aPre/early = Tanner \leq II, middle/late = Tanner \geq III.

^bMissing data: *n* = 8.

^cMissing data: *n* = 9.

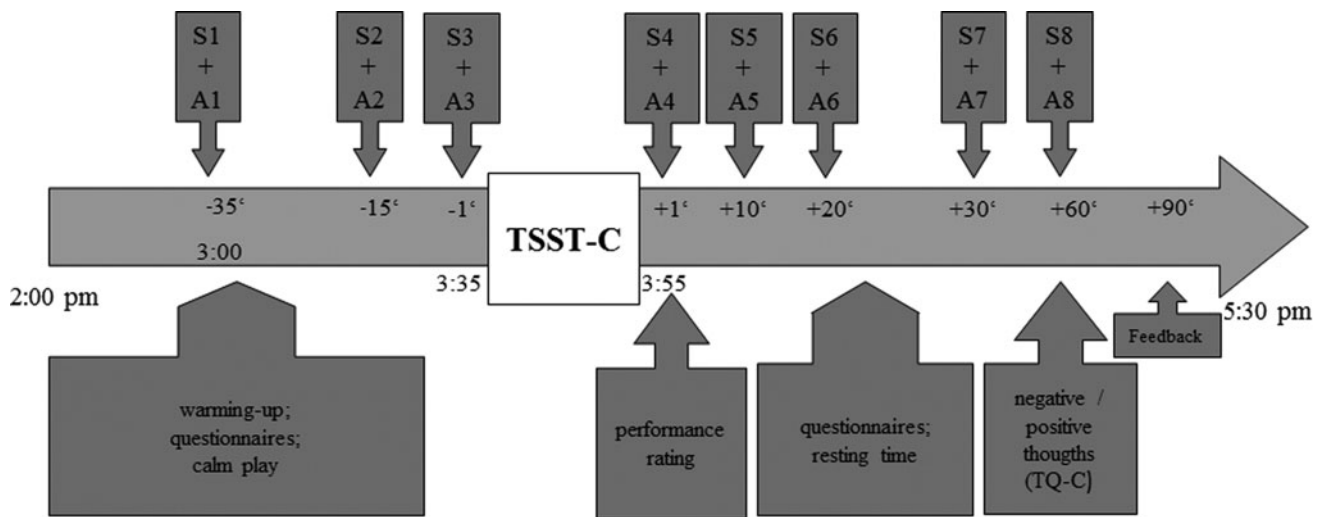


Figure 1. Testing procedure including the Trier Social Stress Test for Children (TSST-C) during the afternoon session. S1–8, saliva samples 1–8; A1–8, subjective arousal ratings 1–8; TQC, Thoughts Questionnaire for Children.

cording to the DSM-IV-TR (American Psychiatric Association, 2000; Sass, Wittchen, Zaudig, & Houben, 2003). The interrater reliability (10% double-rated interview audiotapes) was adequate with $\kappa = 0.76$.

Psychosocial stress test. TSST-C (Buske-Kirschbaum et al., 1997) is a well-established standardized laboratory stressor (Gunnar, Talge, & Herrera, 2009). The investigator accompanies the child to the TSST-C room where the “scientific committee” is already waiting (consisting of two unknown persons wearing lab coats). The investigator explains the testing procedure and reads the beginning of the story that the child should continue to tell during the testing phase. After this introduction, the investigator leaves the room, and the child has 5 min to prepare his/her story. After that, the “scientific committee” asks the child to step in front of the microphone and tell his/her story facing the camera. The speech task lasts 5 min, followed by a 5-min arithmetic task (e.g., counting backward from 758 in increments of 7). At the end of the TSST-C, the scientific committee explains that they will review the tape and provide feedback later. The child leaves the room and is escorted back to the main testing room by the investigator waiting outside of the TSST-C room.

This TSST-C protocol is aimed at inducing stress through the challenging tasks, the lack of nonverbal or positive verbal feedback, the videotaping of the session, and the standardized procedure in a sterile environment. Furthermore, participants receive negative feedback if their story ends before the 5-min session is over (after a 15-s waiting period: “You have more time, please continue your story”). The arithmetic task is chosen in accordance with the age of the participants and changed to a more challenging one if the participant does not make any mistakes. Every mistake is followed by the same standardized negative feedback (“That was a mistake, please start over from the beginning”; for a detailed description, see Buske-Kirschbaum et al., 1997). The TSST-C is well suited

to elicit stress as it meets the criteria of social evaluation, lack of controllability, and unpredictability described by Dickerson and Kemeny (2004) in their review. Furthermore, Zoccola and Dickerson (2012) showed that a stressor characterized by this kind of social-evaluative threat elicits more rumination than a stressor without this social-evaluative component. This result persisted from 3 to 5 days after the stressor in their study.

Salivary biomarkers. We collected saliva eight times (T_1 – T_8) at -35 (T_1), -15 (T_2), and -1 (T_3) min before the TSST-C and $+1$ (T_4), $+10$ (T_5), $+20$ (T_6), $+30$ (T_7), and $+60$ (T_8) min after the TSST-C. We collected the saliva samples with the Salivette[®] for Cortisol (Sarstedt, Nümbrecht, Germany). Collected saliva was centrifuged and aliquoted for the measurement of cortisol activity. Eating was prohibited for 30 min prior to sampling. Samples were stored at -80 °C until the measurement of the analytes. In contrast to earlier studies, which mostly used immunoassay as the analytic method for measuring salivary cortisol, we measured cortisol with liquid chromatography tandem mass spectrometry. Even though immunoassay and liquid chromatography tandem mass spectrometry are largely comparable in the interpretation of salivary cortisol dynamics in stress research, the immunoassay method revealed restricted accuracy in the measurement range below 5 nmol/l (Bae et al., 2015). For a detailed description of the analytic method, see Bae et al. (2015). We log transformed all cortisol values. As suggested by Pruessner, Kirschbaum, Meinlschmid, and Hellhammer (2003), we calculated two reactivity indices: the area under the curve with respect to ground (AUC_g) as an index of the total reactivity of the biomarker (sum of trapezoidal areas from time points T_3 to T_8) and the area under the curve with respect to increase (AUC_i) as an index of the magnitude of change in the total reactivity of the biomarker caused by the stressor ($AUC_g - [\text{value at time point } T_3 \times 80]$).¹ We included the cortisol values

only from time points T_3 to T_8 because of their relation to the stressor. At time points T_1 and T_2 , children did not yet know about the upcoming stress task in detail.

Subjectively experienced arousal. Children rated their arousal eight times (T_1 – T_8) at -35 (T_1), -15 (T_2), and -1 (T_3) min before the TSST-C and $+1$ (T_4), $+10$ (T_5), $+20$ (T_6), $+30$ (T_7), and $+60$ (T_8) min after the TSST-C on a 10-point Likert scale ranging from 1 (*not at all aroused*) to 10 (*very aroused*). The rating that occurred immediately after the TSST-C (T_4) consisted of two ratings instead of one, separately focusing on the speech task ($AROUS_{story}$) and the arithmetic task ($AROUS_{calc}$). Parallel to the cortisol measures, we calculated the mean of the arousal ratings from T_3 to T_8 (i.e., T_3 , T_4 $AROUS_{story}$, T_4 $AROUS_{calc}$, T_5 , T_6 , T_7 , and T_8 were averaged to create $AROUS_{mean}$).

Perceived performance. One minute after the TSST-C (T_4), we asked the children to rate their performance in the speech task ($PERF_{speech}$: “How well did you tell the story?”), as well as in the arithmetic task ($PERF_{calc}$: “How well did you calculate?”) using German school grades on a scale ranging from 1 (*best*) to 6 (*worst*). To ease the interpretation of our results, we recoded the scale so that higher values indicated a better performance (i.e., 1 = *worst* to 6 = *best*). $PERF_{speech}$ was significantly negatively associated with negative feedback from the scientific committee during the speech task ($r = -.22$, $p = .005$). $PERF_{calc}$ was not significantly associated with the negative feedback during the arithmetic task ($r = .14$, $p = .086$). We averaged the performance ratings across the two tasks ($PERF_{mean}$: mean of $PERF_{speech}$ and $PERF_{calc}$). Negative feedback during the tasks was not significantly associated with $PERF_{mean}$ ($r = .12$ for negative feedback $_{calc}$; $r = .13$ for negative feedback $_{speech}$; $ps > .05$).

Postevent processing (PEP). One hour after the TSST-C ended, the children completed the Thoughts Questionnaire for Children (Schmitz et al., 2010), a child-adapted version of the Thoughts Questionnaire (Edwards, Rapee, & Franklin, 2003). The questionnaire comprises eight items about positive and eight items about negative thoughts the child might have had about the stressor (“How often did you think: I performed well”; “. . . the scientists did not like me”). All items were rated on a 6-point Likert scale ranging from 1 (*not at all*) to 6 (*very often*). All items about positive thoughts were added together to create a total sum score for positive thoughts ($PEP_{positive}$). All negative thought items were summed to create a total sum score for negative thoughts ($PEP_{negative}$). The two subscales for positive and negative PEP have a maximum score of 48 each. Cronbach α indicated high internal consistency for both the positive and the negative PEP scales ($\alpha = 0.89$ and 0.88 , respectively) in

the current sample. There was a significant negative correlation between the two subscales ($r = -.22$, $p < .01$).

Pubertal status. Children and adolescents rated their pubertal status according to schematic pictures of the five tanner stages (Morris & Udry, 1980). Ratings of genital hair growth and size of breasts/testicles were combined into a general rating (mean score) and dichotomized according to Rapkin, Tsao, Turk, Anderson, and Zeltzer’s (2006) criteria. Tanner Stages I and II describe a prepubertal and early pubertal status, whereas Tanner Stages III and IV describe middle to late pubertal status. Interrater reliability between self-ratings and professional ratings was tested in a child psychiatric sample of 50 children and adolescents between the ages of 8 to 15 years. The correlation between the self and professional ratings was high (Spearman correlation coefficient genital hair growth: $r = .81$, size of breasts/testicles: $r = .85$). Age and self-rated pubertal status (mean score) were significantly positively associated ($r = .75$, $p < .001$).

Data analytic strategy

To analyze the data, we used the statistical software IBM SPSS Statistics 23. For descriptive analyses, differences concerning age, pubertal status, gender, and SES between the four diagnostic groups were analyzed with an analysis of variance (ANOVA) for age and Pearson chi-square tests for pubertal status, gender, and SES. To analyze the bivariate associations between cortisol and the subjective stress response parameters, we calculated Pearson correlation coefficients (r). To analyze the effects of age, pubertal status, and gender concerning stress response parameters, we computed multivariate analyses of variance (MANOVAs). To answer our research questions about differences in single cortisol and subjective stress response parameters in the diagnostic groups, we calculated univariate ANOVAs with single stress-response parameters (cortisol levels, subjectively experienced arousal, performance ratings, and postevent processing) as dependent variables and age and gender as control variables.

To analyze the predictive value of cortisol and subjective stress responses on the diagnostic groups, we calculated multinomial regression analyses. The diagnostic groups were the dependent variable with the healthy control (HC) group as the reference group; the cortisol response (AUC_g/AUC_i), the subjectively experienced arousal ($AROUS_{mean}$), and the postevent processing ($PEP_{negative}/PEP_{positive}$) were the potential predictors of the diagnostic groups. In this regard, the statistical predictions do not imply any direction of effect, because of our cross-sectional design.

Due to high collinearity between children’s self-rated performance ($PERF_{mean}$) and their postevent processing ($PEP_{negative}$), we excluded $PERF_{mean}$ from the analyses. Moreover, due to the high correlation between AUC_g and AUC_i , we performed separate multinomial regression analyses with the two cortisol parameters. In all analyses, we controlled for gender, age, and pubertal status. Because

1. 80 = 80 min from time points T_3 to T_8 .

of the collinearity of the latter two, we performed analyses with either age or pubertal status. Because the results of these analyses were similar, we chose to present the analyses with age as a control variable. Finally, we analyzed interaction effects between cortisol and subjective stress response parameters on psychiatric disorders by including Cortisol \times Subjective Stress Response interaction terms into the above-mentioned regression analyses. Components of the interaction term (cortisol [AUC_g, AUC_i] and subjective stress response measures (AROUS_{mean}, PEP_{negative}, PEP_{positive}) centered on their means to reduce collinearity and facilitate the interpretation of interactions (Kraemer & Blasey, 2004). Significance was set at $p < .05$. When post hoc tests were required, we used the Games–Howell procedure. As effect size measures, we used partial eta-squared (η_p^2) for the ANOVAs and Cramer V for the chi-square tests.

To disentangle possible differences between children with a pure depressive disorder (pureDEPR) and children with depressive and anxiety disorders (DEPR_ANX), we calculated additional multiple regression analyses and differentiated between pureDEPR and DEPR_ANX. Because of the small sample sizes, we present the results of these analyses as exploratory analyses.

Results

Participant characteristics

Table 2 shows demographic and psychometric measures for the diagnostic groups. Children in the DEPR group were significantly older and had significantly more often a middle/high pubertal status than the children in the other three diagnostic groups (ANX, EXT, and HC). The SES of the parents differed significantly across the diagnostic groups with significantly more families with a low status in the ANX and EXT groups compared with the HC group and significantly more families with a high status in the HC group compared with the EXT group. No gender differences were found across the four diagnostic groups.

Bivariate associations between cortisol and subjective TSST-C-response parameters

The results of the correlation analyses are shown in Table 3. We found no significant correlation between cortisol and the subjective stress response parameters, except one significant negative correlation between an increase in cortisol and children's performance rating immediately after the TSST-C ($r = -.17, p < .05$). That is, the higher the cortisol increase was, the worse the children's performance rating was. Separate correlation analyses within the subgroup of children with a psychiatric disorder and the subgroup of healthy controls did not show any significant associations between cortisol and subjective stress response measures (all $ps > .05$). In addition, we found a significant positive correlation between the two cortisol stress parameters, that is, between the total amount of cortisol (AUC_g) and the cortisol increase (AUC_i; $r = .75, p < .01$). For the subjective stress parameters, we found that children's subjectively experienced arousal was significantly negatively correlated with children's performance rating immediately after the TSST-C ($r = -.196, p < .05$) and significantly positively correlated with their negative thoughts 1 hr later ($r = .42, p < .01$). Further, children's performance rating immediately after the TSST-C was significantly negatively correlated with negative thoughts ($r = -.47, p < .01$) and significantly positively correlated with positive thoughts 1 hr after the TSST-C ($r = .397, p < .01$). Finally, positive and negative thoughts were significantly negatively correlated ($r = -.20, p < .01$).

Associations of stress-related parameters with confounding variables (gender, age, pubertal status)

To analyze gender differences in the total sample, we computed a MANOVA with all TSST-C measures (AUC_g, AUC_i, AROUS_{mean}, PERF_{mean}, PEP_{positive}, PEP_{negative}) as dependent variables. The multivariate results showed a significant effect of gender (Wilks $\lambda = 0.91, p = .034, \eta_p^2 = 0.09$). According to the univariate results, this significant effect was due to AUC_g and negative thoughts. Girls showed significantly

Table 3. Bivariate correlations (Pearson) between cortisol and subjective TSST-C response parameters (total sample)

Measure	1	2	3	4	5
1. AUC _g	—				
2. AUC _i	.75**	—			
3. AROUS _{mean}	.05	.06	—		
4. PERF _{mean}	-.04	-.17*	-.20*	—	
5. PEP _{positive}	-.05	.01	.03	.40**	—
6. PEP _{negative}	.03	.01	.42**	-.47**	-.20**

Note: TSST-C, Trier Social Stress Test for Children; AUC_g, area under the curve with respect to ground; AUC_i, area under the curve with respect to increase; AROUS_{mean}, mean subjectively experienced arousal; PERF_{mean}, mean performance ratings; PEP_{positive}/PEP_{negative}, positive/negative postevent processing.

* $p < .05$. ** $p < .01$.

higher values of AUC_g than boys (girls: $M = 34.87$, $SD = 12.90$; boys: $M = 30.72$, $SD = 9.90$), $F(1, 144) = 4.77$, $p = .031$, $\eta_p^2 = 0.03$. Moreover, girls showed significantly higher ratings on the negative thought items than boys ($PEP_{negative}$: girls: $M = 20.56$, $SD = 10.97$; boys: $M = 16.07$, $SD = 11.65$), $F(1, 144) = 5.65$, $p = .019$, $\eta_p^2 = 0.04$. We found no significant gender differences concerning AUC_i , subjectively experienced arousal, performance ratings, and positive thoughts (all $ps > .05$).

To analyze differences due to pubertal status, we again computed a MANOVA with all TSST-C measures as dependent variables. The multivariate result showed a significant effect of pubertal status (Wilks $\lambda = 0.90$, $p = .023$, $\eta_p^2 = 0.10$). According to the univariate results, this significant effect was due to children's performance rating as well as their positive and negative thoughts. Ratings of negative thought items were higher in children with a higher pubertal level (*pre/early*: $M = 16.27$, $SD = 11.91$; *middle/late*: $M = 20.29$, $SD = 11.06$), $F(1, 138) = 4.25$, $p = .041$, $\eta_p^2 = 0.03$. By contrast, performance ratings (*pre/early*: $M = 3.46$, $SD = 1.11$; *middle/late*: $M = 2.84$, $SD = 1.08$), $F(1, 138) = 11.22$, $p = .001$, $\eta_p^2 = 0.08$, and ratings of positive thought items (*pre/early*: $M = 8.87$, $SD = 8.98$; *middle/late*: $M = 5.03$, $SD = 5.54$), $F(1, 138) = 8.91$, $p = .003$, $\eta_p^2 = 0.06$, were lower in children with a higher pubertal level. Cortisol response and subjectively experienced arousal did not differ by children's pubertal status (both $ps > .05$).

To analyze the effects of age (dimensional variable) on all TSST-C measures, a MANOVA was again calculated. The multivariate result showed a significant effect of age (Wilks $\lambda = 0.88$, $p = .005$, $\eta_p^2 = 0.12$). According to the univariate results, this significant effect was due to children's performance ratings and their positive thoughts. Performance ratings ($B = -0.16$), $F(1, 144) = 13.13$, $p = .001$, $\eta_p^2 = 0.08$, as well as ratings of positive thought items ($B = -1.12$), $F(1, 144) = 12.92$, $p = .001$, $\eta_p^2 = 0.08$, were lower in older children. Cortisol response (AUC_g and AUC_i), subjectively experienced arousal, and negative thoughts did not differ by children's age (all $ps > .05$).

Age, pubertal status, and gender turned out to be significantly associated with different study variables. Because of the high correlation between age and pubertal status, we used only age and gender as covariates in the following multivariate analyses.

Cortisol response, subjectively experienced arousal, performance ratings, and PEP in the diagnostic groups

Cortisol response. The patterns of the cortisol-level responses (original cortisol values) before and after the TSST-C are shown in Figure 2 for all four diagnostic groups (DEPR, ANX, EXT, and HC). We calculated ANOVAs with AUC_g and AUC_i (Table 4). A 4×2 , Group \times Gender ANOVA with age as a continuous control variable and AUC_g as the dependent variable yielded a significant main effect of group, $F(3, 143) = 4.79$, $p = .003$, $\eta_p^2 = 0.09$. Post hoc tests revealed

significantly lower total cortisol levels in the ANX ($p = .001$) and EXT groups ($p = .031$) compared with the HC group. Gender, $F(1, 141) = 2.89$, $p = .091$, and age, $F(1, 141) = 0.135$, $p = .714$, were not significant variables.

An ANOVA with AUC_i as the dependent variable yielded a significant main effect of group, too, $F(3, 143) = 5.57$, $p = .001$, $\eta_p^2 = 0.11$. Post hoc tests revealed significantly lower cortisol increases in all three disorder groups compared with the HC group, all $ps < .05$. Again, gender, $F(1, 141) = 0.510$, $p = .476$, and age, $F(1, 141) = 0.388$, $p = .534$, were not significant variables.

Subjectively experienced arousal ($AROUS_{mean}$). A 4×2 , Group \times Gender ANOVA with age as a continuous control variable and $AROUS_{mean}$ as the dependent variable (Table 4) yielded a significant main effect of group, $F(3, 163) = 4.61$, $p = .004$, $\eta_p^2 = 0.08$. Post hoc tests revealed significantly higher subjectively experienced arousal with respect to the TSST-C in children in the ANX group compared with the HC group ($p = .016$). Gender, $F(1, 163) = .012$, $p = .915$, and age, $F(1, 163) = .242$, $p = .624$, were not significant variables.

Performance rating ($PERF_{mean}$). A 4×2 , Group \times Gender ANOVA with age as a continuous control variable and $PERF_{mean}$ as the dependent variable (Table 4) showed a significant main effect of group, $F(3, 164) = 3.27$, $p = .023$, $\eta_p^2 = 0.06$. Post hoc tests revealed significantly lower scores in the DEPR group compared with the HC group ($p = .006$). Moreover, we found a significant main effect of age, $F(1, 164) = 4.62$, $p = .033$, $\eta_p^2 = 0.03$, with lower $PERF_{mean}$ scores in older children. Gender was not a significant variable, $F(1, 164) = 2.86$, $p = .093$.

PEP ($PEP_{positive}/PEP_{negative}$). Negative and positive PEP for all four diagnostic groups (DEPR, ANX, EXT, and HC) are shown in Figure 3. A 4×2 , Group \times Gender ANOVA with age as a continuous control variable and $PEP_{positive}$ as the dependent variable yielded a significant main effect of group, $F(3, 164) = 3.98$, $p = .009$, $\eta_p^2 = 0.07$. Post hoc tests revealed significantly lower $PEP_{positive}$ scores in the DEPR group compared with the ANX ($p = .030$) and EXT ($p = .010$) groups. Moreover, we found a significant main effect of age, $F(1, 164) = 11.42$, $p = .001$, $\eta_p^2 = 0.07$, with lower $PEP_{positive}$ scores in older children. Gender was not a significant variable, $F(1, 164) = 0.000$, $p = .992$.

An ANOVA with $PEP_{negative}$ as the dependent variable yielded a significant main effect of group, too, $F(3, 164) = 2.89$, $p = .037$, $\eta_p^2 = 0.05$. Post hoc tests revealed significantly higher $PEP_{negative}$ scores in the DEPR group compared with the other three diagnostic groups (all $ps < .05$). Moreover, we found a significant main effect of gender, $F(1, 164) = 4.74$, $p = .031$, $\eta_p^2 = 0.03$, with higher $PEP_{negative}$ scores in girls. Age was not a significant variable, $F(1, 164) = 0.300$, $p = .585$.

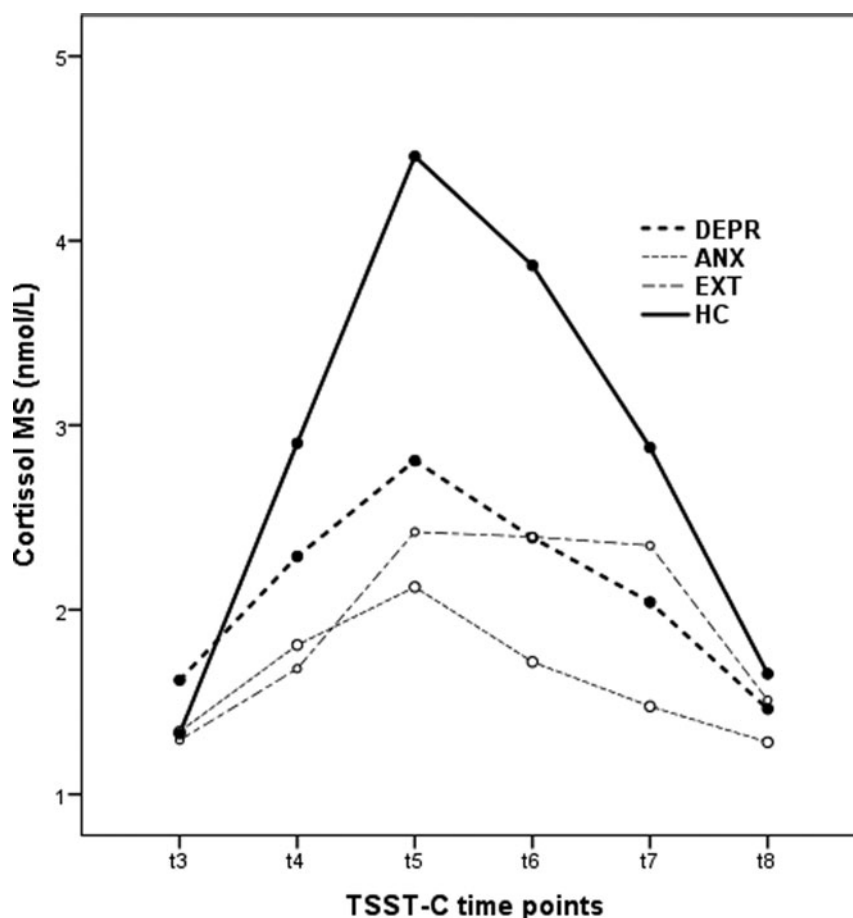


Figure 2. Cortisol responses before and after the Trier Social Stress Test for Children (TSST-C) in the four diagnostic groups. DEPR, depression group; ANX, anxiety group; EXT, externalizing disorder group; HC, healthy control group; TSST-C time points: $t_3 = -1$ min before the TSST-C; $t_4 = +1$ min, $t_5 = +10$ min, $t_6 = +20$ min, $t_7 = +30$ min, and $t_8 = +60$ min after the TSST-C.

Multinomial regression analyses

To analyze the associations of cortisol and subjective stress responses with the diagnostic groups, we calculated multinomial regression analyses² (see Tables 5 and 6).

Depressive disorder. Analyses with AUC_g as a cortisol predictor showed that higher $PEP_{negative}$, Wald $\chi^2(1) = 5.16$, $p = .023$, odds ratio (OR) = 1.07, and higher age, Wald $\chi^2(1) = 12.23$, $p = .000$, $OR = 1.82$, significantly predicted membership in the depressive disorder group versus the healthy control group. AUC_g did not predict depressive disorders (see Table 5). Analyses with AUC_i (see Table 6) showed that lower AUC_i , Wald $\chi^2(1) = 4.33$, $p = .037$, $OR = 0.94$; higher $PEP_{negative}$, Wald $\chi^2(1) = 6.93$, $p = .008$, $OR = 1.88$;

and higher age, Wald $\chi^2(1) = 14.38$, $p = .000$, $OR = 1.92$, significantly predicted membership in the depressive disorder group. $AROUS_{mean}$, $PEP_{positive}$, and gender were not significant predictors of a depressive disorder in any of the analyses.

Anxiety disorder. Analyses with AUC_g as a cortisol predictor showed that lower AUC_g , Wald $\chi^2(1) = 9.22$, $p = .002$, $OR = 0.92$, as well as higher $AROUS_{mean}$, Wald $\chi^2(1) = 8.93$, $p = .003$, $OR = 1.60$, significantly predicted membership in the anxiety disorder group versus the healthy control group (see Table 5). Analyses with AUC_i as a cortisol predictor (see Table 6) also showed that lower AUC_i , Wald $\chi^2(1) = 9.77$, $p = .002$, $OR = 0.93$, and higher $AROUS_{mean}$, Wald $\chi^2(1) = 7.38$, $p = .007$, $OR = 1.48$, were significant predictors. $PEP_{negative}$, $PEP_{positive}$, gender, and age did not predict membership in the anxiety group in any of the analyses.

Externalizing disorder. Analyses with AUC_g showed that lower AUC_g , Wald $\chi^2(1) = 7.81$, $p = .005$, $OR = 0.93$, higher $PEP_{positive}$, Wald $\chi^2(1) = 8.63$, $p = .003$, $OR = 1.10$, and higher age, Wald $\chi^2(1) = 6.12$, $p = .013$, $OR = 1.42$, significantly predicted membership in the externalizing disorder

2. Because of the small group sizes we were limited in the number of variables to be included in the multivariate analyses. Therefore, we report analyses in which we exclusively controlled for child-related factors and refrained from controlling for other variables, such as the parental SES (Rajmil et al., 2014). When including the SES in the regression analyses, the results basically remained the same with slightly lower significance levels of stress response parameters.

Table 4. Group differences in cortisol and subjective TSST-C response parameters between the diagnostic groups

Parameter	M (SD)				Test Statistic ^a
	DEPR (n = 21–26)	ANX (n = 28–32)	EXT (n = 29–36)	HC (n = 69–76)	
AUC _g	32.42 (9.95)	28.09 _a (7.35)	28.47 (11.31)	36.18 _b (12.32)	$F(3, 143) = 4.79$, $p = .003$, $\eta_p^2 = 0.09$
AUC _i	-0.09 _a (8.46)	-0.35 _a (8.10)	0.40 _a (11.00)	7.97 _b (13.77)	$F(3, 143) = 5.57$, $p = .001$, $\eta_p^2 = 0.11$
AROUS _{mean}	4.24 (2.10)	4.43 _a (2.02)	3.80 (1.98)	3.22 _b (1.36)	$F(3, 163) = 4.61$, $p = .004$, $\eta_p^2 = 0.08$
PERF _{mean}	2.52 _a (0.21)	3.13 (0.19)	2.93 (0.18)	3.41 _b (0.12)	$F(3, 164) = 3.27$, $p = .023$, $\eta_p^2 = 0.06$
PEP _{positive}	3.81 _a (1.43)	8.59 _b (1.29)	9.94 _b (1.21)	5.84 (0.83)	$F(3, 164) = 3.98$, $p = .009$, $\eta_p^2 = 0.07$
PEP _{negative}	25.27 _a (9.01)	16.34 _b (10.75)	17.89 _b (11.28)	17.43 _b (11.46)	$F(3, 164) = 2.89$, $p = .037$, $\eta_p^2 = 0.05$

Note: TSST-C, Trier Social Stress Test for Children; DEPR, depression group; ANX, anxiety group; EXT, externalizing disorder group; HC, healthy control group; AUC_g, area under the curve with respect to ground; AUC_i, area under the curve with respect to increase; AROUS_{mean}, mean subjectively experienced arousal; PERF_{mean}, mean performance ratings; PEP_{positive}/PEP_{negative}, positive/negative postevent processing. Different subscript letters indicate number/frequency of significant group differences found in post hoc analyses (Games–Howell).

^aAnalysis of variance, controlled for age and gender.

group versus the healthy control group (see Table 5). Analyses with AUC_i (see Table 6) also showed that lower AUC_i, Wald $\chi^2(1) = 8.00$, $p = .005$, $OR = 0.93$, higher PEP_{positive}, Wald $\chi^2(1) = 10.46$, $p = .001$, $OR = 1.12$, higher age, Wald $\chi^2(1) =$

7.61, $p = .006$, $OR = 1.47$, and male gender, Wald $\chi^2(1) = 6.25$, $p = .012$, $OR = 3.64$, were predictive of externalizing disorders. AROUS_{mean} and PEP_{negative} did not predict membership in the externalizing disorder group in any of the analyses.

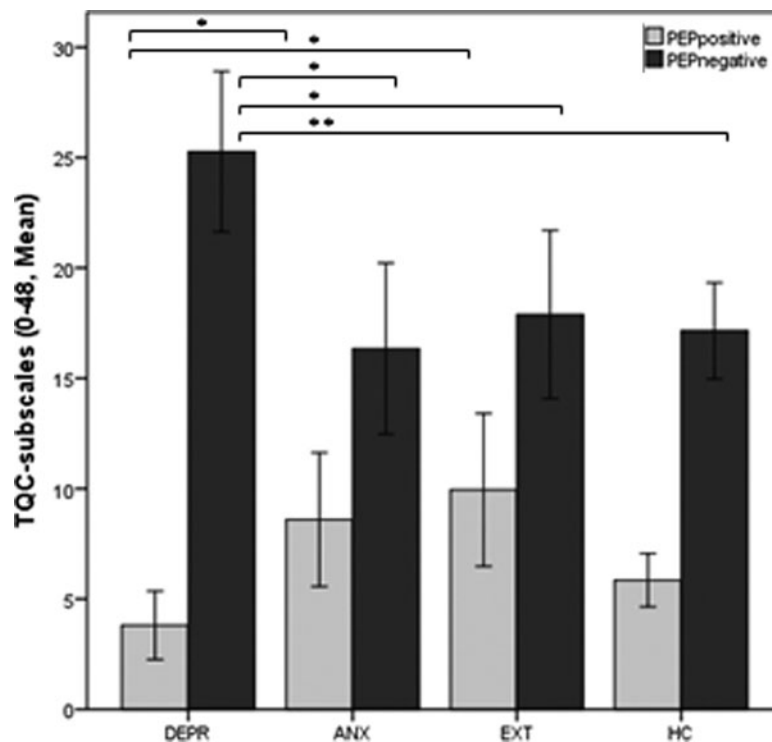


Figure 3. Positive and negative postevent processing 1 hr after the Trier Social Stress Test for Children (TSST-C) in the four diagnostic groups. TQC, Thoughts Questionnaire for Children; PEP_{positive}/PEP_{negative}, positive/negative postevent processing; DEPR, depression group; ANX, anxiety group; EXT, externalizing disorder group; HC, healthy control group. Significant group differences found in post hoc analyses (Games–Howell): * $p < .05$, ** $p < .01$.

Table 5. Multinomial regression analysis with AUC_g as cortisol parameter

Comparison	<i>B</i> (<i>SE</i>)	<i>OR</i>	95 % CI for <i>OR</i>
DEPR vs. HC			
Intercept	-9.34 (2.56)		
Age	0.60 (0.17)**	1.82	[1.30, 2.54]
Gender (boy)	0.38 (0.62)	1.46	[0.43, 4.95]
AUC_g	-0.04 (0.03)	0.96	[0.91, 1.01]
$AROUS_{mean}$	0.24 (0.18)	1.27	[0.89, 1.81]
$PEP_{negative}$	0.07 (0.03)*	1.07	[1.01, 1.14]
$PEP_{positive}$	-0.02 (0.06)	0.98	[0.87, 1.10]
ANX vs. HC			
Intercept	-1.25 (2.02)		
Age	0.14 (0.15)	1.15	[0.87, 1.53]
Gender (boy)	-0.03 (0.53)	0.97	[0.34, 2.75]
AUC_g	-0.08 (0.03)**	0.92	[0.87, 0.97]
$AROUS_{mean}$	0.47 (0.16)**	1.60	[1.18, 2.17]
$PEP_{negative}$	-0.04 (0.03)	0.96	[0.92, 1.02]
$PEP_{positive}$	0.05 (0.03)	1.05	[0.98, 1.13]
EXT vs. HC			
Intercept	-4.38 (2.03)		
Age	0.35 (0.14)*	1.42	[1.08, 1.88]
Gender (boy)	0.56 (0.55)	1.76	[0.60, 5.12]
AUC_g	-0.08 (0.03)**	0.93	[0.88, 0.98]
$AROUS_{mean}$	0.26 (0.16)	1.29	[0.94, 1.77]
$PEP_{negative}$	0.00 (0.03)	1.00	[0.95, 1.05]
$PEP_{positive}$	0.10 (0.03)**	1.10	[1.03, 1.18]

Note: AUC_g , area under the curve with respect to ground; DEPR, depression group; HC, healthy control group; $AROUS_{mean}$, mean subjectively experienced arousal; $PEP_{positive}/PEP_{negative}$, positive/negative postevent processing; ANX, anxiety group; EXT, externalizing disorder group. $n = 162$; $R^2 = .378$ (Cox & Snell); $R^2 = .411$ (Nagelkerke); model $\chi^2(18) = 69.38$; $p < .01$. * $p < .05$. ** $p < .01$.

Interaction effects between cortisol and subjective stress response parameters as predictors of psychiatric disorders. We analyzed the predictive value of the interaction between cortisol and subjective stress response measures in the diagnostic groups by adding AUC_g or $AUC_i \times AROUS_{mean} / \times PEP_{negative} / \times PEP_{positive}$ interaction terms into the above-mentioned regression analyses. For depressive disorders, the main effects of predictors in the regression analyses remained the same after inclusion of the interaction terms. None of the interaction terms were significant predictors. For anxiety disorders, higher positive cognitions turned out to be an additional significant predictor in the analysis with AUC_g , Wald $\chi^2(1) = 4.23$, $p = .040$, $OR = 1.08$ (in addition to the significant predictors AUC_g , AUC_i , and subjectively experienced arousal). None of the interaction terms significantly predicted membership of the anxiety disorder group. For externalizing disorders, the main effects of predictors in the regression analysis with AUC_i remained the same. In the analysis with AUC_g , the main effect of positive cognitions was no longer significant after inclusion of the interaction effects. Instead, the interaction effect of $AUC_g \times AROUS_{mean}$ turned out to be a significant predictor of externalizing disorders, Wald $\chi^2(1) = 4.76$, $p = .029$, $OR = 1.03$. To illustrate this interaction effect, we dichotomized AUC_g and $AROUS_{mean}$ at

Table 6. Multinomial regression analysis with AUC_i as cortisol parameter

Comparison	<i>B</i> (<i>SE</i>)	<i>OR</i>	95% CI for <i>OR</i>
DEPR vs. HC			
Intercept	-11.63 (2.53)		
Age	0.65 (0.17)**	1.92	[1.37, 2.697]
Gender (boy)	0.96 (0.62)	2.62	[0.78, 8.78]
AUC_i	-0.06 (0.03)*	0.94	[0.89, 1.00]
$AROUS_{mean}$	0.18 (0.18)	1.195	[0.84, 1.69]
$PEP_{positive}$	-0.02 (0.06)	0.99	[0.88, 1.11]
$PEP_{negative}$	0.08 (0.03)*	1.08	[1.02, 1.15]
ANX vs. HC			
Intercept	-4.797 (1.84)		
Age	0.18 (0.14)	1.198	[0.91, 1.58]
Gender (boy)	0.74 (0.49)	2.10	[0.80, 5.52]
AUC_i	-0.08 (0.02)**	0.93	[0.88, 0.97]
$AROUS_{mean}$	0.39 (0.14)*	1.48	[1.12, 1.96]
$PEP_{positive}$	0.06 (0.03)	1.06	[0.99, 1.13]
$PEP_{negative}$	-0.02 (0.03)	0.98	[0.93, 1.03]
EXT vs. HC			
Intercept	-7.54 (1.94)		
Age	0.39 (0.14)*	1.47	[1.12, 1.93]
Gender (boy)	1.29 (0.52)*	3.64	[1.32, 10.00]
AUC_i	-0.07 (0.03)*	0.93	[0.89, 0.98]
$AROUS_{mean}$	0.18 (0.15)	1.199	[0.89, 1.61]
$PEP_{positive}$	0.10 (0.03)**	1.11	[1.04, 1.18]
$PEP_{negative}$	0.01 (0.03)	1.01	[0.96, 1.06]

Note: AUC_i , area under the curve with respect to increase; DEPR, depression group; HC, healthy control group; $AROUS_{mean}$, mean subjectively experienced arousal; $PEP_{positive}/PEP_{negative}$, positive/negative postevent processing; ANX, anxiety group; EXT, externalizing disorder group. $n = 162$; $R^2 = .376$ (Cox & Snell); $R^2 = .415$ (Nagelkerke); model $\chi^2(18) = 79.83$, $p < .0$. * $p < .05$. ** $p < .01$.

the mean. Figure 4 shows that the combination low AUC_g /high subjectively experienced arousal occurred significantly more often in children with externalizing disorders, whereas the combination high AUC_g /low subjectively experienced arousal occurred significantly less often in children with externalizing disorders.

Explorative analyses of the subgroups pureDEPR versus DEPR_ANX. For pureDEPR, higher $PEP_{negative}$ turned out to be the only predictor, analysis with AUC_g : Wald $\chi^2(1) = 6.97$, $p = .008$, $OR = 1.10$; analysis with AUC_i : Wald $\chi^2(1) = 6.43$, $p = .011$, $OR = 1.10$. For DEPR_ANX, lower AUC_i , Wald $\chi^2(1) = 4.81$, $p = .028$, $OR = 0.87$, and higher $AROUS_{mean}$, analysis with AUC_g : Wald $\chi^2(1) = 3.95$, $p = .046$, $OR = 1.66$; analysis with AUC_i : Wald $\chi^2(1) = 3.95$, $p = 1.82$, $OR = 0.87$, were significant predictors.

Discussion

Our results indicate that both cortisol and cognitive-emotional responses to a standardized stress test are associated with children's depressive, anxiety, or externalizing disorders. Children with depressive, anxiety, or externalizing disorders all exhibited a lower increase in cortisol after the stress test as compared with

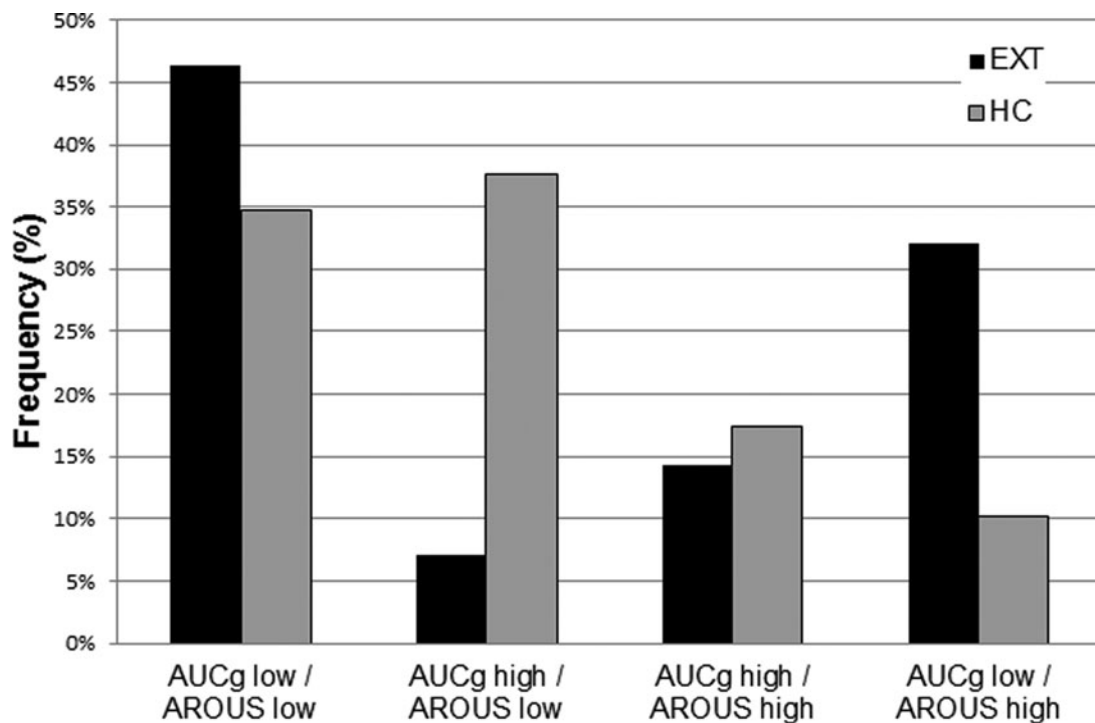


Figure 4. Interaction between AUCg and subjectively experienced arousal in children with externalizing disorders and healthy controls. AUC_g, area under the curve with respect to ground, dichotomized: low \leq mean/high $>$ mean; AROUS, mean subjectively experienced arousal, dichotomized: low \leq mean/high $>$ mean; EXT, externalizing disorder group; HC, healthy control group. Significant group differences found in post hoc analyses (standardized residuals): * $p < .05$.

healthy controls, and those with anxiety or externalizing disorders also showed a reduction in the total release of cortisol. Moreover, children in the different disorder groups exhibited different patterns of subjective stress reactions when compared with the healthy control group: the depressed children rated their test performance lower and showed higher ratings of negative thoughts 1 hr after the test, whereas the children with an externalizing disorder showed higher ratings of positive thoughts. By contrast, the anxious children did not differ from their healthy counterparts with respect to their long-term thoughts, but they reported more arousal before and after the task.

When analyzing the association between endocrinological and subjective stress reactions in the total sample, we found a small negative correlation between the increase in cortisol (AUC_i) and children's performance rating but not with other subjective parameters. Children who showed a greater increase in cortisol during the TSST-C gave lower ratings of their performance during the stress test. When analyzing the association between endocrinological and subjective stress reactions within the group with psychiatric disorders and within the healthy control group separately, we found no significant associations. This is in contrast to our hypotheses and contradicts earlier studies, which found a discrepancy between subjectively experienced arousal and physiological stress responses in psychiatric groups, but a concordance in healthy controls (Fairchild et al., 2008; Kircanski, Waugh, et al., 2016). However, Dieleman et al. (2010) also found that 8- to 12-year-old

children's perceived arousal after a stress test was not significantly correlated with their cortisol responses.

In contrast to findings on the association between stress-related state rumination and cortisol reactivity in adults (Zoccola & Dickerson, 2012), we found no significant association between stress-related negative thoughts and cortisol in our study. Our result is in line with findings in a child sample by Rudolph et al. (2011). Moreover, most interaction effects between the subjective and endocrinological stress reaction were not associated with diagnostic groups, with one exception (see description below on results concerning externalizing disorders). A discordance of subjective and endocrinological stress reactions might be characteristic for the age group of our study. Even though we found a small interdependency in the total sample, we were not able to determine whether endocrinological and subjective stress reactions (subjectively experienced arousal and thoughts) are independent from each other or not. Perhaps we were simply not able to detect the existing interrelations with the measures we used. It is possible that the cortisol stress response represents a stable trait, whereas the cognitive-emotional responses, as measured in our study, represent state aspects. This idea is supported by the findings of a study by van West et al. (2008) in which trait but not state anxiety was associated with higher HPA axis activity. Moreover, trait rumination in response to depressed mood was found to be associated with delayed poststressor cortisol recovery in depressed adolescents (Stewart et al.,

2013). In contrast, it might also be the case that cortisol and the assessed cognitive–emotional responses were not found to be associated, because we assessed explicitly perceived cognitive–emotional stress reactions, whereas physiological (e.g., cortisol) measures represent more cognitive–emotional stress reactions below the threshold of explicit awareness.

In line with our hypothesis, we found a dysregulated cortisol response in preadolescents and young adolescents with depressive, anxiety, or externalizing disorders compared with healthy controls. Children with psychiatric disorders showed a significantly smaller increase in cortisol after the stress condition, and those with anxiety or externalizing disorders also showed a smaller total release of cortisol. So far, the literature on children with internalizing disorders has revealed inconsistent findings on their stress-related cortisol response. Some studies found a stress-related hyperreactivity of cortisol (Lopez-Duran et al., 2009; Rao et al., 2008; van West et al., 2008), whereas other studies found a hyporeactivity of cortisol in children with internalizing disorders (Ayer et al., 2013; de Rooij, 2013). These contradictory results might be due to developmental effects on cortisol reactivity. Developmental changes during the transition from middle childhood to adolescence were found to be associated with alterations in physiological stress response systems (e.g., Gunnar, Wewerka, et al., 2009; Stroud et al., 2009). However, in contrast to developmental effects on basal cortisol levels, effects of age and pubertal status on stress-related cortisol reactivity were not reported consistently (Gunnar, Wewerka, et al., 2009). In our study, cortisol responses (AUC_g and AUC_i) did not differ according to children's pubertal status or age. Even though we had quite a wide age range, most of our participants had an early to middle pubertal status (Tanner stadium I–II, 79% of the sample). Our result corresponds with the results of Gunnar, Wewerka, et al. (2009), who found no significant effects of sexual maturation on cortisol reactivity. Instead, in the total sample, we found a significant gender effect that indicated a higher total cortisol release (AUC_g) in girls than in boys. This finding is in line with results reported in a review on this topic (Gunnar, Talge, et al., 2009). Even though we did not find significant gender differences concerning our diagnostic groups, the higher cortisol reactivity in girls might indicate on their vulnerability for internalizing disorders (Gunnar, Wewerka, et al., 2009).

Previous studies provide evidence that blunted cortisol reactivity was related to a dysphoric state in prepubertal youth (Hankin et al., 2010) and predicted the onset of MDD in girls who were at an earlier stage of puberty (Tanner stage ≤ 2 ; Colich et al., 2015). To our knowledge, there is no study of the developmental profile of cortisol stress reactivity in children and adolescents with anxiety disorders. However, in line with studies of depressive disorders, the blunted cortisol reactivity in children with anxiety disorders in our findings might be due to the high proportion of prepubertal to early pubertal children (Tanner stadium I–II) in this group. In contrast, the blunted cortisol reaction we found in the depression group seems to contradict earlier findings on developmental effects

by Hankin et al. (2010) and Colich et al. (2015), as our depression group mainly included adolescents with Tanner stadium III–IV (and only one adolescent with Tanner stadium V). It is possible that a hyperreactivity of cortisol can only be detected in postpubertal youth.

In contrast, our results might also be influenced by the chronicity of the disorder. Booij et al. (2013), for example, reported that the stress response changed from cortisol hyper- to hyporeactivity as depressive problems continued for a longer period of time. According to Petrowski, Wintermann, Schaarschmidt, Bornstein, and Kirschbaum (2013), who found reduced cortisol in patients with panic disorder, the hyporeactivity might be due to a habituation effect. In line with this idea, Gunnar and Vazquez (2001) argued in their review that hyporeactivity might be an adaptive reaction that follows from the frequent experience of stressors. Recent studies have shown that exposure to stressful life events and chronic stress are associated with blunted cortisol stress reactions (Fries et al., 2005; Jaffee et al., 2015).

In line with earlier studies (e.g., Hartman et al., 2013; Randazzo et al., 2008), we found a blunted cortisol stress response in children with an externalizing disorder. Our findings are in line with the underarousal theory for externalizing disorders (Raine, 2002), which suggests that chronic underactivation of the HPA axis and the adrenergic nervous system promotes a fearless personality and drives individuals to seek stimuli that will reduce the uncomfortable state. The interplay between cortisol reactivity and subjectively experienced arousal seemed to be associated with externalizing disorders. Low AUC_g combined with high subjectively experienced arousal were associated with a significantly higher risk of externalizing disorders. The finding is contrary to our theoretical expectations. A closer look at further characteristics of this specific group of children as well as their future development is needed to understand this finding.

Partly in line with our hypothesis and in line with previous findings of Dieleman et al. (2010) and Krämer et al. (2012), we found that children with at least one anxiety disorder were significantly more aroused by the TSST-C than healthy controls. No significant differences were found between healthy controls and children with a depressive or externalizing disorder. This difference in the mean of children's subjectively experienced arousal in children with anxiety disorders was based on the ratings that were given after the TSST-C. These children mainly seem to have difficulties downregulating their arousal after experiencing the stressor. Thus, our result might reflect that anxious children have trouble withdrawing their attention from negative stimuli (Gotlib & Joormann, 2010; Mathews & MacLeod, 2005). According to previous findings (e.g., Stroud et al., 2009), subjectively experienced arousal did not change with age.

In partial agreement with our hypothesis and with the known tendency of children with depressive disorders to engage in negative self-evaluation (Beck, 1967; de Raedt & Koster, 2010; Sass et al., 2003), we found that children with a depressive disorder, but not those with anxiety disorder

ders, gave significantly lower performance ratings (mean rating speech/calculation) than healthy controls. Thereby, the performance ratings were not affected by the amount of negative feedback given by the “scientific committee” during the stress test. Performance ratings were instead associated with age and pubertal status, showing that children gave worse ratings of their performance with growing age and pubertal status. This result is in accordance with findings on children’s decreasing evaluations of scholastic competence between childhood and adolescence (Harter & Whitesell, 2003; Stadelmann et al., 2017).

Again, partly in line with our hypothesis, we found that children with a depressive disorder, but not those with an anxiety disorder, reported significantly more negative thoughts than healthy controls 1 hr after the TSST-C. Our results support findings on adults with a depressive disorder, who tended to engage in postevent negative thoughts about a stressor (so-called stress-reactive rumination; Alloy et al., 2000; Robinson & Alloy, 2003). However, our study did not replicate the results of Schmitz et al. (2010), who found significantly higher stress-related negative thoughts in children with social phobia as well.

Furthermore, negative thoughts were affected by gender, age, and pubertal status. We found that girls showed higher ratings of negative thoughts than boys. Similar effects were reported by Johnson and Whisman (2013), who found more rumination in women than in men. In our study, negative thought ratings also increased with higher age and pubertal status. These findings are in line with the results of a study by Rood et al. (2010), who found a slight increase in stress-reactive rumination with age in a nonclinical sample of 10- to 18-year-olds. In addition, children’s negative thoughts were connected to their own performance ratings immediately after the stress test (t_4) and their subjectively experienced arousal before and after the TSST-C (mean of ratings t_3 to t_8 ; both correlations: medium-sized effects). Higher subjectively experienced arousal and worse performance ratings went along with more negative thoughts.

In general, our findings on disorder-specific associations of subjectively experienced arousal and negative thoughts indicate that the pathophysiological processes of depressive and anxiety disorders are different. Our exploratory analyses of pure depressive disorders versus co-occurring depressive and anxiety disorders showed that postevent negative thoughts were associated only with pure depressive disorders, but not with co-occurring depressive and anxiety disorders. In contrast, subjectively experienced arousal and cortisol reactivity were associated with co-occurring depressive and anxiety disorders. These stress response characteristics we have also found in pure anxiety disorders. Thus, the result maybe indicates a dominance of anxiety disorders in co-occurring depressive and anxiety disorders (Kircanski, LeMoult, Ordaz, & Gotlib, 2016). This is partly in line with Young, Abelson, and Cameron (2004), who found that cortisol was a significant predictor of co-occurring depressive and anxiety disorders, but not of pure depression. Considering the

high comorbidity of depressive and anxiety disorders (Kessler et al., 2005), the results of our exploratory analyses thus indicate the need to consider different pathophysiological processes in children with co-occurring depressive and anxiety disorders. In a recent review, Kircanski, LeMoult, et al. (2016) discuss the very inconsistent findings on psychobiological profiles of co-occurring depression and anxiety in adults as a problem of diagnostic categories. The authors recommend a dimensional approach to capture the unique, shared, and interactive features of depressive and anxiety symptom dimensions.

All children showed significantly higher ratings of negative than positive thoughts 1 hr after the TSST-C. It is interesting that the children with externalizing disorders gave the highest ratings of positive thoughts compared with the children in the other diagnostic groups. In the multinomial regression analyses, positive thoughts turned out to be specifically associated with externalizing disorders and (although less robust) for anxiety disorders as well. Maybe these positive postevent thoughts are an expression of a specific coping strategy employed by children with an externalizing disorder or anxiety disorders, which might be related to a contraphobic attitude or to a narcissistic self-concept (Barry, Frick, & Killian, 2003; Ha, Petersen, & Sharp, 2008). In a study by Barry et al. (2003), the maladaptive dimension of narcissism, which encompasses entitlement (e.g., “I want the world to think that I am something special”), exploitativeness (e.g., “I can make anybody believe anything I want them to”), and exhibitionism (e.g., “I like to be the center of attention”) was associated with lower self-esteem and externalizing problems.

Our study has a number of strengths. It is among the first to investigate endocrinological and subjective stress-related responses in children with different psychiatric disorders and in healthy children. To investigate this, we used an ecologically valid stress induction procedure (Dickerson & Kemeny, 2004). Children were in preadolescence and early adolescence, a time in which they face a large number of changes, as well as growing demands from their environment. To define our diagnostic groups, we used a parental diagnostic interview (K-SADS-PL; Delmo et al., 2001). Out of a larger mixed sample of children (i.e., involving both clinical and community children) with all kinds of comorbid disorders (Quante et al., 2012), we selected diagnostic groups of depressive disorders, anxiety disorders, and externalizing disorders, as well as a healthy control group. This enabled us to investigate disorder-specific stress-related maladaptive response characteristics of children in a challenging period of life.

However, our study also has to be considered in light of some limitations. In the diagnostic interviews, it was usually mothers who were the informants about children’s psychiatric disorders. Unfortunately, we were not able to conduct interviews with the children or other informants (e.g., teachers and psychiatrists). For the assessment of internalizing disorders in particular, children’s self-reports would have been relevant because these disorders, unlike externalizing disorders, are characterized by symptoms that involve the individuals’ thoughts and emotions (Kovacs & Devlin, 1998; Sass et al., 2003).

Moreover, multi-informant reports are known to be more reliable (Kraemer et al., 2003). However, our results represent associations between children's stress responses following a laboratory stressor and their mental health that encompass independent endocrinological and subjective measures that were based on different informants (parent and child) and methods.

The consideration of (gender-specific) developmental influences on endocrinological and subjective stress reactions, as well as on psychiatric disorders, is of great importance. This is also emphasized by findings of Gunnar, Wewerka, et al. (2009) on a more pronounced increase in cortisol stress response in girls around age 13 in contrast to boys. In our analyses, we controlled for age and gender and found significant age effects on depressive and externalizing disorders and significant gender effects on total cortisol release (AUC_g) and negative thoughts. However, due to the small sample sizes of the diagnostic groups, we were not able to analyze the moderating effects of pubertal status, age, or gender. A developmental approach to psychopathology suggests that a longitudinal analysis of the moderating effects of age, pubertal status, and gender is needed to disentangle their developmental influence and thereby fully understand the impact of stress responses on the course of psychiatric disorders between childhood and adulthood.

Moreover, we reported analyses in which we exclusively controlled for child-related factors and refrained from control-

ling for other variables, such as the parental SES (Rajmil et al., 2014), parental psychopathology (LeMoult et al., 2015), the chronicity of disorders (e.g., Boij et al., 2013) or chronic stress (Fries et al., 2005), which have also been reported to influence the development of psychiatric disorders as well as the stress response of children. In addition, future research should look closely at children with co-occurring disorders (e.g., pure depressive disorders vs. comorbid depressive and anxiety disorders), as we assume that these children might show different stress responses.

In our study, we found a significant interaction effect between AUC_g and subjectively experienced arousal in predicting externalizing disorders. This finding should be regarded as preliminary due to low statistical power. To promote a fine-grained understanding of how endocrinological and cognitive-emotional factors work together in the developmental course of different psychiatric disorders, longitudinal analyses of moderating and mediating effects within larger samples are needed.

From a clinical point of view, our results indicate that stress management is an important part of the prevention and therapy of psychiatric disorders in children. Specifically, our findings underpin therapeutic approaches that take into account developmentally relevant stress factors and stress-related thoughts to help children disengage from negative stress-related thoughts and emotions.

References

- Achenbach, T. M. (1991). *Manual for the Child Behavior Checklist/4–18 and 1991 Profile*. Burlington, VT: University of Vermont, Department of Psychiatry.
- Alink, L. R., van IJzendoorn, M. H., Bakermans-Kranenburg, M. J., Mesman, J., Juffer, F., & Koot, H. M. (2008). Cortisol and externalizing behavior in children and adolescents: Mixed meta-analytic evidence for the inverse relation of basal cortisol and cortisol reactivity with externalizing behavior. *Developmental Psychobiology*, *50*, 427–450. doi:10.1002/dev.20300
- Alloy, L. B., Abramson, L. Y., Hogan, M. E., Whitehouse, W. G., Rose, D. T., Robinson, M. S., & Lapkin, J. B. (2000). The Temple-Wisconsin Cognitive Vulnerability to Depression Project: Lifetime history of Axis I psychopathology in individuals at high and low cognitive risk for depression. *Journal of Abnormal Psychology*, *109*, 403–418. doi:10.1037/0021-843X.109.3.403
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington DC: Author.
- Angold, A., Costello, E. J., & Worthman, C. M. (1998). Puberty and depression: The roles of age, pubertal status, and pubertal timing. *Psychological Medicine*, *28*, 51–61.
- Axelson, D., Birmaher, B., Zelazny, J., Kaufman, J., Gill, M. K., & Brent, D. (2009). *K-SADS-PL 2009 working draft*. Pittsburgh, PA: Western Psychiatric Institute and Clinic, Advanced Center for Intervention and Services Research for Early Onset Mood and Anxiety Disorders.
- Ayer, L., Greaves-Lord, K., Althoff, R. R., Hudziak, J. J., Dieleman, G. C., Verhulst, F. C., & van der Ende, J. (2013). Blunted HPA axis response to stress is related to a persistent dysregulation profile in youth. *Biological Psychiatry*, *93*, 343–351. doi:10.1016/j.biopsych.2013.04.002
- Bae, Y. J., Gaudl, A., Jaeger, S., Stadelmann, S., Hiemisch, A., Kiess, W., ... Kratzsch, J. (2015). Immunoassay or LC-MS/MS for the measurement of salivary cortisol in children? *Clinical Chemistry and Laboratory Medicine*. Advance online publication. doi:10.1515/cclm-2015-0412
- Barry, C. T., Frick, P. J., & Killian, A. L. (2003). The relation of narcissism and self-esteem to conduct problems in children: A preliminary investigation. *Journal of Clinical Child and Adolescent Psychology*, *32*, 139–152. doi:10.1207/S15374424JCCP3201_13
- Beck, A. T. (1967). *Depression: Clinical, experimental, and theoretical aspects*. London: Staples Press.
- Booij, S. H., Bouma, E. M. C., de Jonge, P., Ormel, J., & Oldehinkel, A. J. (2013). Chronicity of depressive problems and the cortisol response to psychosocial stress in adolescents: The TRAILS study. *Psychoneuroendocrinology*, *38*, 659–666. doi:10.1016/j.psyneuen.2012.08.004
- Brosschot, J. F., Gerin, W., & Thayer, J. F. (2006). The perseverative cognition hypothesis: A review of worry, prolonged stress-related physiological activation, and health. *Journal of Psychosomatic Research*, *60*, 113–124. doi:10.1016/j.jpsychores.2005.06.074
- Burke, H. M., Davis, M. C., Otte, C., & Mohr, D. C. (2005). Depression and cortisol responses to psychological stress: A meta-analysis. *Psychoneuroendocrinology*, *30*, 846–856. doi:10.1016/j.psyneuen.2005.02.010
- Buske-Kirschbaum, A., Jobst, S., Wustmans, A., Kirschbaum, C., Rauh, W., & Hellhammer, D. H. (1997). Attenuated free cortisol response to psychosocial stress in children with atopic dermatitis. *Psychosomatic Medicine*, *59*, 419–426. doi:10.1097/00006842-199707000-00012
- Chida, Y., & Hamer, M. (2008). Chronic psychosocial factors and acute physiological responses to laboratory-induced stress in healthy populations: A quantitative review of 30 years of investigations. *Psychological Bulletin*, *134*, 829–885. doi:10.1037/a0013342
- Chrousos, G. P. (2009). Stress and disorders of the stress system. *Nature Reviews Endocrinology*, *5*, 374–381. doi:10.1038/nrendo.2009.106
- Colich, N. L., Kircanski, K., Foland-Ross, L. C., & Gotlib, I. H. (2015). HPA-axis reactivity interacts with stage of pubertal development to predict the onset of depression. *Psychoneuroendocrinology*, *55*, 94–101. doi:10.1016/j.psyneuen.2015.02.004
- Compas, B. E., Connor-Smith, J. K., Saltzman, H., Harding Thomsen, A., & Wadsworth, M. E. (2001). Coping with stress during childhood and adolescence: Problems, progress, and potential in theory and research. *Psychological Bulletin*, *127*, 87–127. doi:10.1037//0033-2909.127.1.87
- Costello, E. J., Copeland, W., & Angold, A. (2016). The Great Smoky Mountains Study: Developmental epidemiology in the southeastern United States. *Social Psychiatry and Psychiatric Epidemiology*, *51*, 639–646. doi:10.1007/s00127-015-1168-1

- Crick, N. R., & Zahn-Waxler, C. (2003). The development of psychopathology in females and males: Current progress and future challenges. *Development and Psychopathology*, *15*, 719–742.
- de Kloet, E. R., Joëls, M., & Holsboer, F. (2005). Stress and the brain: From adaptation to disease. *Nature Reviews Neuroscience*, *6*, 463–475. doi:10.1038/nrn1683
- Delmo, C., Weiffenbach, O., Gabriel, M., Stadler, C., & Poustka, F. (2001). *Kiddie-Sads—Present and Lifetime Version (K-SADS-PL) Diagnostisches Interview* (5. Auflage). Frankfurt am Main: Klinik für Psychiatrie und Psychotherapie des Kindes- und Jugendalters.
- de Raedt, R., & Koster, E. H. W. (2010). Understanding vulnerability for depression from a cognitive neuroscience perspective: A reappraisal of attentional factors and a new conceptual framework. *Cognitive, Affective, & Behavioral Neuroscience*, *10*, 50–70. doi:10.3758/CABN.10.1.50
- de Rooij, S. R. (2013). Blunted cardiovascular and cortisol reactivity to acute psychological stress: A summary of results from the Dutch Famine Birth Cohort Study. *International Journal of Psychophysiology*, *90*, 21–27. doi:10.1016/j.ijpsycho.2012.09.011
- Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, *130*, 355–391. doi:10.1037/0033-2909.130.3.355
- Dieleman, G. C., van der Ende, J., Verhulst, F. C., & Huizink, A. C. (2010). Perceived and physiological arousal during a stress task: Can they differentiate between anxiety and depression? *Psychoneuroendocrinology*, *35*, 1223–1234. doi:10.1016/j.psyneuen.2010.02.012
- Edwards, S. L., Rapee, R. M., & Franklin, J. (2003). Postevent rumination and recall bias for a social performance event in high and low socially anxious individuals. *Cognitive Therapy and Research*, *27*, 603–617. doi:10.1023/A:1026395526858
- Fairchild, G., van Goozen, S. H. M., Stollery, S. J., Brown, J., Gardiner, J., Herbert, J., & Goodyer, I. M. (2008). Cortisol diurnal rhythm and stress reactivity in male adolescents with early-onset or adolescence-onset conduct disorder. *Biological Psychiatry*, *64*, 599–606. doi:10.1016/j.biopsych.2008.05.022
- Fries, E., Hesse, J., Hellhammer, J., & Hellhammer, D. H. (2005). A new view on hypocortisolism. *Psychoneuroendocrinology*, *30*, 1010–1016. doi:10.1016/j.psyneuen.2005.04.006
- Goodman, R. (1997). The Strengths and Difficulties Questionnaire: A research note. *Journal of Child Psychology and Psychiatry*, *38*, 581–586. doi:10.1111/j.1469-7610.1997.tb01545.x
- Gotlib, I. H., & Joormann, J. (2010). Cognition and depression: Current status and future directions. *Annual Review of Clinical Psychology*, *6*, 285–312. doi:10.1146/annurev.clinpsy.121208.131305
- Gunnar, M. R., Brodersen, L., Nachmias, M., Buss, K., & Rigatuso, J. (1996). Stress reactivity and attachment security. *Developmental Psychobiology*, *29*, 191–204. doi:10.1002/(SICI)1098-2302(199604)29:3<191:AID-DEV1>3.0.CO;2-M
- Gunnar, M. R., & Quevedo, K. (2007). The neurobiology of stress and development. *Annual Review of Psychology*, *58*, 145–173. doi:10.1146/annurev.psych.58.110405.085605
- Gunnar, M. R., Talge, N. M., & Herrera, A. (2009). Stressor paradigms in developmental studies: What does and does not work to produce mean increases in salivary cortisol. *Psychoneuroendocrinology*, *34*, 953–967. doi:10.1016/j.psyneuen.2009.02.010
- Gunnar, M. R., & Vazquez, D. M. (2001). Low cortisol and a flattening of expected daytime rhythm: Potential indices of risk in human development. *Development and Psychopathology*, *13*, 515–538. doi:10.1017/S0954579401003066
- Gunnar, M. R., Wewerka, S., Frenn, K., Long, J. D., & Griggs, C. (2009). Developmental changes in hypothalamus–pituitary–adrenal activity over the transition to adolescence: Normative changes and associations with puberty. *Development and Psychopathology*, *21*, 69–85. doi:10.1017/S0954579409000054
- Ha, C., Petersen, N., & Sharp, C. (2008). Narcissism, self-esteem, and conduct problems: Evidence from a British community sample of 7–11 year olds. *European Child and Adolescent Psychiatry*, *17*, 406–413. doi:10.1007/s00787-008-0682-z
- Hankin, B. L., Badanes, L. S., Abela, J. R. Z., & Watamura, S. E. (2010). Hypothalamic-pituitary-adrenal axis dysregulation in dysphoric children and adolescents: Cortisol reactivity to psychosocial stress from preschool through middle adolescence. *Biological Psychiatry*, *68*, 484–490. doi:10.1016/j.biopsych.2010.04.004
- Harter, S., & Whitesell, N. R. (2003). Beyond the debate: Why some adolescents report stable self-worth over time and situation, whereas others report changes in self-worth. *Journal of Personality*, *71*, 1027–1058.
- Hartman, C. A., Hermanns, V. W., de Jong, P. J., & Ormel, J. (2013). Self- or parent report of (co-occurring) internalizing and externalizing problems, and basal or reactivity measures of HPA-axis functioning: A systematic evaluation of the internalizing-hyperresponsivity versus externalizing-hyporesponsivity HPA-axis hypothesis. *Biological Psychology*, *94*, 175–184. doi:10.1016/j.biopsycho.2013.05.009
- Hastings, P. D., Shirtcliff, E. A., Klimes-Dougan, B., Allison, A. L., Derose, L., Kendziora, K. T., . . . Zahn-Waxler, C. (2011). Allostatic and the development of internalizing and externalizing problems: Changing relations with physiological systems across adolescence. *Development and Psychopathology*, *23*, 1149–1165. doi:10.1017/S0954579411000538
- Jaffee, S. R., McFarquhar, T., Stevens, S., Ouellet-Morin, I., Melhuish, E., & Belsky, J. (2015). Interactive effects of early and recent exposure to stressful contexts on cortisol reactivity in middle childhood. *Journal of Child Psychology and Psychiatry*, *56*, 138–146. doi:10.1111/jcpp.12287
- Johnson, D. P., & Whisman, M. A. (2013). Gender differences in rumination: A meta-analysis. *Personality and Individual Differences*, *55*, 367–374. doi:10.1016/j.paid.2013.03.019
- Jose, P. E., & Brown, I. (2008). When does the gender difference in rumination begin? Gender and age differences in the use of rumination by adolescents. *Journal of Youth and Adolescence*, *37*, 180–192. doi:10.1007/s10964-006-9166-y
- Juster, R., McEwen, B. S., & Lupien, S. J. (2010). Allostatic load biomarkers of chronic stress and impact on health and cognition. *Neuroscience & Biobehavioral Reviews*, *35*, 2–16. doi:10.1016/j.neubiorev.2009.10.002
- Kaufman, J., Birmaher, B., Brent, B., Rao, U., & Ryan, N. D. (1996). *Kiddie-Sads—Present and Lifetime Version (K-SADS-PL)*. Pittsburgh: University of Pittsburgh School of Medicine, Department of Psychiatry.
- Keenan, K., Hipwell, A., Babinski, D., Bortner, J., Henneberger, A., Hinze, A., . . . Sapotichne, B. (2013). Examining the developmental interface of cortisol and depression symptoms in young adolescent girls. *Psychoneuroendocrinology*, *38*, 2291–2299. doi:10.1016/j.psyneuen.2013.04.017
- Kessler, R. C., Chiu, W. T., Demler, O., Merikangas, K. R., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the national comorbidity survey replication. *Archives of General Psychiatry*, *62*, 709.
- Kircanski, K., LeMoult, J., Ordaz, S., & Gotlib, I. H. (2016). Investigating the nature of co-occurring depression and anxiety: Comparing diagnostic and dimensional research approaches. *Journal of Affective Disorders*. Advance online publication. doi:10.1016/j.jad.2016.08.006
- Kircanski, K., Waugh, C. E., Camacho, M. C., & Gotlib, I. H. (2016). Aberrant parasympathetic stress reactivity in pure and co-occurring major depressive disorder and generalized anxiety disorder. *Journal of Psychopathology and Behavioral Assessment*, *38*, 5–19. doi:10.1007/s10862-015-9493-y
- Klumbies, E., Brauer, D., Hoyer, J., & Kirschbaum, C. (2014). The reaction to social stress in social phobia: Discordance between physiological and subjective parameters. *PLOS ONE*, *9*, e105670. doi:10.1371/journal.pone.0105670
- Kovacs, M., & Devlin, B. (1998). Internalizing disorders in childhood. *Journal of Child Psychology and Psychiatry*, *39*, 47–63. doi:10.1111/1469-7610.00303
- Kraemer, H. C., & Blasey, C. M. (2004). Centring in regression analyses: A strategy to prevent errors in statistical inference. *International Journal of Methods in Psychiatric Research*, *13*, 141–151.
- Kraemer, H. C., Measelle, J. R., Ablow, J. C., Essex, M. J., Boyce, W. T., & Kupfer, D. J. (2003). A new approach to integrating data from multiple informants in psychiatric assessment and research: Mixing and matching contexts and perspectives. *American Journal of Psychiatry*, *160*, 1566–1577. doi:10.1176/appi.ajp.160.9.1566
- Krämer, M., Seefeldt, W. L., Heinrichs, N., Tuschen-Caffier, B., Schmitz, J., Wolf, O. T., & Blechert, J. (2012). Subjective, autonomic, and endocrine reactivity during social stress in children with social phobia. *Journal of Abnormal Child Psychology*, *40*, 95–104. doi:10.1007/s10802-011-9548-9
- Kudielka, B. M., Buske-Kirschbaum, A., Hellhammer, D. H., & Kirschbaum, C. (2004). Differential heart rate reactivity and recovery after psychosocial stress (TSST) in healthy children, younger adults, and elderly adults: The impact of age and gender. *International Journal of Behavioral Medicine*, *11*, 116–121. doi:10.1207/s15327558ijbm1102_8
- Kudielka, B. M., Hellhammer, D. H., & Wüst, S. (2009). Why do we respond so differently? Reviewing determinants of human salivary cortisol responses to challenge. *Psychoneuroendocrinology*, *34*, 2–18. doi:10.1016/j.psyneuen.2008.10.004

- Lange, M., Kamtsiuris, P., Lange, C., Schaffrath, R. A., Stolzenberg, H., & Lampert, T. (2007). Messung soziodemographischer Merkmale im Kinder- und Jugendgesundheitsurvey (KiGGS) und ihre Bedeutung am Beispiel der Einschätzung des allgemeinen Gesundheitszustands [Sociodemographic characteristics in the German Health Interview and Examination Survey for Children and Adolescents (KiGGS)—Operationalisation and public health significance, taking as an example the assessment of general state of health]. *Bundesgesundheitsblatt, Gesundheitsforschung, Gesundheitsschutz*, 50, 578–589. doi:10.1007/s00103-007-0219-5
- LeMoult, J., Ordaz, S. J., Kircanski, K., Singh, M. K., & Gotlib, I. H. (2015). Predicting first onset of depression in young girls: Interaction of diurnal cortisol and negative life events. *Journal of Abnormal Psychology*, 124, 850–859.
- Lopez-Duran, N. L., Kovacs, M., & George, C. J. (2009). Hypothalamic-pituitary-adrenal axis dysregulation in depressed children and adolescents: A meta-analysis. *Psychoneuroendocrinology*, 34, 1272–1283. doi:10.1016/j.psyneuen.2009.03.016
- Maldonado, E. F., Trianes, V., Cortés, A., Moreno, E., & Escobar, M. (2009). Salivary cortisol response to a psychosocial stressor on children diagnosed with attention-deficit/hyperactivity disorder: Differences between diagnostic subtypes. *Spanish Journal of Psychology*, 12, 707–714. doi:10.1017/S1138741600002079
- Mathews, A., & MacLeod, C. (2005). Cognitive vulnerability to emotional disorders. *Annual Review of Clinical Psychology*, 1, 167–195. doi:10.1146/annurev.clinpsy.1.102803.143916
- McEwen, B. S. (1998). Stress, adaptation, and disease: Allostasis and allostatic load. *Annals of the New York Academy of Sciences*, 840, 33–44. doi:10.1111/j.1749-6632.1998.tb09546.x
- Morris, N. M., & Udry, J. R. (1980). Validation of a self-administered instrument to assess stage of adolescent development. *Journal of Youth and Adolescence*, 9, 271–280. doi:10.1007/BF02088471
- Muris, P., Meesters, C., Merckelbach, H., Sermon, A., & Zwakhalen, S. (1998). Worry in normal children. *Journal of the American Academy of Child & Adolescent Psychiatry*, 37, 703–710. doi:10.1097/00004583-199807000-00009
- Petrowski, K., Wintermann, G., Schaarschmidt, M., Bornstein, S. R., & Kirschbaum, C. (2013). Blunted salivary and plasma cortisol response in patients with panic disorder under psychosocial stress. *International Journal of Psychophysiology*, 88, 35–39. doi:10.1016/j.ijpsycho.2013.01.002
- Pruessner, J. C., Kirschbaum, C., Meinlschmid, G., & Hellhammer, D. H. (2003). Two formulas for computation of the area under the curve represent measures of total hormone concentration versus time-dependent change. *Psychoneuroendocrinology*, 28, 916–931. doi:10.1016/S0306-4530(02)00108-7
- Quante, M., Hesse, M., Döhrner, M., Fuchs, M., Hirsch, C., Sergeyev, E., . . . Kiess, W. (2012). The LIFE child study: A life course approach to disease and health. *BMC Public Health*, 12. doi:10.1186/1471-2458-12-1021
- Quas, J. A., Hong, M., Alkon, A., & Boyce, W. T. (2000). Dissociations between psychobiologic reactivity and emotional expression in children. *Developmental Psychobiology*, 37, 153–175.
- Raine, A. (2002). Biosocial studies of antisocial and violent behavior in children and adults: A review. *Journal of Abnormal Child Psychology*, 30, 311–326. doi:10.1023/A:1015754122318
- Rajmil, L., Herdman, M., Ravens-Sieberer, U., Erhart, M., Alonso, J., & European KIDSCREEN Group. (2014). Socioeconomic inequalities in mental health and health-related quality of life (HRQOL) in children and adolescents from 11 European countries. *International Journal of Public Health*, 59, 95–105. doi:10.1007/s00038-013-0479-9
- Randazzo, W. T., Dockray, S., & Susman, E. J. (2008). The stress response in adolescents with inattentive type ADHD symptoms. *Child Psychiatry and Human Development*, 39, 27–38. doi:10.1007/s10578-007-0068-3
- Rao, U., Hammen, C., Ortiz, L. R., Chen, L., & Poland, R. E. (2008). Effects of early and recent adverse experiences on adrenal response to psychosocial stress in depressed adolescents. *Biological Psychiatry*, 64, 521–526. doi:10.1016/j.biopsych.2008.05.012
- Rapkin, A. J., Tsao, J. C. I., Turk, N., Anderson, M., & Zeltzer, L. K. (2006). Relationships among self-rated Tanner staging, hormones, and psychosocial factors in healthy female adolescents. *Journal of Pediatric and Adolescent Gynecology*, 19, 181–187. doi:10.1016/j.jpag.2006.02.004
- Reardon, L. E., Leen-Feldner, E. W., & Hayward, C. (2009). A critical review of the empirical literature on the relation between anxiety and puberty. *Clinical Psychology Review*, 29, 1–23. doi:10.1016/j.cpr.2008.09.005
- Robinson, M. S., & Alloy, L. B. (2003). Negative cognitive styles and stress-reactive rumination interact to predict depression: A prospective study. *Cognitive Therapy and Research*, 27, 275–291. doi:10.1023/A:1023914416469
- Rood, L., Roelofs, J., Bögels, S. M., & Alloy, L. B. (2010). Dimensions of negative thinking and the relations with symptoms of depression and anxiety in children and adolescents. *Cognitive Therapy and Research*, 34, 333–342. doi:10.1007/s10608-009-9261-y
- Rood, L., Roelofs, J., Bögels, S. M., & Meesters, C. (2012). Stress-reactive rumination, negative cognitive style, and stressors in relationship to depressive symptoms in non-clinical youth. *Journal of Youth and Adolescence*, 41, 414–425. doi:10.1007/s10964-011-9657-3
- Rudolph, K. D., Troop-Gordon, W., & Granger, D. A. (2011). Individual differences in biological stress responses moderate the contribution of early peer victimization to subsequent depressive symptoms. *Psychopharmacology*, 214, 209–219. doi:10.1007/s00213-010-1879-7
- Sass, H., Wittchen, H. U., Zaudig, M., & Houben, I. (2003). *Diagnostisches und statistisches manual psychischer störungen textrevision (DSM-IV-TR)*. Göttingen: Hogrefe.
- Schmitz, J., Krämer, M., Blechert, J., & Tuschen-Caffier, B. (2010). Post-event processing in children with social phobia. *Journal of Abnormal Child Psychology*, 38, 911–919. doi:10.1007/s10802-010-9421-2
- Schwartz, A. R., Gerin, W., Davidson, K. W., Pickering, T. G., Brosschot, J. F., Thayer, J. F., . . . Linden, W. (2003). Toward a causal model of cardiovascular responses to stress and the development of cardiovascular disease. *Psychosomatic Medicine*, 65, 22–35. doi:10.1097/01.PSY.0000046075.79922.61
- Stadelmann, S., Grunewald, M., Gibbels, C., Jaeger, S., Matuschek, T., Weis, S., . . . Döhrner, M. (2017). Self-esteem of 8–14-year-old children with psychiatric disorders: Disorder- and gender-specific effects. *Child Psychiatry and Human Development*, 48, 40–52. doi:10.1007/s10578-016-0651-6
- Stetler, C., & Miller, G. E. (2011). Depression and hypothalamic-pituitary-adrenal activation: A quantitative summary of four decades of research. *Psychosomatic Medicine*, 73, 114–126. doi:10.1097/PSY.0b013e31820ad12b
- Stewart, J. G., Mazurka, R., Bond, L., Wynne-Edwards, K. E., & Harkness, K. L. (2013). Rumination and impaired cortisol recovery following a social stressor in adolescent depression. *Journal of Abnormal Child Psychology*, 41, 1015–1026. doi:10.1007/s10802-013-9740-1
- Stroud, L. R., Foster, E., Papandonatos, G. D., Handwerker, K., Granger, D. A., Kivlighan, K. T., . . . Niaura, R. (2009). Stress response and the adolescent transition: Performance versus peer rejection stressors. *Development and Psychopathology*, 21, 47–68. doi:10.1017/S0954579409000042
- Susman, E. J. (2006). Psychobiology of persistent antisocial behavior: Stress, early vulnerabilities and the attenuation hypothesis. *Neuroscience & Biobehavioral Reviews*, 30, 376–389. doi:10.1016/j.neubiorev.2005.08.002
- van Goozen, S. H., Matthys, W., Cohen-Kettenis, P. T., Buitelaar, J. K., & van Engeland, H. (2000). Hypothalamic-pituitary-adrenal axis and autonomic nervous system activity in disruptive children and matched controls. *Journal of the American Academy of Child & Adolescent Psychiatry*, 39, 1438–1445. doi:10.1097/00004583-200011000-00019
- van Goozen, S. H. M., Matthys, W., Cohen-Kettenis, P. T., Gispen-de Wied, C., Wiegant, V. M., & van Engeland, H. (1998). Salivary and cardiovascular activity during stress in oppositional-defiant disorder boys and normal controls. *Biological Psychiatry*, 43, 531–539. doi:10.1016/S0006-3223(97)00253-9
- van West, D., Claes, S., Sulon, J., & Deboutte, D. (2008). Hypothalamic-pituitary-adrenal reactivity in prepubertal children with social phobia. *Journal of Affective Disorders*, 111, 281–290. doi:10.1016/j.jad.2008.03.006
- von Klitzing, K., White, L. O., Otto, Y., Fuchs, S., Egger, H. L., & Klein, A. M. (2014). Depressive comorbidity in preschool anxiety disorder. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, 55, 1107–1116. doi:10.1111/jcpp.12222
- Watkins, E. R. (2008). Constructive and unconstructive repetitive thought. *Psychological Bulletin*, 134, 163–206. doi:10.1037/0033-2909.134.2.163
- Weiss, R. H. (2006). *Sprachfreier Grundintelligenztest Skala 2—Revision (CFT-20-R)*. Göttingen: Hogrefe.
- Young, E. A., Abelson, J. L., & Cameron, O. G. (2004). Effect of comorbid anxiety disorders on the hypothalamic-pituitary-adrenal axis response to a social stressor in major depression. *Biological Psychiatry*, 56, 113–120. doi:10.1016/j.biopsych.2004.03.017
- Zoccola, P. M., & Dickerson, S. S. (2012). Assessing the relationship between rumination and cortisol: A review. *Journal of Psychosomatic Research*, 73, 1–9. doi:10.1016/j.jpsychores.2012.03.007