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Low basal salivary cortisol is associated with teacher-reported symptoms of conduct disorder

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Abstract

Cortisol has been implicated in psychobiological explanations of antisocial behavior. This study measured basal salivary cortisol in a sample of 25 children (age range 6 to 12 years) selected to vary in levels of antisocial behavior. Regression analyses were used to predict cortisol concentrations from parent- and teacher-reported symptoms. Parent-reported symptoms did not predict basal cortisol. Teacher-reported conduct disorder (CD) symptoms explained 38% of the variance in the cortisol concentrations, with high symptom severity associated with low cortisol. When a distinction was made between aggressive and non-aggressive CD symptoms, aggressive CD symptoms were more clearly related to low cortisol than non-aggressive CD symptoms. In contrast to previous research, no evidence was found for a mediating role of anxiety symptoms in the relationship between CD and cortisol. The results support biologically based models of antisocial behavior in children that involve reduced autonomic activity.

Keywords: Autonomic activity; Antisocial behavior; Aggression; Oppositional defiant disorder; Anxiety

1. Introduction

Antisocial behavior in children is of great concern because of its devastating impact on the child, the child's social environment, and society, as well as the high risk for antisocial personality disorder and criminality (Loeber et al., 2000). Current diagnostic classification systems (American Psychiatric Association, 1994) distinguish between two forms of antisocial behavior: oppositional defiant disorder (ODD) and conduct disorder (CD). The essential features of ODD are a recurrent pattern of negativistic, defiant, disobedient, and hostile behavior toward authority figures, whereas the essential features of CD are a repetitive and persistent pattern of behavior in which the basic rights of others and major age-appropriate societal norms or rules are violated (American Psychiatric Association, 1994).

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One of the most provocative neuroendocrinologic findings reported in ODD and CD is abnormal basal cortisol concentrations (Vanyukov et al., 1993; Scerbo and Kolko, 1994; Moss et al., 1995; Van Goozen et al., 1998; McBurnett et al., 2000; Pajer et al., 2001; Karyawasam et al., 2003; Shoal et al., 2003; Van de Wiel et al., 2004). Cortisol is one of the main outputs of the stress system. Stress activates the rapidly responding sympathetic division of the autonomic nervous system, and the slower but longer-acting hypothalamic–pituitary–adrenal axis (HPA axis). In the HPA axis, stress triggers the release from the hypothalamus of corticotropin-releasing hormone (CRH) and arginine–vasopressin (AVP), which both stimulate secretion of adrenocorticotropic hormone (ACTH) from the anterior pituitary. ACTH is released into the general circulation and acts on the adrenal cortex, stimulating it to produce glucocorticoid hormones, mainly cortisol (Stratakis and Chrousos, 1995; Pliszka, 1999; Miller and O'Callaghan, 2002; Tsigos and Chrousos, 2002). Cortisol has been implicated in psychobiological explanations of antisocial behavior from two partially overlapping perspectives.

First, cortisol may be regarded as a peripheral indicator of autonomic activity. Several studies have found evidence for reduced autonomic activity in children, adolescents and adults with antisocial behavior, including lowered heart rate and skin conductance (Pliszka, 1999). Activity in the sympathetic branch of autonomic nervous system goes hand in hand with the release of cortisol from the HPA axis. Thus, low basal cortisol concentrations may reflect reduced autonomic activity.

Second, the stress system has an important role in learning and memory. Antisocial behavior has been related to a failure to learn from aversive consequences of behavior. It appears that antisocial individuals are less afraid of the aversive consequences of their actions than others are. Normal autonomic reactivity is required for normal conditioned emotional responding to signals of threat of punishment and passive avoidance learning to occur (McBurnett et al., 1996). Furthermore, activation of the stress system triggers the amygdala, the principal brain locus for fear-related behaviors, and facilitates the long-term storage of aversively charged emotional memories in sites such as the hippocampus and striatum (Tsigos and Chrousos, 2002). Thus, reduced activity in the stress system may impede the process of learning from aversively charged stimuli, which may contribute to the development and maintenance of antisocial behavior (McBurnett et al., 1996; King et al., 1998). Low basal cortisol concentrations may thus be associated with impaired learning and memory.

Several studies provide evidence for low basal cortisol in children characterised by antisocial behavior. Correlational studies have shown that measures of antisocial behavior including symptom counts of ODD and CD are associated with low cortisol (Vanyukov et al., 1993; Scerbo and Kolko, 1994; Moss et al., 1995; McBurnett et al., 2000; Van de Wiel et al., 2004). Others have found lower levels of cortisol in children with ODD and CD as than in normal developing peers (Van Goozen et al., 1998; Karyawasam et al., 2003). While most of these studies were restricted to boys, low cortisol concentrations have also been reported in girls with CD (Pajer et al., 2001). Interestingly, a recent longitudinal study by Shoal et al. (2003) has demonstrated that low resting cortisol levels may actually predict aggression 5 years later. However, other studies have found no evidence for the relationship between low cortisol concentrations and antisocial behavior either measured dimensionally or categorically (Dabbs et al., 1991; Stoff et al., 1992; Scerbo and Kolko, 1994; Schulz et al., 1997; Jansen et al., 1999; Van Goozen et al., 2000; Snoek et al., 2002). All in all, evidence for low basal cortisol in children with antisocial behavior is inconsistent.

To elucidate further the relationship between antisocial behavior and low basal cortisol, research has distinguished between aggressive (overt confrontational, such as fighting) and non-aggressive (covert non-confrontational, such as theft) CD symptoms (Berkowitz, 1993, 1994; Frick et al., 1993; Fergusson et al., 1994). Low cortisol concentrations seem to be associated with aggressive CD symptoms in particular and less clearly with non-aggressive CD symptoms (McBurnett et al., 1996, 1997, 2000; Pajer et al., 2001), although not all studies found support for this (Schulz et al., 1997).

Furthermore, studies of basal cortisol in children with antisocial behavior have focused on the impact of comorbid anxiety. In contrast to antisocial behavior, anxiety has been associated with heightened cortisol

concentrations (Kagan et al., 1987, 1988; Scerbo and Kolko, 1994). According to the position that anxiety is related to heightened cortisol levels, it would seem plausible that low anxiety is associated with low resting cortisol and antisocial behavior. This leads to the hypothesis that the relationship between CD and low basal cortisol is mediated by anxiety. Consistent with this reasoning, McBurnett et al. (1991) found that children with CD and comorbid anxiety disorder had higher levels of cortisol than children with CD without comorbid anxiety disorder. These findings suggest that CD is predictive of low cortisol only in the absence of anxiety. More recent studies, however, have failed to replicate this finding (Schulz et al., 1997; Van Goozen et al., 1998).

The purpose of the present study was to elucidate further the relationship between basal cortisol and antisocial behavior in a sample of 25 children that covers a broad range of antisocial behavior. In this cross-sectional study, regression analyses were used to predict cortisol concentrations from parent- and teacher-reported symptoms separately. First, CD symptoms were used to predict cortisol to test the hypothesis that CD is associated with low cortisol concentrations. Second, the hypothesis was tested that the relationship between CD and low cortisol holds in particular for aggressive as opposed to non-aggressive CD symptoms. Finally, exploratory analyses focused on whether anxiety symptoms mediate the relationship between CD and cortisol.

The present study improves upon previous research by investigating the cortisol relationships separately for parent- and teacher-reported symptoms. A substantial body of evidence suggests that parents and teachers report on different aspects of behavior (Achenbach et al., 1987; Loeber et al., 1991; Hart et al., 1994; Offord et al., 1996) and show different associations with psychobiological measures (Riccio et al., 1994; Oosterlaan et al., 2005).

2. Methods

2.1. Participants

Subjects were 25 children (4 girls) in the age range 6 to 12 years (M=9.2, S.D.=1.7). On the basis of research diagnostic criteria, 18 children were diagnosed with ODD/CD. No distinction was made between ODD and CD, and the two disorders were treated as lying on a dimension that varies in terms of the severity of antisocial behavior. ODD is generally considered a milder form of CD and is frequently found to be a developmental antecedent of CD (American Psychiatric Association, 1994). Both disorders have similar risk factors, may evince a common genetic underpinning, and respond favorably to the same forms of treatment (Loeber et al., 2000; Burke et al., 2002). To create a sample of children that covers a broad range of antisocial behavior, the sample was extended by adding seven children that did not meet research diagnostic criteria for ODD/CD.

Children with ODD/CD were recruited from special educational facilities, which are aimed at the education of children with extreme behavioral problems. Inclusion in this study was based on parent and teacher ratings on the Disruptive Behavior Disorder Rating Scale (DBDRS, Pelham et al., 1992; Dutch translation: Oosterlaan et al., 2000). The DBDRS is a symptom severity rating scale and contains four scales, with two scales tapping into symptoms of ODD and CD as listed in DSM-IV (American Psychiatric Association, 1994) and two scales tapping into the DSM-IV symptoms of attention deficit/hyperactivity disorder (AD/HD). Adequate psychometric properties have been reported for this instrument (Pelham et al., 1992; Oosterlaan et al., 2000). To be included in this study, children were required to meet a research diagnosis of ODD/CD as manifested in a rating above the 95th percentile clinical cut-off on the ODD and/or CD scale by both parent and teacher. Thus, 18 children were diagnosed as ODD/CD of whom 13 had a comorbid diagnosis of AD/HD. Parents of all but two of the children were also interviewed using the Diagnostic Interview Schedule for Children for DSM-IV (PDISC-IV, National Institute of Mental Health [NIMH], Shaffer et al., 2000; Dutch translation: Ferdinand et al., 1998). The DISC-IV is a structured diagnostic interview that generates DSM-IV diagnoses. In this study, the Disruptive Behavior Disorder section was used, which covers ODD, CD, and AD/HD. Adequate reliability and validity have been reported for earlier versions of the PDISC-IV (Schwab-Stone et al., 1996). For 12 children, a diagnosis of ODD or CD was established, of whom 10 also met criteria for AD/HD. Three

children were diagnosed as only AD/HD, and one child did not meet criteria for one of the disruptive behavior disorders on the PDISC-IV.

The seven children not meeting research diagnostic criteria for ODD/CD were recruited from regular elementary schools. None of these seven children had ever received a clinical diagnosis according to their parents (i.e., a behavioral problem or a learning disability), and none of their scores on the parent and teacher DBDRS exceeded the 75th percentile.

Children were excluded from the study if (1) they used medication that could not be discontinued at least 48 h before cortisol sampling and (2) their estimated full-scale IQ on a four-subtest short form of the Revised Wechsler Intelligence Scale for Children (WISC-R) was below 80 (Groth-Marnat, 1997). The average IQ was 99.0 (S.D.=16.4, range 80–151).

The University Ethical Committee approved the study, and informed consent was obtained from parents.

2.2. Cortisol

Cortisol was measured in saliva. Saliva collection is a stress-free approach and avoids potential confounds produced by a stress response due to venipuncture (Moss et al., 1995; Dawes et al., 1999). The concentration of cortisol in saliva is closely correlated with the level of free biologically active cortisol in blood that can engage brain receptors (Kirschbaum and Hellhammer, 1994).

A single saliva sample was collected with Salivettes for each child during a neuropsychological testing session. Subjects were provided cotton wool plugs pledged in citric acid to chew on to stimulate an adequate flow of saliva. Cotton plugs with saliva were then inserted into the accompanying plastic vials, centrifuged, and stored at −20 °C until analysis.

After thawing, salivary cortisol concentrations were determined by competitive radioimmunoassay (Orion Diagnostica SPECTRIA RIA test, Espoo, Finland). The sensitivity of this method, defined as the concentration equivalent to twice the standard deviation of the zero-binding value, is 0.8 nmol/l. The inter-assay (between-run) coefficient of variation was 19% at 2 nmol/l and 7% at 8 nmol/l. The intra-assay (within-run) coefficient of variation as determined from duplicate measurements was 5.8% in the range of 1.5–5 nmol/l (n=39), 5.9% in the range of 5–10 nmol/l (n=20), and 4.2% in the range above 110 nmol/l (n=25). All assays were run in duplicate and were performed in batches of about 60 samples each. Cortisol concentrations were transformed to natural logarithm scale to render the distributions more symmetrical.

The mean cortisol level for the entire group was 6.6 nmol/l (S.D.=4.6, range 2.4–22.0). Cortisol levels were similar in boys (M=6.5) and girls (M=7.1), and it was decided to include girls in subsequent analyses. Cortisol concentrations were neither correlated with age [r (N=25)=−0.05, ns] nor IQ [r (N=25)=0.33, ns]. The time of saliva collection was recorded to investigate diurnal variation in cortisol at the group level. Samples were collected between 10.00 h and 14.25 h. To investigate whether time of collection was related to cortisol in the present study and in this limited time frame, a regression analysis was conducted in which time of collection (in min) and time of collection squared were used to predict cortisol. These two predictors investigated the possibility of a linear and curvilinear relationship between time of collection and cortisol concentrations. The relationship between the two predictors and cortisol was not significant, R^2=0.14, F(2, 21)=1.76, P=0.196. Consistent with other studies (McBurnett et al., 1991; Kirschbaum and Hellhammer, 1994; McBurnett et al., 1997), time of collection was not a confound in the present study.

2.3. Psychopathology measures

Parent and teacher ratings of behavior were obtained using the DSM-IV screener (Hartman et al., 2001). This questionnaire was developed to assess syndromes of childhood psychopathology in terms of the DSM-IV. The questionnaire contains 181 items. Items were rated on a four-point scale ranging from zero (does not apply) to three (applies very much). Higher scores indicate greater impairment. For the current study, two scales were used: CD (36 items) and anxiety (40 items). For the calculation of scale scores, item scores were given equal weights.

According to Berkowitz (1993, 1994), aggression is any form of behavior directed toward the goal of harming or injuring another living being who is motivated to avoid such treatment. Aggressive CD symptoms thus refer to overt confrontational as

opposed to covert non-confrontational behavior. To test the hypothesis that the relationship between CD and low basal cortisol holds in particular for aggressive as opposed to non-aggressive CD symptoms, items reflecting aggressive (overt confrontational) and non-aggressive (covert non-confrontational) CD symptoms were selected from the CD scale. Items measuring aggressive CD symptoms are the following: tries to make others afraid by threatening (29); intentionally tries to give others bodily pain (30); often initiates physical fights (31); has deliberately injured someone with a dangerous object (32); baits animals (33); has stolen while threatening a victim (34); has assaulted someone (36); forces others to do things that they do not want to do (40); enjoys making others look a fool (41); bullies (43a); threatens with a weapon (43b); speaks ill of others (43c); purposively leaves someone out to hurt that person (43d); has set fire to cause damage (53); and has committed vandalism (56). Non-aggressive CD symptoms include the following: lies in order to get things from other people (44); lies to avoid obligations (45); exploits others for own sake (48); steals (51); has broken into a car, home, or building (52); stays away from home at night despite the wishes of parents/caregivers (58); has run away from home for more than 1 day (59); is absent from school without the knowledge of his/her parents/caregivers (60); and hangs out with others that easily get themselves into trouble (62).

2.4. Statistical analyses

To test the hypothesis that CD is associated with low cortisol concentrations, teacher- and parent-reported CD symptoms were used to predict cortisol in two separate regression analyses. Multiple hierarchical regression analyses were used to test the hypothesis that the relationship between CD and low cortisol holds in particular for aggressive as opposed to non-aggressive CD symptoms. In these analyses, aggressive and non-aggressive CD symptoms were entered in two separate steps and the order of entry of the predictors was alternated to determine the unique contribution of the two predictors. These analyses were also run separately for teacher and parent ratings. The analytic framework of Baron and Kenny (1986) was used to investigate whether anxiety symptoms mediate the relationship between CD and cortisol. Again these analyses were run separately for teacher- and parent-reported symptoms.

Scores on the DSM-IV scales represented a wide range of symptom intensity. To render the distributions of symptom ratings more symmetrical, individual symptom ratings were transformed to the natural logarithm scale with a constant of 1 added.

All tests of hypotheses were two-tailed with a significance level of α=0.05. Following Cohen's guidelines (Cohen, 1992), R^2 values of 0.01, 0.09, and 0.25 are generally used as thresholds to define small, medium, and large effects, respectively.

This study combined the data of children with a research diagnosis of ODD/CD and children without such a diagnosis. The appropriateness of this procedure was confirmed by a regression analysis showing that the presence of absence of a diagnosis did not significantly predict cortisol concentrations, R^2=0.09, $F(1,23)$=2.30, ns.

3. Results

3.1. CD symptoms

3.1.1. Teacher ratings

The relationship between teacher-reported CD symptoms and basal cortisol was significant, R^2=0.38, $F(1,23)$=13.90, P=0.001. CD symptoms predicted 38% of the variance in cortisol concentrations. As hypothesised and illustrated in Fig. 1, higher ratings of CD symptoms were associated with lower cortisol.

3.1.2. Parent ratings

In contrast to the teacher ratings, parent-reported CD symptoms did not significantly predict cortisol, R^2<0.01, $F(1,23)$=0.01, ns.

3.2. Aggressive and non-aggressive CD symptoms

3.2.1. Teacher ratings

To test the hypothesis that the relationship between CD and basal cortisol holds in particular for aggressive as opposed to non-aggressive CD symptoms, two regression analyses were run. In the first analysis, aggressive CD symptoms were entered in step 1 and non-aggressive CD symptoms were entered in step 2.

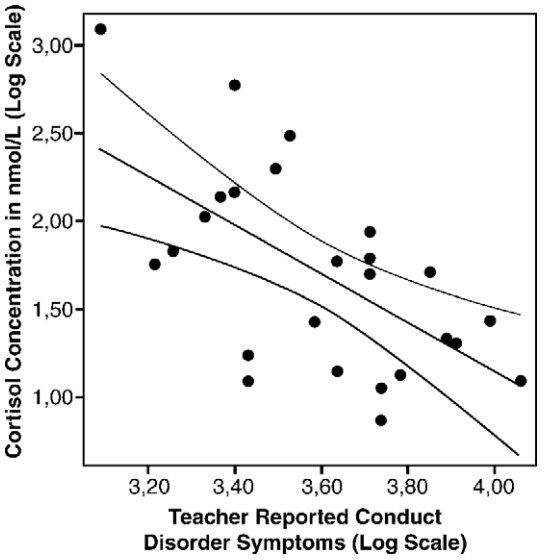

Fig. 1. Relationship between teacher-reported conduct disorder symptoms and basal cortisol concentrations (variables transformed to the natural logarithm). The middle line represents the linear regression (R^2=0.38). The upper and lower lines represent the 95% confidence interval.

Teacher-reported aggressive CD symptoms explained 32% of the variance in the cortisol concentrations in step 1, R^2=0.32, F(1,23)=10.89, P=0.003. In step 2, non-aggressive CD symptoms did not predict significantly over and above the aggressive CD symptoms, R^2 change=0.01, F(1,22)=0.39, ns. When the order of entry of the predictors was reversed in the second analysis, non-aggressive CD symptoms predicted cortisol in step 1, R^2=0.14, F(1,23)=3.77, P=0.064, although this effect did not reach conventional levels of significance. Aggressive CD symptoms predicted a significant proportion of the variance in the cortisol concentrations in step 2, R^2 change=0.19, F(1,22)=6.34, P=0.020. An additional 19% of the variance in the cortisol concentrations was explained by aggressive CD symptoms over and above non-aggressive CD symptoms. These results support the hypothesis that aggressive CD symptoms are more clearly related to low cortisol than non-aggressive CD symptoms.

3.2.2. Parent ratings

Parent-reported aggressive CD symptoms did not predict cortisol in step 1, R^2<0.01, F(1,23)=0.01, ns. In step 2, non-aggressive CD symptoms did not predict significantly over and above the aggressive CD symptoms, R^2 change=0.02, F(1,22)=0.53, ns. When the order of entry of the predictors was reversed, similar results were obtained.

3.3. Exploratory analyses: anxiety symptoms as mediator

3.3.1. Teacher ratings

These analyses tested whether the inverse relationship between CD symptoms and basal cortisol was mediated by anxiety symptoms. Baron and Kenny (1986) indicate a series of requirements that must be true for a mediation model to hold. In regression analyses, significant relations should be demonstrated between (1) the predictor (CD symptoms) and the dependent variable (cortisol), (2) the predictor and the mediator (anxiety symptoms), and (3) the mediator and the dependent variable while controlling for the effect of the predictor. The fourth requirement is necessary only for complete mediation: If the predictor no longer has any effect on the dependent variable when the mediator has been controlled, complete mediation has occurred. Partial mediation occurs if the first three requirements are met. The previously described inverse relationship between CD symptoms and cortisol satisfies the first requirement. The second requirement was also met: CD symptoms explained 76% of the variance in the mediator anxiety symptoms, R^2=0.76, F(1,23)=71.11, P<0.001. This result shows that CD and anxiety symptoms are strongly correlated and suggests multicollinearity. The third requirement involves establishing that the mediator anxiety symptoms affects cortisol after controlling for CD symptoms: anxiety symptoms did not account for a significant proportion of the variance in cortisol after controlling for the effects of CD symptoms, R^2 change<0.01, F(1,22)=0.16, ns. In contrast to what was expected, high ratings of anxiety symptoms were associated with low cortisol concentrations. These results suggest that anxiety does not mediate the relationship between CD symptoms and cortisol. Neither of the two predictors significantly contributed to the prediction of cortisol, t(22)=0.13, ns and t(22)=1.69, ns, for anxiety and CD symptoms, respectively. The partial correlation between CD symptoms and cortisol concentrations, partialling out the effects of anxiety symptoms, was −0.34. The partial correla-

tion between anxiety symptoms and cortisol was almost zero (−0.03).

3.3.2. Parent ratings

The mediator hypothesis for parent-reported anxiety symptoms was not investigated because of the absence of a relationship between parent-reported CD symptoms and cortisol.

4. Discussion

In this study, basal salivary cortisol concentrations were measured in a sample of 25 children that covered a broad range of antisocial behavior. The predictive power of CD symptoms for cortisol concentrations was investigated for both parent- and teacher-reported symptoms separately. Furthermore, the role of parent- and teacher-reported aggressive and non-aggressive CD symptoms, as well as the possible mediating role of anxiety symptoms, was investigated.

Significant findings were obtained only for teacher-reported symptoms. None of the regression models with parent-reported symptoms significantly predicted basal cortisol. The findings for teacher ratings were as follows: Severity of CD symptoms was inversely related to cortisol concentrations. CD symptoms predicted 38% of the variance in cortisol. The current finding of an inverse relationship between CD symptoms and basal cortisol converges with earlier studies (Vanyukov et al., 1993; Scerbo and Kolko, 1994; Moss et al., 1995; Van Goozen et al., 1998; McBurnett et al., 2000; Pajer et al., 2001; Karyawasam et al., 2003; Shoal et al., 2003; Van de Wiel et al., 2004).

Aggressive CD symptoms predicted basal cortisol, but non-aggressive CD symptoms did not. Aggressive CD symptoms explained a similar amount of the variability in the cortisol concentrations compared with ratings of all CD symptoms (38% and 32%, respectively). These findings replicate earlier studies (McBurnett et al., 1996, 1997, 2000; Pajer et al., 2001).

Anxiety symptoms were not found to mediate the relationship between CD and basal cortisol (McBurnett et al., 1991). In contrast to what was expected (Kagan et al., 1987, 1988; Scerbo and Kolko, 1994), high ratings of anxiety symptoms were associated with low cortisol concentrations in the present sample. This finding might be related to the nature of the present sample, which includes children with disruptive behavior problems. Furthermore, CD symptoms were strongly correlated with anxiety symptoms. After the study of McBurnett et al. (1991), subsequent studies, including the present study, failed to find support for a mediating role of anxiety in the relationship between CD and cortisol (Schulz et al., 1997; Van Goozen et al., 1998).

The associations between basal cortisol concentrations and symptom dimensions differed depending on the source of information used. Significant findings were only obtained using teacher-reported symptoms. One reason for this may be that teachers are more adept at observing CD symptoms than parents (Achenbach et al., 1987; Loeber et al., 1991; Hart et al., 1994; Offord et al., 1996). Few studies that investigated cortisol in children with antisocial behavior differentiated between parent and teacher measures. Schulz et al. (1997) found that neither parent nor teacher ratings of antisocial behavior were correlated with cortisol. Scerbo and Kolko (1994) found that clinic staff ratings of ODD symptoms predicted cortisol, whereas parent and teacher ratings of ODD symptoms did not. One study reported no differences between a parent-defined group of children with ODD and CD and a normal control group (Stoff et al., 1992). In that study, teacher information was not collected. One study reported a negative relationship between teacher-rated antisocial behavior and cortisol (Van Goozen et al., 1998), but that study did not report parent-rated behavior. The evidence for low basal cortisol in antisocial behavior seems to be somewhat stronger for teacher-reported antisocial behavior.

The precise mechanism linking low basal cortisol to CD is currently unclear. The release of cortisol has been linked to functioning of the serotonergic (5-hydroxytryptamine, 5-HT) system, although the precise relationship between cortisol and the serotonergic system has not yet been elucidated (Porter et al., 2004). A large body of evidence supports the notion that a decreased central serotonergic function is related to impulsivity, aggression, and other aspects of antisocial behavior (Pliszka, 1999). Our finding of an inverse relationship between CD symptoms and cortisol may be compatible with the hypothesis of

decreased serotonergic functioning in antisocial behavior.

It is currently unknown whether low basal cortisol concentrations are the cause or the consequence of antisocial behavior. This issue calls for longitudinal research. There are at least two possible explanations for the inverse relationship between CD and cortisol that differ in the temporal relationship between onset of low cortisol levels and onset of antisocial behavior (Moss et al., 1995).

One possibility is that low cortisol levels result from chronic exposure to contextual stressors, which leads to a blunted response to stress of the HPA axis, resulting in reduced cortisol secretion (Tsigos and Chrousos, 2002; Miller and O'Callaghan, 2002; McBurnett et al., 2003). Children with CD have a higher risk of being exposed to chronically stressful social environments. Family adversity is a significant contributor to the risk for CD (McGee and Williams, 1999). In other words, low levels of cortisol may reflect a muted response to stress, more specifically an increased threshold for stress (McBurnett et al., 1996).

A second possibility is that low cortisol levels precede the onset of antisocial behavior. In this view, low cortisol concentrations might be an early manifestation of antisocial behavior. McBurnett et al. (1996) have even suggested that low cortisol might be a risk factor for early-onset chronic aggressive antisocial behavior. In this view, antisocial behavior is interpreted aimed at restoring low levels of autonomic activity. This is consistent with the finding that antisocial behavior is associated with sensation seeking (White et al., 1994). Several studies have shown that children at high risk for antisocial behavior show low basal cortisol concentrations. Moss et al. (1995) found lower cortisol levels in sons of fathers with a lifetime diagnosis of psychoactive substance use disorder (PSUD) than in sons of fathers without psychopathology. Vanyukov et al. (1993) found that cortisol was lower in sons of fathers with a history of CD that progressed to antisocial personality disorder than in sons of fathers without such a history or fathers that had CD but did not develop antisocial personality disorder. Strong evidence for the idea that low cortisol levels precede the onset of antisocial behavior was found in a recent longitudinal study by Shoal et al. (2003) who demonstrated that low resting cortisol concentrations predicted aggression 5 years later.

This study has some shortcomings that could be addressed in future studies. First, the sample size was small, limiting the power to detect significant effects. Nevertheless, most findings were consistent with the hypotheses and converged with previous research. Second, by using a single saliva sample and not controlling time of saliva collection, we may have increased variability in the cortisol values. Cortisol is released in a pulsatile fashion in response to homeostatic control and is known to follow a circadian rhythm (Kirschbaum and Hellhammer, 1989; McBurnett et al., 2003). Multiple assessments would reduce the variability as would control of time of sampling. The limitations in our cortisol sampling, however, do not seem to invalidate our findings. Between-subject variation in the time of saliva collection was limited (4 h and 25 min maximum), and consistent with other studies (McBurnett et al., 1991, 1997), there was no linear or curvilinear relationship between time of collection and cortisol concentrations. Importantly, most of our findings were consistent with the hypotheses and converged with previous research. Third, this study uses a cross-sectional approach and was not suited to investigate a possible causal relationship between low basal cortisol and the later emergence of antisocial behavior (Shoal et al., 2003).

In the present study, teacher-reported CD symptoms explained a substantial proportion of the variance in basal cortisol concentrations as measured in saliva. This finding supports biologically based models of antisocial behavior in children involving reduced autonomic activity. The mechanism by which low cortisol is implicated in antisocial behavior in children calls for further study.

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