Elevated Trajectories of Externalizing Problems Are Associated With Lower Awakening Cortisol Levels in Midadolescence

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A growing body of research suggesting a negative association between basal levels of cortisol and persistent antisocial behavior has emerged. The present study examined relations between awakening cortisol levels and antisocial trajectories from ages 5 to 15 years among individuals in the National Institute of Child Health and Human Development Study of Early Child Care and Youth Development. Antisocial behavior was defined by semiparametric group modeling techniques, which revealed antisocial patterns parallel to Moffitt’s (1993) taxonomy of antisocial trajectories. In contrast to the claim that biological diatheses are uniquely characteristic of individuals who demonstrate an early-onset pattern of antisocial behavior, our results suggest that individuals with elevated patterns of antisocial behavior between 5 and 15 years of age—irrespective of the timing of onset or desistance—are more likely to evidence lower awakening cortisol levels compared with individuals with persistently low levels of antisocial behavior.

Keywords: antisocial behavior, cortisol, externalizing problems, taxonomies of antisocial behavior
In the last 2 decades a growing body of biopsychological research has generally found negative associations between basal levels of cortisol and levels of externalizing or antisocial behavior (Kobak, Zajac, & Levine, 2009; Lahey, McBurnett, Loeb, & Hart, 1995; Van Goozen, 2005; Van Goozen, Fairchild, Snoek, & Harold, 2007). These findings accord with a larger body of theory (see e.g., Brennan & Raine, 1997) and psychobiological evidence that has pointed to a pattern of psychophysiological hypoarousal in individuals both at risk for and who have evinced a chronic pattern of antisocial behavior (Magnusson, 1986; Raine, 2002; Raine, Brennan, & Farrington, 1997; Raine & Venables, 1984; Raine, Venables, & Williams, 1990). Negative associations between basal cortisol levels and antisocial behavior specifically implicate a dysregulation of the hypothalamic pituitary adrenal (HPA) axis as a risk factor for antisocial behavior and are consistent with the attenuation hypothesis of persistent antisocial behavior (Roisman et al., 2009; Susman, 2006). In the present study we examine whether awakening cortisol levels at age 15 years are associated with trajectories of antisocial behavior from ages 5 through 15 among individuals in the NICHD (National Institute of Child Health and Human Development) Study of Early Child Care and Youth Development (SECCYD).

Two principal arms of the endocrine system constitute the human stress response, and these pathways can be distinguished, in part, on the basis of their hormonal secretions from the adrenal glands. The locus coeruleus (LC)/noradrenergic sympathetic system or the sympathetic adrenal medullary (SAM) system responds immediately to threatening stimuli in the environment producing fight or flight reactions. In contrast, the HPA axis is activated subsequent to the SAM system when cortisol is secreted from the adrenal cortex (Chrousos, 1998; Chrousos & Gold, 1992). The secretion of cortisol from the adrenal cortex is the end product of a cascade of events that begins with the release of corticotropin releasing factor (CRF), vasopressin, and other regulatory neuropeptides from the paraventricular nucleus of the hypothalamus. The release of CRF promotes the secretion of adrenocorticotropic hormone in the pituitary, which in turn leads to the release of cortisol from the adrenal cortex. Suppression of the HPA system is achieved via negative feedback of cortisol on sites in the pituitary and hypothalamus that inhibit further release of CRF. Over time, chronic activation of the HPA axis can lead to a down-regulation of cortisol production from the adrenal cortex, leading to cortisol hyporeactivity (i.e., HPA attenuation; Fries, Hesse, Hellhammer, & Hellhammer, 2005; Gunnar & Vazquez, 2001; Susman, 2006).

Although a number of empirical studies have found evidence for a negative relation between basal cortisol levels and externalizing or antisocial behavior (Flinn & Englund, 1995; Kobak et al., 2009; Lahey, McBurnett, Loeb, & Hart, 1995; McBurnett et al., 1991; McBurnett, Lahey, Rathouz, & Loeb, 2000; Shirtcliff, Granger, Booth, & Johnson, 2005), others have failed to show this negative relation (Hart, Burock, London, Atkins, & Bonilla-Santiago, 2005; van Bokhoven et al., 2005) or demonstrated that the association varied by age (Shirtcliff & Essex, 2008). Moreover, evidence of the cortisol–antisocial behavior relation in adolescents and in nonclinical samples (Shirtcliff et al., 2005; Susman, Nottelmann, Dorn, Inoff-Germain, & Chrousos, 1988; Walker, Walder, & Reynolds, 2001) is thin relative to studies of children and clinical samples. Studies that have compared clinical groups of conduct-disordered children and adolescents with matched controls have revealed inconsistent findings regarding the negative association between low morning cortisol levels and antisocial behavior. For example, Fairchild et al. (2008) found no group differences in morning cortisol levels between participants with early-onset conduct disorder, adolescent-onset conduct disorder, and control participants. Further, they found higher basal cortisol levels in both conduct-disordered groups relative to control participants. Conversely, Popma et al. (2007) found significantly lower cortisol levels in the 1st hr after awakening in adolescent males with a disruptive behavior disorder who attended a delinquency diversion program compared with those in the program without a disruptive behavior disorder or matched normal controls. Alink and colleagues (2008) conducted a meta-analysis examining the cortisol–externalizing association in children and found a small but significant overall negative relationship between basal cortisol and externalizing problems (r = −.05). The largest combined effect size in the hypothesized negative direction was found for school-age children (r = −.14). However, the effect was in the opposite direction for preschool-age children (r = .09) and was nonexistent among adolescents (r = −.01). In discussing the implications of the meta-analysis, Alink et al. (2008) noted that one limitation of their meta-analytic approach was the inclusion of studies that represented a heterogeneous array of antisocial behavior types that precluded a more fine grained understanding of relations between cortisol and antisocial behavior subtypes or trajectories. Indeed, consistent use of well-established theoretical antisocial groupings (see e.g., Moffitt, 1993), as well as empirical derivations of them (see e.g., Roisman et al., 2010), are both likely to provide needed coherence to the literature on antisocial behavior. Moreover, utilizing these theoretical groupings will likely lead to a greater shared understanding of the etiological processes that characterize the emergence and maintenance of externalizing behavior.

Moffitt's (1993) seminal distinction between two patterns of antisocial behavior—one that begins in childhood and persists (life-course persistent; LCP) and one that begins and ends in adolescence (adolescence-limited; AL)—is perhaps the most well known and empirically validated taxonomy of antisocial trajectories in life-span developmental research. Moffitt originally referenced two patterns of antisocial behavior distinguishable with respect to both age of onset (preadolescent vs. adolescent) and persistence over time (adolescence-limited vs. not). In contrast, empirical tests of the theory (Aguiar, Sroufe, Egeland, & Carlson, 2000; Moffitt, Caspi, Dickson, Silva, & Stanton, 1996) have mainly compared children persistently antisocial throughout childhood and adolescence (early-onset/persistent; EOP) and those whose antisocial behavior emerges in adolescence (adolescence-onset; AO) with one another and with youths who show little evidence of externalizing problems through their mid- to late teens (low or never antisocial; NA). Additionally, a small group of individuals who show elevated antisocial behavior in childhood but not adolescence (recoveries, or childhood limiteds; CLs) has been routinely identified across studies (Aguiar et al., 2000; Moffitt et al., 1996).

Moffitt (1993) originally theorized that, whereas the AO pattern of antisocial behavior reflected a developmentally normative, temporary deviation involving mimicry of antisocial peers, the EOP pattern is rooted in early intrapersonal risks—such as ill health, difficult temperament, and subtle neuropsychological deficits—that are amplified by chronic contextual adversity. Given this
account, one might expect that associations between HPA-axis functioning and antisocial behavior would most likely be associated with the EOP antisocial pattern. However, since Moffitt’s (1993) original account of the LCP and AL trajectories, research (Aguilar et al., 2000; Ogdiers et al., 2008; Roisman et al., 2010) examining antecedents and sequelae of these groups has suggested that etiological differences between early and adolescent-onset individuals may not be clear-cut. More importantly, AO antisocial behavior may also have negative implications for mental health outcomes (Moffitt, Caspi, Harrington, & Milne, 2002) and may be less benign than is implied by the notion that it is a short-term behavioral deviation involving mimicry of antisocial peers (Roisman et al., 2010). Of particular relevance to the current study, Roisman et al. (2010) found, using empirically derived antisocial groups paralleling Moffitt’s (1993) taxonomy, that, compared with those individuals with consistently low levels of antisocial behavior—irrespective of its timing of occurrence (i.e., early or adolescence onset)—were characterized by both contextual (i.e., maternal insensitivity, low income-to-needs, single-parent status) and intraindividual (i.e., poor cognitive functioning, ill health, difficult temperament) risk from early childhood to adolescence.

Building on earlier work from the NICHD SECCYD examining both the antecedents of awakening cortisol in adolescence (Roisman et al., 2009) and antisocial trajectories through early adolescence (Roisman et al., 2010), we explored relations between antisocial trajectories from ages 5 through 15 years and awakening cortisol in midadolescence. In keeping with Roisman et al.’s (2009) procedure, early demographic differences (i.e., child ethnicity, child gender, and family income-to-needs ratio) were used as covariates in our analyses. Of particular import in the current report, we identified antisocial trajectories prospectively from ages 5 to 15 via semiparametric group modeling (SPGM). This technique has been increasingly used in research on externalizing trajectories to define groups (Piquero, 2007; van Dulmen, Goncy, Vest, & Flannery, 2009). As aforementioned, Roisman et al. (2010) used these techniques to identify antisocial trajectories in the NICHD SECCYD consistent with Moffitt’s (1993) classification of developmental patterns of antisocial behavior (i.e., NA, EOP, and AO). By examining associations between awakening cortisol and empirically well-defined antisocial trajectories over the first 15 years of life, we sought to determine whether the expected negative relationship between awakening cortisol and antisocial behavior would emerge in a normative sample. Further, in light of Moffitt’s account that EOP pathway is thought to have its roots in early biological diatheses that are amplified by contextual risk, we were interested in ascertaining whether relations between awakening cortisol and antisocial behavior trajectories would be specific to the EOP group.

**Method**

**Participants**

Families were recruited during hospital visits to mothers shortly after the birth of a child in 1991 at 10 locations in the United States. Recruitment and selection procedures are described in detail elsewhere (see http://secc.rti.org). Briefly, during selected 24-hr intervals, all women giving birth were screened for eligibility and willingness to be contacted again. Of the 8,986 mothers who gave birth during the sampling period, 5,416 (60%) agreed to be telephoned in 2 weeks and met the eligibility requirements (mother over 18, spoke English, mother healthy, baby not multiple-birth or released for adoption, family lives within an hour of research site, move from the area not planned in the next year, neighborhood not deemed too dangerous by police to visit). Of that group, a conditionally random sample of 3,015 (56%) was selected for a phone call in 2 weeks. The conditioning assured adequate representation (at least 10%) of single mothers, mothers without a high school degree, and ethnic minority mothers. At the 2-week call, families were excluded if the baby had been hospitalized for more than 7 days, they expected to move in the next 3 years, or they could not be reached in at least three attempts at telephone contact. On the basis of the calls made, a total of 1,525 families were selected as eligible and agreed to an interview. Of these, 1,364 completed a home interview when the infant was 1 month old and became study participants.

The resulting sample was diverse: 24% were minority, 11% of the mothers had not completed high school, and 14% were single at the time of the infant’s birth. Mothers had an average of 14.4 years of education. Average family income was 3.6 times the poverty threshold. The participating families were similar to the eligible hospital sample in terms of maternal education, percentage in different ethnic groups, and presence of a husband/partner in the household. Following Roisman et al. (2010), antisocial trajectories were identified in the current report on the basis of 990 children with Child Behavior Checklist (CBCL; Achenbach, 1991b) and/or Youth Self-Report (YSR; Achenbach, 1991c) scores at age 15 (see Roisman et al., 2010, for attrition analyses). The final analytic sample, however, consisted of the subset of 813 individuals from whom cortisol data were collected at age 15 and key cortisol (e.g., time of awakening) and demographic (e.g., income-to-needs) covariate data were also available.

**Identification of Empirically Derived Antisocial Groupings**

In the NICHD SECCYD, antisocial behavior from childhood to adolescence was assessed with the Externalizing scale of the parent (CBCL), teacher (TRF), and youth (YSR) versions of the CBCL (Achenbach, 1991a, 1991b, 1991c, 1997; Achenbach & Edelbrock, 1986) for the following assessment points, at which data were concurrently obtained from both mothers and teachers: kindergarten and Grades 1, 3, 4, 5, and 6. Antisocial behavior in adolescence was assessed with the parent and youth self-report versions of the CBCL collected when the youths were 15. At each time point, the highest raw score of externalizing among reporters was used as a measure of a study child’s externalizing behavior. Of note, because children were all the same age at each assessment point, the use of raw scores (vs. T scores) is an appropriate analytic strategy. The Externalizing scale showed adequate reliability across time, with the coefficient alpha averaging .89 for maternal reports and .95 for teacher reports across the childhood assessments. Antisocial behavior in adolescence was also reliably assessed (for mother report, α = .91, and for youth self-report, α = .86). The TRF and CBCL Externalizing scores were moderately correlated within assessment points during childhood, with correlations ranging from .23 to .41 (all ps < .01; mean r = .34). The
SPG (Nagin, 2005; Nagin & Land, 1993) was used to derive an empirically based antisocial grouping solution that best described these data from the NICHD SECCYD. Briefly, SPG is an exploratory, data-driven analytic technique that identifies groups of individuals through a clustering algorithm rather than by a priori conceptualizations. A benefit of this technique over theoretically derived antisocial groupings is that group-based modeling ensures that groups are significantly different from one another on the characteristic of interest (here, patterns of antisocial behavior). SPG modeling yielded a five-group solution consisting of the following antisocial groupings: (1) low: 41.8% of the sample (n = 414; 173 male, 241 female); (2) moderate: 33.7% of the sample (n = 334; 176 male, 158 female); (3) CL: 7.1% of the sample (n = 70; 45 male, 25 female); (4) AO: 12.4% of the sample (n = 123; 68 male, 55 female); and (5) EOP: 4.9% of the sample (n = 49; 31 male, 18 female; see Figure 1; see also Roisman et al., 2010).1

**Cortisol Assessment**

A complete description of the age-15 cortisol assessment can be found in Roisman et al. (2009). In brief, at the age-15 home visit, adolescents and parents were given detailed instructions and a demonstration of the saliva collection procedure. Following written instructions, adolescents collected saliva for cortisol assay upon awakening in the morning for three consecutive school days using a salivette (Sarstedt, Numbrecht, Germany) provided by the research team. They were instructed not to eat anything prior to saliva collection and to wash their mouth with water immediately on awakening using a cup of water and an empty cup placed by the bedside the previous evening. Adolescents were also told to keep the cotton roll from the salivette in their mouth for 3 min, then place the moist roll in the salivette and place it in a freezer until taken to the data collection site. All samples were assayed with a highly sensitive enzyme immunoassay specifically designed for use with saliva (Cat. No.1-0102/1-0112; Salimetrics, http://www.salimetrics.com). The test has a calibrator range of 0.012–3.000 µg/dl and a sensitivity of <0.003 µg/dl. Average intra- and interassay coefficients of variation were 5.34% and 9.86%, respectively. The standard curve was highly reproducible (mean $R^2 = .998$). Samples were assayed in duplicate (0.8%, 1.2%, and 1.3% of participants lacked duplicates on Days 1, 2, and 3, respectively), and the average was used in analyses. On the basis of prior analyses of potential correlates of awakening cortisol (see Roisman et al., 2009), a brief report of general sleep problems and average time of awakening (in minutes after midnight) were used as covariates in order to examine whether these two variables accounted for any association between the antisocial groupings and awakening cortisol levels (see the Results section). Cortisol values (µg/dl) were averaged over the 3 days of data collection.2

**Analytic Plan**

In order to examine potential awakening cortisol differences between the empirically derived antisocial groupings, we conducted two analyses of covariance with the antisocial groups as predictors and the average age-15 awakening cortisol as the outcome. In the first model, we utilized general sleep problems and time of awakening as covariates. In the second model, we also included child gender, child race, family income-to-needs, and maternal education in early childhood as covariates. The low antisocial group was used as the reference group in both analyses. In both models, the primary contrasts of interest were each of the elevated antisocial groups (EOP, AO, CL, and moderate) in relation to the low externalizing group (NA). In light of Moffitt’s (1993) claim that endogenous neurobiological risk distinguishes EOPs from AOs, we also examined whether the AO and EOP groups significantly differed from one another on mean levels of cortisol but found no evidence of it.

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1 Recent evidence from a Monte Carlo simulation study has suggested that, in the case of strong model fit and high posterior probabilities, classifying individuals into their most likely trajectory group produces better specification of effects than do those that allow these models to vary probabilistically (Clark & Muthén, 2010). Given the high posterior probabilities of group membership for individuals in each of our trajectory groups (i.e., $>.70$; see Roisman et al., 2010), hard-binning individuals into their most likely trajectory of antisocial behavior is the most appropriate method of answering the research questions posed in this study.

2 Distributions of the cortisol values were examined to ascertain the cutoff at $\pm 3$ SDs above the mean. In order to adjust for outliers, values above this cutoff were assigned the next highest value that was less than or equal to the mean plus 3 SDs (there were no values minus 3 SDs). Because the average cortisol data were only moderately skewed (skew = 1.08), we used raw, untransformed data in analyses. Table 1 also includes results of analyses with outliers excluded. Inclusion of cortisol covariates was informed by Roisman et al. (2009), who, using the same data, found that only general sleep problems and average time awakening were significantly associated with awakening cortisol levels. Descriptive information regarding potential additional covariates, which were not significantly associated with awakening cortisol—including average minutes elapsed from awakening to cortisol acquisition, morning/evening preference, and (for female participants) days since first day of last menstruation—can be found in Roisman et al.
Group Differences on Age-15 Average Awakening Cortisol Levels

Table 1 reports the results of our analyses with outliers (i.e., <3 SDs) both included and excluded. In the models with outliers included, EOP (M = 0.30, SD = 0.18), CL (M = 0.32, SD = 0.18), and AO (M = 0.32, SD = 0.18) participants had significantly lower awakening cortisol levels (µg/dl) at age 15 than did low (M = 0.38, SD = 0.18) externalizers (the difference between the moderate [M = 0.36, SD = 0.18] and low groups was marginally significant). These differences in general were robust to demographic controls. In this model, the AO (M = 0.32, SD = 0.18) group had significantly lower awakening cortisol levels at age 15 than did the low (M = 0.38, SD = 0.18) externalizers. The EOP (M = 0.32, SD = 0.18) and CL (M = 0.33, SD = 0.18) groups also demonstrated marginally lower awakening cortisol levels than did the low externalizers (the difference between the moderate and low group was nonsignificant controlling for demographic covariates).

In the models with outliers excluded, we found similar results. Specifically, EOP (M = 0.27, SD = 0.16), CL (M = 0.31, SD = 0.16), and AO (M = 0.30, SD = 0.16) participants had significantly lower awakening cortisol levels at age 15 than did low (M = 0.37, SD = 0.16) externalizers (the difference between the moderate [M = 0.35, SD = 0.16] and low groups was no longer significant). These differences were also generally robust to demographic controls. In this model, the AO (M = 0.31, SD = 0.16) and EOP (M = 0.29, SD = 0.17) groups had significantly lower awakening cortisol levels at age 15 than did the low (M = 0.36, SD = 0.16) externalizers. The CL (M = 0.32, SD = 0.16) group also demonstrated marginally lower awakening cortisol levels than did the low externalizers. It is worth pointing out that the findings for the EOP group in this model were somewhat stronger than in the same model that included cortisol outliers.

Discussion

Although a growing body of extant research has suggested a link between attenuated basal cortisol levels and persistent antisocial behavior, questions concerning the specificity and developmental timing of this link remain (Alink et al., 2008). The current findings are particularly noteworthy because the results extend the evidence for a negative association between cortisol levels and elevated antisocial behavior into midadolescence. In addition, the findings were uncovered within the context of theoretically meaningful, empirically derived antisocial groupings parallel to Moffitt’s (1993) classic taxonomy of antisocial pathways. Important to this latter point, we found evidence that individuals characterized by CL and AO antisocial trajectories also demonstrated lower age-15 awakening cortisol levels than did individuals who showed the lowest levels of externalizing behavior. These findings were in general robust to demographic controls (i.e., gender, ethnicity, income-to-needs, and maternal education), with most findings remaining at least marginally significant. Of particular note, the negative relationship between low awakening cortisol and antisocial behavior was most robust in the AO antisocial trajectory group in that it remained significant after the inclusion of the demographic covariates. Last, it is noteworthy that we found that boys evinced lower levels of awakening cortisol than did girls. However, prior findings in the literature with respect to gender differences in cortisol levels have been equivocal (Maestripieri, Baran, Sapienza, & Zingales, 2010; Vigil, Geary, Granger, & Flinn, 2010; see also Clow, Thorn, Evans, & Hucklebridge, 2004). As such,

Table 1

<table>
<thead>
<tr>
<th>Variable</th>
<th>Outliers included (n = 813)</th>
<th>Outliers excluded (n = 783)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cortisol covariates</td>
<td></td>
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<tr>
<td></td>
<td>β (SE) p</td>
<td>β (SE) p</td>
</tr>
<tr>
<td>Intercept</td>
<td>.381 (.010) &lt;.000</td>
<td>.378 (.017) &lt;.000</td>
</tr>
<tr>
<td>General sleep problems</td>
<td>.003 (.001) .005</td>
<td>.003 (.001) .017</td>
</tr>
<tr>
<td>Average time of awakening</td>
<td>.001 (.003) .003</td>
<td>.001 (.000) .004</td>
</tr>
<tr>
<td>EOP</td>
<td>−.077 (.029) .009</td>
<td>−.060 (.031) .053</td>
</tr>
<tr>
<td>CL</td>
<td>−.057 (.026) .027</td>
<td>−.046 (.026) .084</td>
</tr>
<tr>
<td>AO</td>
<td>−.061 (.021) .003</td>
<td>−.052 (.021) .014</td>
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<tr>
<td>Mod</td>
<td>−.024 (.014) .099</td>
<td>−.018 (.015) .220</td>
</tr>
<tr>
<td>Low (reference)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gender (1 = male, 0 = female)</td>
<td>−.036 (.013) .005</td>
<td>−.023 (.011) .05</td>
</tr>
<tr>
<td>Ethnicity (1 = white, 0 = nonwhite)</td>
<td>.022 (.015) .161</td>
<td>.030 (.014) .05</td>
</tr>
<tr>
<td>Income-to-needs (6–36 mo.)</td>
<td>.002 (.003) .500</td>
<td>.002 (.002) .49</td>
</tr>
<tr>
<td>Mother’s education</td>
<td>−.001 (.003) .64</td>
<td>.000 (.003) .99</td>
</tr>
<tr>
<td>Model R²</td>
<td>.04 .05</td>
<td>.05 .06</td>
</tr>
</tbody>
</table>

Note. Effect sizes (Cohen’s d) for the cortisol covariates model (outliers included) ATG contrasts are as follows: EOP vs. low (0.44); CL vs. low (0.33); AO vs. low (0.34); and mod vs. low (0.13). Effect sizes for the demographic controls model (outliers included) ATG contrasts are as follows: EOP vs. low (0.32); CL vs. low (0.25); AO vs. low (0.28); mod vs. low (0.10); and gender (0.15). Effect sizes for the cortisol covariates model (outliers excluded) ATG contrasts are as follows: EOP vs. low (0.58); CL vs. low (0.33); AO vs. low (0.38); and mod vs. low (0.10). Effect sizes for the demographic controls model (outliers excluded) ATG contrasts are as follows: EOP vs. low (0.43); CL vs. low (0.25); AO vs. low (0.32); mod vs. low (0.06); gender (0.11); and ethnicity (0.14). ANCOVA = analysis of covariance; ATG = antisocial trajectory group; EOP = early-onset/persistent; CL = childhood-limited; AO = adolescence-limited; Mod = moderately low.
future research should explore whether observed relations between gender and HPA-axis functioning are associated with patterns of antisocial behavior over time.

These findings position the field to more informatively understand the relation between human stress system physiology—particularly HPA-axis activity—and the development of antisocial behavior. Because Moffitt’s (1993) developmental account of discrete antisocial pathways has generated a robust body of theory, research, and related findings, linkages between HPA-system activity and the ontogeny of antisocial behavior can be better understood rather than obscured. Our findings help contextualize earlier work with the NICHD SECCYD cohort that showed that early interpersonal experiences of maternal insensitivity and increased time in child-care centers were associated with lower awakening cortisol levels at age 15 (Roisman et al., 2009). Perhaps more importantly, the current findings demonstrate that both CL and AO externalizers also show evidence of hypocortisolism in adolescence, lending further support to the possibility that the AO antisocial trajectory in particular might not be as benign as once thought (Aguilar et al., 2000; Odgers et al., 2008; Roisman et al., 2010).

Some limitations of this work are important to note. Recruitment constraints (i.e., families were excluded if they lived in a neighborhood deemed dangerous by the police) likely resulted in highly antisocial youths being underrepresented in the NICHD SECCYD cohort. That said, it is possible that inclusion of highly antisocial youths would have strengthened the negative basal cortisol–antisocial relation found in this study, because studies of highly aggressive and disruptive antisocial youths have also found this negative relationship (see e.g., Hawes, Brennan, & Dadds, 2009; Popma et al., 2007). Second, because collection of cortisol did not begin until age 15, it is impossible to determine whether the current results of negative relations found between awakening cortisol and elevated antisocial behavior were preceded by a period during which positive relations between awakening cortisol and antisocial behavior might have been expected, consistent with the attenuation hypothesis (Susman, 2006), or alternatively, whether a consistent pattern of antisocial behavior over time may have altered the functioning of the HPA axis, leading to attenuated basal cortisol levels in adolescence. A third possibility may be that children with low cortisol levels are underaroused (Zuckerman, 1979) and have low levels of fear (Raine, 1996), both of which may lead to more persistent patterns of antisocial behavior. A final limitation of the current study is that because cortisol was sampled only once during the day we were unable to determine whether cortisol levels were robustly lower in the high externalizing groups or they were lower at only this specific point during the day.

In sum, the findings lend further support to the view that attenuated awakening cortisol, at least by midadolescence, is associated with persistent antisocial behavior over the early life course. This association was characteristic of all elevated antisocial trajectory groups (with the exception of the moderate group), regardless of the developmental timing of onset or desistance. As such, the current work adds to the growing database of research that seeks to refine our understanding of the psychobiology of antisocial behavior. Nonetheless, further prospective, longitudinal data are necessary to examine the processes involved in HPA-axis attenuation and whether this stress-system attenuation mediates relations between early contextual adversity and trajectories of persistent antisocial behavior. Future work examining HPA-axis functioning with respect to distinguishing trajectories of antisocial behavior consistent with developmental taxonomies such as Moffitt’s (1993) remains an important and rich area of investigation for developmental psychopathology.

References


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