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A longitudinal biosocial study of cortisol and peer influence on the development of adolescent antisocial behavior

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Summary It is increasingly recognized that in order to understand the complex phenomenon of antisocial behavior, interrelations between biological and social risk factors should be taken into account. In the current study, this biosocial approach was applied to examine the mediating role of deviant peers in longitudinal associations linking the level of hypothalamic-pituitary-adrenal (HPA) axis activity to aggression and rule-breaking.

Participants were 425 boys and girls from the general population, who were assessed yearly at ages 15, 16, and 17. As a measure of HPA axis activity, cortisol was assessed at awakening, 30, and 60 min later (the cortisol awakening response, CAR). Participants, as well as their best friend, reported on their own aggressive and rule-breaking behavior, thereby allowing to assess bidirectional influences within friendships.

Aggression was only predicted by a decreased cortisol level at awakening, and not by aggressive behavior of their friend. Decreased levels of cortisol at awakening predicted adolescents' rule-breaking, which subsequently predicted increased rule-breaking of their best friend. The latter was only found for adolescents who changed friends, as compared to adolescents with the same friend in every year. Gender differences were not found.

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These findings suggest that interrelations between biological and social risk factors are different for the development of aggression versus rule-breaking. Furthermore, decreased levels of HPA axis activity may represent a susceptibility to selecting deviant peers.
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1. Introduction

It is increasingly recognized that in order to understand the complex phenomenon of antisocial behavior, interrelations between biological and social risk factors should be taken into account (Bassarath, 2001; Dodge and Pettit, 2003; Raine, 2002; Susman, 2006). A frequently hypothesized and examined biological risk factor for antisocial behavior, is a decreased level of hypothalamic-pituitary-adrenal (HPA) axis activity (e.g. McBurnett et al., 2000; Popma et al., 2007). Associations between a decreased level of HPA axis activity and antisocial behavior have been confirmed, but less consistently for adolescent than for childhood samples (see review by Alink et al., 2008). At the same time, in adolescence deviant peer influence becomes a major social risk factor for antisocial behavior (Brown, 2004; Gardner and Steinberg, 2005). Moreover, there are indications that deviant peers may mediate the association between the level of HPA axis activity and antisocial behavior (Raine et al., 2005; Yanovitzky, 2005). To capture the developmental changes in HPA axis activity levels, peer relations, and antisocial behavior, which are characteristic for adolescence, longitudinal studies are required. Therefore, the current study focused on the mediating role of peer influences in longitudinal associations linking decreased levels of HPA axis activity to antisocial behavior.

A biological perspective on the development of antisocial behavior is offered by the low arousal theories (Raine, 1993; Zuckerman and Neeb, 1979). Low arousal is considered to constitute a negative physiological state, which could be increased (i.e., normalized) by seeking sensation through antisocial behavior (Zuckerman and Neeb, 1979). Alternatively, low arousal might reflect fearlessness, as a result of which youngsters may not fear the negative consequences of antisocial behavior (Raine, 1993). Although the exact mechanisms are unknown, it has been posed in theoretical models that low arousal may have resulted from genetic vulnerabilities or early life adversities. The amygdala is considered to link such early stressors to dysfunctions in arousal (Susman, 2006; van Goozen et al., 2007). The HPA axis is one of the major physiological stress systems, and low arousal can be operationalized as low levels of HPA axis activity. As a measure of HPA axis activity, salivary cortisol levels are often assessed. For instance, McBurnett et al. (2000) found that persistent aggression in school aged boys was associated with lower day time cortisol levels. Popma et al. (2007) specifically studied the cortisol awakening response (CAR) in adolescent boys, and reported that the level of the CAR, but not the response to awakening, was decreased in antisocial boys compared to normal controls. In a meta-analysis, however, associations between the level of HPA axis activity and antisocial behavior were not found in adolescent samples (Alink et al., 2008).

It is in adolescence when affiliation with deviant peers becomes an important social risk factor for developing antisocial behavior (Brown, 2004; Gardner and Steinberg, 2005; Hartup and Stevens, 1997). Peer influences are dynamic and bidirectional (Dishion and Owen, 2002; Popp et al., 2008): adolescents select friends who are similar to themselves in behavior and attitudes (selection), and friends become more similar to one another over time (socialization) (Brechwald and Prinstein, 2011; Kandel, 1978). Imbalance or dissimilarity between mutual friends' behavior and attitudes is likely to result in ending the friendship and seeking more similar friends, or to stay friends and modifying their own behavior to that of the friend (Kandel, 1978). Selection and socialization are not mutually exclusive, but can coexist and enhance one another. For instance, antisocial adolescents may select friends showing more antisocial behavior than themselves, which can exacerbate their own antisocial behavior (Gatti et al., 2005; Thornberry et al., 1993). However, friends also tend to overestimate the similarity between their behaviors, that is, an adolescent may feel his/her friends are equally antisocial as he/she is, whereas in fact the friends may be less antisocial (Aseltine, 1995). To overcome this overestimating of the similarities, and provide an accurate view of the friends' antisocial behavior, the best friends reported on their own behavior in the current study.

Antisocial friendships may mediate associations between the level of HPA axis activity and antisocial behavior. It has for instance been shown that sensation seeking, as associated with lowered levels of HPA axis activity (cf. the low arousal theory, see above), is also associated with affiliating with deviant friends (Yanovitzky, 2005). These deviant friends in turn may influence the adolescent toward behaving antisocially (Moffitt, 1993; Thornberry et al., 1994). Also, it has been shown that persistent antisocial youth show neurocognitive impairments compared to adolescence limited antisocial youth (Raine et al., 2005). This could imply that biological risk factors, including decreased levels of HPA axis activity, may be specific for persistent antisocial youths. As they are already involved in deviant behaviors, in adolescence they are more likely to select antisocial friends and influence others into antisocial behavior (Moffitt, 1993). Hence, two paths may be present linking the level of HPA axis activity to deviant friends: (1) influences of decreased levels of HPA axis activity may operate via deviant friends, and also (2) lower levels of HPA axis activity may first lead to adolescent antisocial behavior, which makes these adolescents more likely to have antisocial friends. Both pathways of influence will be tested in this study.

To clarify when and how the influence of friends comes into play, and to compare the two paths, a longitudinal design is required. To the best of our knowledge, the only study which has investigated HPA axis activity levels and peer influences, was cross-sectional in nature (Dorn et al., 2009). Dorn et al. found that children with disruptive behavior disorders showed lowest levels of HPA axis activity if they had friends who showed low levels of antisocial behavior. As these children already showed antisocial behavior, these

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findings indicate that their behavior was not the result of peer influence. However, these children were aged 6–11 years, and deviant peers do not become a major risk factor for antisocial behavior until adolescence (Brown, 2004; Gardner and Steinberg, 2005). To further investigate these promising findings, and incorporate the dynamics and bidirectionality of peer influences toward antisocial behavior in youths with decreased levels of HPA axis activity, a longitudinal design was applied in the current study.

Furthermore, the relative influence of biological and social risk factors may differ by the type of antisocial behavior. Aggression and rule-breaking are two main types of behavior often recognized within adolescent antisocial behavior (e.g. Achenbach et al., 1989; Burt, 2012). Both types are thought to result from biological as well as social risk factors (Moffitt, 1993; Raine et al., 2005). However, there are indications that aggression may be more strongly related to decreased levels of HPA axis activity (Burt, 2012; McBurnett et al., 2000; Platje et al., 2013a,b), whereas rule-breaking may be more strongly related to affiliation with and influence of deviant peers (Barnow et al., 2005; Reitz et al., 2007). Therefore, both types of antisocial behavior were assessed in this study.

For these reasons, in the current study, the role of deviant peer influences in longitudinal associations linking the level of HPA axis activity to aggression and rule-breaking was investigated in a general population sample of both boys and girls. Because within friendships bidirectional influences can occur, which may increase antisocial behavior, two indirect paths were examined: (1) do lower levels of HPA axis activity predict higher levels of antisocial behavior of the best friend, which in turn predicts higher levels of adolescent antisocial behavior, (2) do lower levels of HPA axis activity predict higher levels of adolescent antisocial behavior, which in turn predicts higher levels of antisocial behavior of the best friend? This was examined in a large sample of boys and girls, who participated in three annual assessments at ages 15, 16 and 17. As a measure of HPA axis activity, the CAR was assessed, and specified in two ways; firstly as cortisol levels at awakening, and secondly as the response in cortisol levels to awakening. The response to awakening has been shown to be influenced by situational factors (Fries et al., 2009; Hellhammer et al., 2007), therefore associations are expected to be stronger for the cortisol level at awakening. The best friends of the adolescents also participated, reporting on their own aggressive and rule-breaking behavior over the years. The best friend could change from year to year, and stability of the friendship was examined to account for selection and socialization effects. As the sample consisted of both boys and girls, and gender differences may be present in antisocial behavior (Moffitt, 2001) and/or HPA axis activity (e.g. Fries et al., 2009), gender was taken into account in all analyses.

2. Methods

2.1. Participants

Participants were 425 adolescents (239 boys, 186 girls) taking part in three annual assessments; at ages 15, 16 and 17 years. They were recruited from the RADAR (Research on Adolescent Development And Relationships) study. RADAR is a Dutch population based cohort study, with over-sampling (50%) of boys and girls with a borderline-clinical score on the externalizing scale of the Teacher's Report Form (TRF, Achenbach, 1991a) at age 11. All participants and their parents have provided written informed consent and received a reimbursement for their participation. The RADAR study has been approved by the responsible medical ethics committee, and was conducted in accordance with the Declaration of Helsinki. This study is based on data from the third (2008) to the fifth wave (2010) of RADAR, in this paper referred to as ages 15–17.

Of the 425 participants, 379 adolescents (89.2%) participated in the HPA axis measurements at any year. Participants in the HPA axis measurements did not differ in age, gender, pubertal status, BMI, nicotine use at age 16 and 17, or alcohol use at age 15 and 16 (t-tests and χ^2, all $ps > .05$) from those who did not participate, yet participants more often used alcohol at age 17 ($\chi^2(1) = 5.016$, $p = .05$) and nicotine at age 15 ($\chi^2(1) = 4.483$, $p = .05$). After exclusion on the basis of sampling errors, technical problems in the lab, or statistical outliers (see below), for 362 adolescents (332 at age 15, 283 at age 16 and 254 at age 17) HPA data was available for analyses.

Adolescents were asked to invite their best friend to participate in the study, and at any year, for 407 (95.8%) adolescents their best friend participated. At age 15 for 387 adolescents the best friend participated, for 381 at age 16, and 361 at age 17. They did not differ from participants for whom no friend participated on gender, pubertal status, BMI, nicotine or alcohol use (t-tests and χ^2, all $ps > .05$), but were on average 3 months younger ($t(423) = -3.719$, $p \leq 001$). Reciprocity in these friendships was examined, and the large majority (91.6% at age 15, 98.4% at age 16, and 98.3% at age 17) of best friends mentioned the adolescent as a friend.

Adolescents were specifically instructed to invite their *best friend*, and could therefore invite another best friend from year to year. All best friends were given their own ID number in the study, enabling the examination of stability of the friendship. At one or both intervals, 150 (38.8%) adolescents changed friends.

The number of participants fluctuated per year, with 417 participants at age 15, 405 at age 16 and 389 at age 17. Attrition was low over the three years, 16 (3.4%) dropped out at age 16, and 19 (4.7%) at age 17. Drop-out was not associated with gender ($\chi^2(1) = 1.899$, $p = .18$), but drop-outs were on average 3 months older ($t(423) = 5.027$, $p \leq 001$). To estimate the pattern of missing values, Little's Missing Completely at Random (MCAR) test (Little, 1988) was conducted. Although this very stringent test was significant ($\chi^2(2376) = 2609.032$, $p = .001$), the χ^2/df ratio of 1.10 indicated a good fit between sample scores with and without imputation (Bollen, 1989). Participants with partially missing data could thus be included in the analyses. The final models were ran on the 425 adolescents participating at any year, applying a full-information maximum likelihood estimation (Enders and Bandalos, 2001).

2.2. Aggression and rule-breaking

Antisocial behavior was assessed by means of the externalizing scales of the Youth Self Report (YSR, Achenbach, 1991b).

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Good reliability and validity have been reported for the Dutch YSR version (Verhulst et al., 1997). The adolescents and their best friends each reported on their own behavior. Within the externalizing dimension, sub-scales differentiate aggression and rule-breaking behavior. The aggression subscale consisted of 19 items (α's ranging from .86 to .87) assessing physical acts against persons or things (i.e., fighting, being cruel to others). The rule-breaking sub-scale consisted of 11 items (α's ranging from .70 to .73) assessing behaviors such as truancy and stealing. Items are scored on a three-point scale (0 = not true, 1 = somewhat true, 2 = very true or often true). Missings were handled according to the YSR manual guidelines; no more than 2 missings were allowed and these were replaced by the mean of the sub-scale.

2.3. HPA axis assessment

Cortisol was measured in saliva. Saliva samples were collected by passive drooling, immediately after awakening (Cort0), and 30 min (Cort30) and 60 min (Cort60) later. These three samples constitute the Cortisol Awakening Response (CAR, Pruessner et al., 1997). Cortisol sampling took place in February and March of each consecutive year, as soon as possible after assessing aggression and rule-breaking. Participants were first given detailed verbal and written information regarding cortisol measurements. Subsequently, saliva sampling was planned for a suitable morning on a regular weekday. The first sample (at awakening) was planned before 8 a.m., while taking into consideration the participant's normal schedule. Sampling times were set and written on a detailed instruction form.

Participants were instructed to rinse their mouths with water before sampling, and not to eat, drink milk or juice, smoke or brush their teeth before completing Cort60. They were requested to report the exact sampling times on the instruction form on the day of sampling, and also to report if mistakes were made in any of the above instructions. After collection, participants were asked to store the samples in the refrigerator and send them by mail to the research center the same day.

At the research center, all samples were checked for correctness of sampling. When necessary, e.g. when Cort0 was sampled after 8:00 a.m. or sampling time of Cort30 or Cort60 was over 15 min late, or mistakes were made in any of the other instructions, participants were asked to collect new saliva samples, and a new sampling day was scheduled. At age 15, 28 participants collected new saliva samples, 20 at age 16, and 15 at age 17. If, despite this, participants had still not sampled correctly, the incorrect samples were excluded. In total 39 samples (4 samples at age 15, 15 at age 16, and 20 at age 17) were excluded for incorrect sampling.

Saliva was stored uncentrifuged at $-20\,^{\circ}C$ until analysis. Salivary cortisol levels were analyzed using electrochemiluminescence immunoassay ECLIA (E170 Roche, Switzerland). The lower detection limit was 0.5 nmol/l, and mean intra-assay and inter-assay coefficients of variation were respectively 3.4% and 12.2%. Due to technical problems in the lab (i.e. in 84% of the samples too little saliva was present and 6% contaminated samples), 133 samples could not be assayed. Participants with samples that could not be assayed did not differ in age, gender, pubertal status, BMI, nicotine use at age 15 or alcohol use (t-tests and χ^2, all $ps > .05$), yet more often used nicotine at age 16 ($\chi^2(1) = 10.286$, $p = .003$) and 17 ($\chi^2(1) = 4.920$, $p = .034$).

2.4. Control variables

All control variables were assessed yearly though self-report at the same times as aggression and rule-breaking. As physical development, substance use and stressful experiences use are related to development of HPA axis activity (Platje et al., 2013a,b; Trickett et al., 2010) and may be associated with antisocial behavior, we took these variables into account in the analyses. Physical development was assessed as pubertal development and the body mass index. Pubertal development was measured by a modification of the Pubertal Development Scale (PDS, Petersen et al., 1988) consisting of seven questions regarding physical development, i.e. growth spurt, axillary hair, pubarche, menarche, thelarche, voice change and facial hair. It was assessed only at ages 15 and 16, as it was expected that at age 17 the large majority would be fully matured. At age 15, 41.9% scored in/over late pubertal range, at age 16 this was 73.6%. The body mass index was calculated from self-reported height and weight as weight in kg/(length in m)^2. Substance use was assessed as nicotine and alcohol use (Monshouwer et al., 2008). Alcohol use over the last four weeks was assessed by means of a six-option question, ranging from "none" to "daily". Nicotine use was assessed by a nine-option question ranging from "I have never smoked" to "I smoke every day". Stressful experiences in the past year, such as sexual assault, physical assault, and being threatened with violence, were assessed with a questionnaire based on the International Crime Victims Survey (ICVS; Nieuwbeerta, 2002), and specified by perpetrator (parent = 2, someone else = 1, not = 0).

2.5. Statistical analyses

Cortisol values over 3SD above the mean were defined as outliers and excluded (28 samples). The CAR was defined as the cortisol level at awakening (CAR_{level}) and the cortisol response to awakening ($CAR_{response}$) per year, with Latent Growth Modeling (LGM; e.g. Kline, 2005) within *Mplus* 6.0 (Muthén and Muthén, 2007) with maximum likelihood estimation (Satorra and Bentler, 1994). For each year, cortisol levels at awakening, and 30 and 60 min later, were used as indicators to estimate the latent intercept (i.e. the level of cortisol at awakening $-$ CAR_{level}) and slope (i.e. changes in cortisol from awakening through 30 and 60 min after awakening $-$ $CAR_{response}$) factors in LGM. The CAR_{level} and the $CAR_{response}$ were normally distributed. Of the control variables, only substance use was associated with predictors as well as outcome variables. The CAR was therefore controlled for substance use effects, by adding alcohol and nicotine use in the model for the CAR_{level} and $CAR_{response}$ as time-varying ordinal covariates on the cortisol levels at awakening for each year.

Structural equation modeling was performed in *Mplus*, with a full-information maximum likelihood estimation (Enders and Bandalos, 2001). Two cross-lagged panel models were used for aggression and rule-breaking respectively, on the three years of adolescent antisocial behavior, the CAR

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level and response, and best friend's antisocial behavior. The 1- and 2-year stability effects over the years for all variables were added, and within-year correlations between the variables, as longitudinal associations were of interest. Two annual intervals were available to assess longitudinal effects (ages 15–16, and ages 16–17). Six longitudinal cross-lagged effects were estimated per annual interval, from: (1) CAR_{level} to adolescent behavior one year later, (2) $CAR_{response}$ to adolescent behavior one year later, (3) CAR_{level} to best friend's behavior one year later, (4) $CAR_{response}$ to best friend's behavior one year later, (5) adolescent's behavior to best friend's behavior one year later and (6) best friend's behavior to adolescent's behavior one year later. Note that paths 5 and 6 are bidirectional, the other paths were also tested for bidirectionality, but non-significant Wald tests indicated that these could be removed to keep the model as parsimonious as possible (for aggression: *Wald(8)* = 1.976, $p = .98$; for rule-breaking: *Wald(8)* = 6.979, $p = .54$). The cross-lagged paths were found to be time-invariant, as constraining the effects of age 15–16, and from age 16–17, to be estimated the same did not worsen model fit (for aggression: *Wald(6)* = 6.812, $p = .34$; for rule-breaking: *Wald(6)* = 7.237, $p = .30$). This modification was therefore retained, to keep the model as parsimonious as possible. Finally, the mediating role of peer influences was modeled by estimating two indirect paths: one indirect path from CAR_{level} via the adolescent's behavior to the best friend's behavior, and one with an indirect path from CAR_{level} via the best friend's behavior to the adolescent's behavior. To test whether different models for boys and girls, or stable and changing friendships would be warranted, the models as described above were also performed as multi-group models by gender, and stability of friendship. Differences in the cross paths due to gender or stability of friendship were tested by constraining the paths to be equal, and evaluating a decrease in model fit with chi-square difference tests. Significant chi-square tests would indicate that the paths were significantly different. If the paths did not differ for boys and girls, gender was controlled for by adding gender to the model as a covariate at age 15.

3. Results

In Table 1 descriptive statistics are shown. It can be seen that the CAR_{level} was not correlated to aggression or rule-breaking, and a weak positive correlation was found between the $CAR_{response}$ and adolescent rule-breaking. The level of aggression or rule-breaking was moderately positively correlated to aggression or rule-breaking of the best friend. These correlations were comparable for age 16 and age 17.

Adolescents who changed best friends, showed more rule-breaking behavior at age 15, than adolescents who kept the same best friend ($t(382) = 2.091$, $p = .04$). No effects of stability of friendship on rule-breaking behavior were found at age 16 or 17, on aggressive behavior of the adolescent or aggression or rule-breaking behavior of the best friend at any time (t-tests, all $ps > .05$).

First, it was examined whether gender differences were present in longitudinal interrelations between the CAR, antisocial behavior of the best friend, and adolescent antisocial behavior. Therefore, structural equation models were performed for aggression and rule-breaking with gender as grouping variable in multi-group models. Overall the cross paths did not differ between boys or girls, for aggression ($\Delta\chi^2(6) = 2.945$, $p = .82$) or rule-breaking ($\Delta\chi^2(6) = 2.758$, $p = .84$).

To examine longitudinal interrelations between the CAR, antisocial behavior of the best friend, and adolescent antisocial behavior, structural equation models were tested for aggression and rule-breaking respectively. All variables showed significant stability over time, except for the $CAR_{response}$. Results of the cross paths between the CAR, antisocial behavior of the best friend, and adolescent antisocial behavior are shown in Table 2.

As can be seen in Fig. 1 and Table 2, higher levels of aggressive behavior were predicted by a lower CAR_{level}, over and above the effect of aggressive behavior in the previous year. Aggressive behavior of the adolescent was not predicted by the level of aggression of the best friend or vice versa.

Higher levels of rule-breaking behavior of the adolescent were predicted by a lower CAR_{level}, over and above the effect of rule-breaking behavior of the adolescent in the previous year. Also, more rule-breaking behavior of the adolescent predicted increased rule-breaking of the best friend one year later, over and above the effect rule-breaking behavior of the best friend in the previous year. Rule-breaking behavior of the adolescent was not predicted by the level of rule-breaking of the best friend (see Table 2).

Based on the notion that bidirectional influences within friendships toward aggression or rule-breaking may occur, two indirect paths were examined: (1) from CAR_{level} to

Table 1 Descriptive statistics.

	Means and SDs			Correlations at age 15				
	Age 15	Age 16	Age 17	1	2	3	4	5
1. CARlevel[a]	18.94 (2.60)	18.38 (3.21)	21.50 (4.23)					
2. CARresponse	−1.74 (2.17)	−0.83 (2.55)	−0.59 (1.86)	.507**				
3. Aggression adolescent	6.76 (5.65)	6.71 (5.67)	6.10 (5.21)	.036	.103			
4. Rule-breaking adolescent	3.28 (2.92)	3.68 (2.83)	3.63 (2.79)	.101	.119*	.700**		
5. Aggression friend	7.40 (5.31)	6.79 (4.99)	6.68 (4.63)	−.022	.038	.232**	.242**	
6. Rule-breaking friend	3.64 (2.94)	3.73 (2.83)	3.94 (2.84)	.018	.008	.247**	.329**	.624**

[a] in nmol/l.
* $p \leq .05$.
** $p \leq .01$.

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Table 2 Results of the structural equation models for aggression and rule-breaking.

Direct effects		Aggression β	Rule-breaking β
CAR_{level} age 15	→ASB adolescent age 16	−0.051*	−0.072**
$CAR_{response}$ age 15		0.036	0.013
ASB best friend age 15		0.015	0.036
CAR_{level} age 16	→ASB adolescent age 17	−0.068*	−0.090**
$CAR_{response}$ age 16		0.046	0.015
ASB best friend age 16		0.015	0.036
CAR_{level} age 15	→ASB best friend age 16	−0.037	0.006
$CAR_{response}$ age 15		0.013	−0.035
ASB adolescent age 15		0.028	0.100***
CAR_{level} age 16	→ASB best friend age 17	−0.049	0.007
$CAR_{response}$ age 16		0.016	−0.041
ASB adolescent age 16		0.030	0.098***
Indirect effects			
CAR_{level} age 15 → ASB best friend age 16 → ASB adolescent age 17		−0.001	0.000
CAR_{level} age 15 → ASB adolescent age 16 → ASB best friend age 17		−0.005	−0.008*

Note. Correlations between variables within years and stability of variables over years were also included in the models.
* $p \leq .05$.
** $p \leq .01$.
*** $p \leq .001$.

behavior of the best friend, to behavior of the adolescent, and (2) from CAR_{level} to behavior of the adolescent, to behavior of the best friend (see Table 2). For aggressive behavior, both indirect paths were not significant. For rule-breaking behavior, however, the second indirect path was significant: a lower CAR_{level} at age 15 predicted increased adolescent rule-breaking at age 16, which subsequently predicted increased rule-breaking of the best friend at age 17 (see Table 2).

Additionally, as 150 adolescents changed friends, it was examined whether the effects would differ by stability of friendship. For aggression, overall the cross paths did not differ between changing or stable friendships ($\Delta\chi^2(6) = 6.790$, $p = .34$). For rule-breaking however, the cross paths did differ by stability of friendship ($\Delta\chi^2(6) = 13.046$, $p = .04$). The effects as described above were only present for adolescents who changed friends. As can be seen in Table 3 and Fig. 2, higher levels of rule-breaking behavior of the adolescent were predicted by a lower CAR_{level}, over and above the effect of rule-breaking behavior of the adolescent in the previous year. Higher levels of rule-breaking behavior of the best friend were predicted by rule-breaking behavior of the adolescent. Rule-breaking behavior of the adolescent was not predicted by the level of rule-breaking of the best friend. Also, an indirect effect was found: a lower CAR_{level} at age 15 predicted increased adolescent rule-breaking at age 16, which subsequently predicted increased rule-breaking of the best friend at age 17.

4. Discussion

In the current study, the mediating role of antisocial behavior of best friends in longitudinal associations linking the level of HPA axis activity to rule-breaking and aggressive behavior,

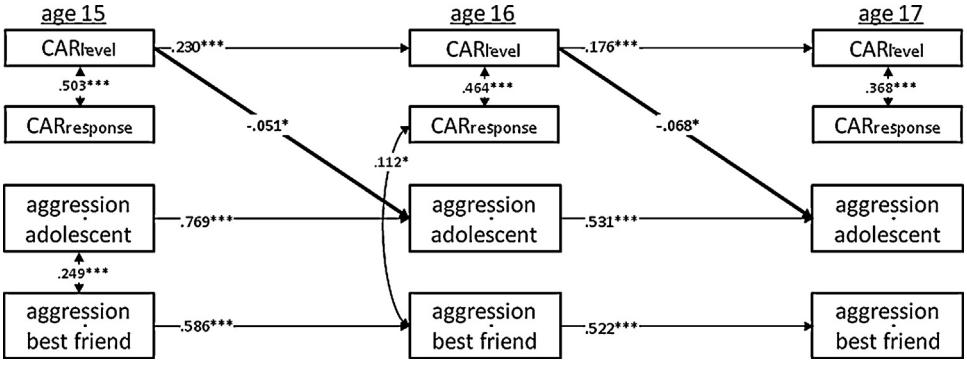

Figure 1 Cross-lagged panel model predicting aggressive behavior of the adolescent and the best friend, by aggressive behavior of respectively the best friend and the adolescent, as well as CAR_{level} and $CAR_{response}$. *Note*. Model fit: $X^2(38) = 45.714$; RMSEA = 0.022, CFI = 0.994, TLI = 0.988. Only significant effects are displayed. *$p \leq .05$, ***$p \leq .001$.

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Table 3 Results of the structural equation models for rule-breaking, by stability of friendship.

Direct effects	Rule-breaking	Changing friends β	Same friends β
CAR_{level} age 15	→ASB adolescent age 16	−0.133*	−0.044
$CAR_{response}$ age 15		−0.007	0.035
ASB best friend age 15		0.079	0.011
CAR_{level} age 16	→ASB adolescent age 17	−0.119*	−0.065
$CAR_{response}$ age 16		−0.008	0.039
ASB best friend age 16		0.072	0.012
CAR_{level} age 15	→ASB best friend age 16	−0.075	0.043
$CAR_{response}$ age 15		−0.044	−0.030
ASB adolescent age 15		0.244***	0.044
CAR_{level} age 16	→ASB best friend age 17	−0.071	0.063
$CAR_{response}$ age 16		−0.054	−0.033
ASB adolescent age 16		0.219***	0.046
Indirect effects			
CAR_{level} age 15 → ASB best friend age 16 → ASB adolescent age 17		−0.016	0.002
CAR_{level} age 15 → ASB adolescent age 16 → ASB best friend age 17		−0.087*	−0.006

Note. Correlations between variables within years and stability of variables over years were also included in the models.
* $p \leq .05$.
*** $p \leq .001$.

was investigated. Results revealed that a decreased level of HPA axis activity predicted adolescent rule-breaking, which subsequently predicted increased rule-breaking of the best friend. This was only found for adolescents who changed friends, and not for those with stable best friends. Aggression, on the other hand, was only predicted by a decreased level of HPA axis activity and not associated with aggressive behavior of friends. These effects were present over and above the prediction by prior antisocial behavior.

The findings on rule-breaking behavior indicate that adolescents with a decreased level of HPA axis activity may change their friendships toward more rule-breaking peers. In line with the low arousal theories, a decreased level of HPA axis activity predicted antisocial behavior (Raine, 1993; Zuckerman and Neeb, 1979). Decreased levels of HPA axis activity may be specific for persistent antisocial youths (Raine et al., 2005), who in adolescence, already being involved in deviant behaviors, are more likely to also select friends who are antisocial (Moffitt, 1993). It may also indicate that non-deviant adolescents reject friends who are becoming increasingly deviant. Nevertheless, the current results point to a selection effect rather than a socialization effect as it was present for those who changed friends only. This is in line with Knecht et al. (2010) who also found evidence for a selection effect in deviant friendships, but not for a socialization effect. However, scholars largely agree that these processes coexist (Haynie and Osgood, 2005; Kandel, 1978). Attention is therefore warranted, as by affiliating with other antisocial youths in adolescence, antisocial behavior within these friendships is likely to exacerbate (Gatti et al., 2005).

Aggression and rule-breaking are not only different expressions of antisocial behavior, the current results also point to a different biosocial interplay associated with these behaviors. This is in accordance with a meta-analysis on heritability of aggression and rule-breaking, which showed that whereas aggression was largely influenced by genetic

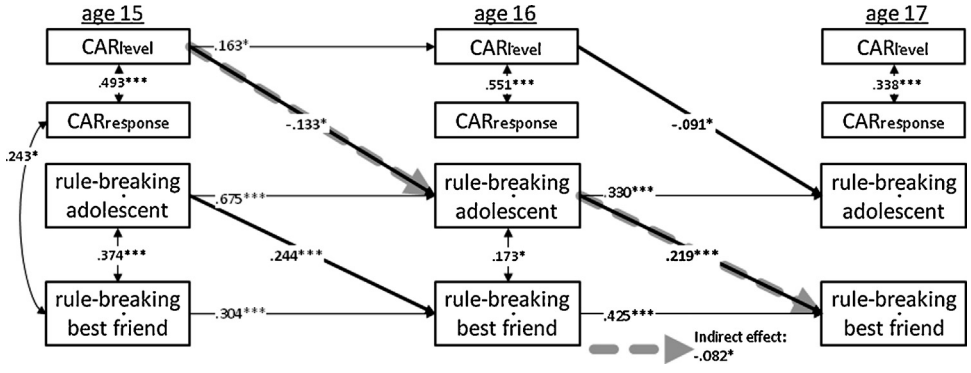

Figure 2 Cross-lagged panel model predicting rule-breaking behavior of the adolescent and the best friend, by rule-breaking behavior of respectively the best friend and the adolescent, as well as CAR_{level} and $CAR_{response}$. Model for changing friendships. Mediation (indirect effect) is depicted by the dashed arrow. *Note*. Model fit: $X^2(76) = 125.286$; RMSEA = 0.058; CFI = 0.959, TLI = 0.916. Only significant effects are displayed. *$p \leq .05$, ***$p \leq .001$.

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factors, with little environmental influences, the influence of genetic factors was significantly smaller for rule-breaking, where environmental influence had an important role (Burt, 2009). Also, another neurobiological parameter for low arousal, low resting heart rate, has been found to predict aggression, but not rule-breaking (Raine et al., 1997), whereas deviant peers have been found to be closely related to rule-breaking, and much less to aggression (Barnow et al., 2005). Together these results suggest, that although both aggression and rule-breaking result from an interplay between biological and social risk factors, the balance may differ by behavior. Aggression appears to be more strongly associated with biological risk, whereas rule-breaking appears to be associated with both biological and social risk factors.

The findings as described above were only present for the CAR_{level}, that is, the level of HPA axis activity at awakening, and not for the response to awakening. This could be explained by the finding that the $CAR_{response}$ did not show stability over the years. Indeed, the response to awakening has been shown to be influenced by situational factors (Fries et al., 2009; Hellhammer et al., 2007), and matures over adolescence (Platje et al., 2013a,b). The level of HPA axis activity may be a better reflection of trait-like HPA axis activity (Hellhammer et al., 2007), which facilitates finding longitudinal associations with behavior. Another explanation may be that, because of the continued maturation in adolescence, the response to awakening was often negative. A negative response may need a different interpretation, which could also explain the absence of an association with antisocial behavior.

In order to obtain a larger variance in antisocial behavior, youth with teacher-reported borderline clinical scores on externalizing behavior at age 11 were over-sampled (50%). At ages 15–17 only 11–14% scored in the borderline clinical range according to self-report, which is only slightly higher than what would be expected without over-sampling (Achenbach, 1991a). This sample thus largely reflect general population adolescent antisocial behavior, and results cannot be generalized to clinical or referred samples. Noteworthy is that equal numbers of boys and girls were over-sampled, resulting in relatively more girls showing high levels of antisocial behavior than generally found in the general population. Due to this equal gender distribution, the level of antisocial behavior in girls is similar to that in boys.

Although investigating gender differences was not an aim of this study and this was not examined in-depth, gender was taken into account. As overall gender differences did not become apparent, this suggests that the role of deviant peer influences in longitudinal associations linking the level of HPA axis activity to aggression and rule-breaking is similar for boys and girls. However, further research is needed to confirm this, especially as girls have been hypothesized to show differential interpersonal expressions of HPA reactivity to stress, marked by "tend and befriend" responses, as opposed to "fight or flight" responses (Taylor et al., 2000).

An important avenue for future longitudinal research is the causal directionality of associations between HPA axis activity and antisocial behavior. This is important because the HPA axis is considered to be one of the main systems involved in adaptation to the environment (McEwen, 2004). Behaving antisocially frequently could theoretically lead to habituation, which may result in (further) decreased HPA axis activity (van Goozen et al., 2007). It may also lead to more extreme forms of sensation seeking, and potentially more severe antisocial behavior. The body could continuously adapt to arousal as a result of sensation seeking, and requires increasingly more arousal to achieve the original effects. Although effects from antisocial behavior to HPA axis activity were not the aim of the current study, we did examine such reverse associations, which were not found. This may indicate that sensation seeking through antisocial behavior was successful, without habituation effects. Perhaps in younger samples, or with a shorter assessment interval, possible bidirectional effects may be revealed.

There are some methodological limitations of the study that should be noted. First, each year the CAR was assessed in saliva sampled at home on one day only. Correcting for day-to-day variation was therefore not possible. Previous studies have however reported that the CAR shows medium to high stability across days (Edwards et al., 2001; Kudielka and Wust, 2010; Roisman et al., 2009; Wust et al., 2000). Although we took all possible precautions in the sampling procedure, among which self-report of exact sampling times, directly monitoring participant's compliance to the CAR assessment was not possible. However, self-reported sampling times have been found to be preferable to automatic time recording (Kraemer et al., 2006) and sampling of the CAR at home was previously found not to differ from sampling in a controlled laboratory environment (Wilhelm et al., 2007). Second, information on medication use was unavailable, and could not be controlled for in the analyses. However, psychostimulantia, which are most frequently used by antisocial adolescents, have previously been reported not to be associated with differences in salivary cortisol in the morning (Hibel et al., 2007). Third, use of oral contraceptives was not controlled for, as this information was only available for circa 60% of the girls, and no effects on the CAR were previously found in this sample (Platje et al., 2013a,b). Another study showed that girls using OC displayed a slightly blunted response, but the level of the CAR was not different from that of free-cycling girls (Bouma et al., 2009). As the findings in the current study were found on the level of the CAR, potential effects of OC use are expected to be minimal. Fourth, this study was performed in a general population sample, and although youths with scores in the borderline clinical range at age 11 were over-sampled, this was effective only to a limited extend, results therefore reflect normative levels of antisocial behavior. As such, the results cannot be generalized to e.g. clinic-referred youths with disruptive behavior disorders or severe delinquent populations. As especially for the most severely disturbed youths their neurobiological deficits are expected to interact cumulatively with their adverse social environment (Moffitt, 1993), biosocial studies are particularly warranted in these youths, in order to provide tools to intervene in this adverse process.

In conclusion, these findings suggest that a decreased level of HPA axis activity may represent a susceptibility to selecting deviant peers. The current results are an important first step, yet further research is essential to examine whether a decreased level of HPA axis activity could potentially serve as a biomarker for friendship selection toward deviancy. Also, as friendships often concern more than two

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friends, future research should investigate whether the same principle holds true in groups of antisocial youths. Furthermore, these results add to the increasing evidence of different pathways to aggression and rule-breaking. This implies that eventually, intervention and prevention may also need to be directed at behavior specific factors in order to be successful. Therefore, more research is required to elucidate the possible different etiologies for aggression and rule-breaking, and provide tools for behavior specific interventions.

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Conflict of interest

All authors declare that they have no conflict of interest.

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