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

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Received 8 February 2013; received in revised form 9 July 2013; accepted 10 July 2013

KEYWORDS
HPA axis; Cortisol; Aggression; Rule-breaking; Peer influences; Longitudinal

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.
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 Neub, 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 Neub, 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 Goorzen 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; Kendel, 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 (Kendel, 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 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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