

## Neuroendocrine and neurotransmitter correlates in children with antisocial behavior

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### Abstract

When antisocial behavior becomes a persistent pattern that affects diverse domains of children's functioning, psychiatrists refer to oppositional defiant disorder (ODD) or conduct disorder (CD). The term disruptive behavior disorder (DBD) covers both ODD and CD. Research shows that in the absence of effective interventions, the prognosis for DBD children is relatively unfavorable: their disorder can extend into adolescence, manifest itself in delinquency, and convert into other psychiatric symptoms, such as addiction or personality disorders. Although environmental factors have traditionally attracted most attention in explaining the origin and persistence of DBDs, it is important not to overlook the vulnerability of the child in the development of antisocial behavior. Relatively few studies have been conducted on the neurobiological factors involved in the development of DBDs in children. In this paper, we explain how problems in hypothalamic–pituitary–adrenal (HPA) axis and serotonergic system functioning could be important factors in the behavioral problems of DBD children. Low fear of punishment and physiological underactivity may predispose antisocial individuals to seek out stimulation or take risks and may explain poor (social) conditioning and socialization. Findings consistent with this hypothesis are presented. Finally, we explain how stress in general, and adverse early life experiences in particular, could have an impact on the development of the HPA and serotonergic systems. An investigation of the neurobiological factors involved in antisocial behavior disorder might ultimately guide the development of new forms of intervention.

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### Introduction

Antisocial behavior in children can be operationalized in different ways. Antisocial behavior can be defined in terms of psychiatric diagnoses (oppositional defiant disorder (ODD), conduct disorder (CD), or disruptive behavior disorder (DBD); [American Psychiatric Association, 1994](#)), in terms of the violation of social or legal norms (delinquency, criminality), or as aggressive behavior ([Plomin et al., 1990](#)). These operationalizations are related but not synonymous.

Aggression, defined as behavior deliberately aimed at inflicting physical and/or psychological damage on persons or property, represents a problem of significant clinical and social

concern. In psychiatry, aggression does not constitute a separate diagnostic entity but appears in several psychopathological conditions, the most important being CD, substance use disorder, neurological disorders involving the frontal and temporal lobes, and personality disorders such as borderline and antisocial personality disorder ([American Psychiatric Association DSM-IV, 1994](#)).

The term disruptive behavior disorder (DBD) covers both ODD and CD. The prevalence of these disorders is relatively high: 2% for CD and 3.2% for ODD ([Lahey et al., 1999](#)). The problem behavior of DBD children is often quite stable and persistent ([Offord et al., 1992](#)). Conduct problems in childhood are associated with a host of negative outcomes in adulthood; they predict not only future antisocial behavior ([Fombonne et al., 2001](#)), but also substance abuse and dependence in adulthood ([Kazdin, 1995](#)), early pregnancy in antisocial girls ([Bardone et al., 1998](#)), persistent health problems ([Bardone et al., 1998](#)), and depression.

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Although the short-term effectiveness of intervention strategies (e.g., parent management training, cognitive behavioral therapy) has been demonstrated (Kazdin, 2001), the long-term effectiveness of treatment appears to be limited (Offord and Bennett, 1994). In particular, the high persistence, poor prognosis and limited effectiveness of current treatments of childhood antisocial behavior lend importance to the investigation of the biological correlates of antisocial behavior in childhood. An understanding of these factors in antisocial children should generate hypotheses concerning the underlying neurobiological mechanisms and etiology of antisocial behavior.

It is known that both child-specific and environmental factors contribute to the development and maintenance of antisocial behavior. Most interest in research has focused on environmental factors. It is, for example, known that stress in the family (as a consequence of adverse life events), relationship problems between the parents, and depressive symptoms in the mother play a role in this type of problem behavior (Conger et al., 1994). These factors are likely to result in the affective neglect of the child (Erel and Burman, 1995). However, there is an increasing body of evidence that the child him- or herself also plays an important role. The concept of ‘vulnerability’ indicates that certain children have an increased risk of developing psychiatric disorders. This natural disposition is presumably partly biologically determined.

### The development and long-term outcome of antisocial children

DBD children have an increased risk of showing violently aggressive and other forms of criminal behavior in adolescence and adulthood. The percentages found in the various studies range between 35 and 75%. In other words, early-onset antisocial behavior is an important predictor of chronic and increasingly more serious forms of violent behavior (Loeber and Hay, 1997).

The development of antisocial and violent behavior should be seen as the outcome of a complicated interplay of individual (including biological), developmental, and social factors. Some social factors play a more or less important role as a function of the age of the child. For example, harsh parental discipline with cruel punishment plays a causal role in the development of antisocial behavior in childhood, whereas the absence of parental supervision is an important factor in late childhood or adolescence (Lahey et al., 1999).

If we consider young children it is most likely that the origin of antisocial behavior lies in a combination of a toddler/child with a difficult temperament and a non-optimal environment in which ineffective socialization plays a key role: a difficult child elicits harsh, inconsistent and negative socialization behaviors, as a result of which a difficult temperament ultimately develops into antisocial behavior (Lahey et al., 1995). However, although there are factors that contribute to antisocial behavior in childhood becoming chronic, not all antisocial children become antisocial adolescents, and not all antisocial adolescents become antisocial adults (Lynam, 1996; Robins, 1966). Research suggests that a number of different biological factors may be

involved in antisocial behavior, and that these factors could play a role in the development and maintenance of antisocial behavior over time. In the following sections, we will review the evidence that stress hormones (e.g., cortisol) and neurotransmitters (e.g., serotonin) are involved in childhood-onset antisocial behavior.

### The stress system

There are clear indications that stress plays an important role in explaining individual differences in antisocial behavior. The systems that are involved in the regulation of stress are the neuroendocrine hypothalamic–pituitary–adrenal (HPA) axis, and the psychophysiological autonomic nervous system (ANS). Stress reactions are measured, for example, by assessing cortisol (in blood, urine or saliva) or by measuring changes in heart rate, skin conductance or systolic blood pressure. The starting point of research on the relationship between stress and antisocial behavior is that aggressive individuals are less sensitive to stress. This can be deduced from the fact that these individuals are more often engaged in risky, stressful or dangerous situations than other people. If this is true, there are two possible explanations for a relationship between lower stress sensitivity and antisocial behavior. One theory claims that antisocial individuals have low levels of fear (Raine, 1996). A relative lack of fear would lead to antisocial or delinquent behavior because one is insensitive to the negative consequences of one’s own or other people’s behavior in general and the experience of receiving punishment in particular. If this is the case, the implications for the treatment of these problem behaviors are clear. It would mean that DBD children have problems in conditioning: therefore pointing out the negative consequences of their behavior, or punishing unacceptable behavior are both likely to have little or no effect.

A different theory involving stress focuses on sensation seeking (Zuckerman, 1979). Here it is argued that a certain level of stress is needed in order to feel pleasant and that too little or too much stress is experienced as aversive. Aggressive individuals are considered to have an elevated threshold for stress: they are easily bored and are not put off by situations that the average person finds too exciting, stressful or dangerous.

What evidence is there that a dysfunctional HPA or ANS system plays a role in children’s antisocial behavior? The activity of the ANS seems to be lower in aggressive and antisocial individuals. A British study (Raine et al., 1990) measured heart rate and skin conductance in 100 15-year-old boys. When the boys were about 24 years old, the researchers established who had committed a crime in the intervening 9-year period. The heart rate and skin conductance values of adolescents who had been convicted for committing a crime were lower than those of boys who had not committed a crime. Heart rate measured in toddlers also seems to have a predictive value for aggressive behavior in later childhood (Raine et al., 1997). Apart from this relation between lower stress levels and antisocial behavior, the reverse relationship has also been found: Children with higher stress sensitivity are more fearful. Within the domain of antisocial behavior this is an important finding.

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