The Role of Neurobiological Deficits in Childhood Antisocial Behavior

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ABSTRACT—Childhood-onset antisocial behavior is an important predictor of chronic and serious forms of antisocial behavior in later life. Both biological and social factors are involved in the development of abnormal behavior. We examine the underlying role of stress-response systems in the link between early social adversity and juvenile antisocial behavior, and propose that children with genetically and/or perinatally based neurobiological deficits have problems in activating these systems and therefore experience difficulties in regulating affect and behavior. Underactivity or attenuated reactivity of the stress-response systems may predispose antisocial individuals to seek out stimulation or take risks, and thereby explain deficits in learning and socialization. Further investigations of neurobiological functioning in antisocial children might not only indicate which children are more likely to persist in behaving antisocially but also guide the development of new interventions.

KEYWORDS—antisocial behavior; stress; cortisol; autonomic arousal; children

Antisocial behavior is a significant social and clinical concern. Every year, more than 1.6 million people are killed as a result of violence, and many more suffer from physical or mental health problems stemming from violence (World Health Organization, 2002). Antisocial behavior committed by youths is an issue of particular concern. A recent survey showed that citizens of European nations see themselves as having “significant” difficulties with antisocial behavior, and that the problem is above all associated with people under 25 years of age (“Bad behaviour,” 2006).

The term antisocial behavior refers to the fact that people who are on the receiving end of the behavior are disadvantaged by it, and that social norms and values are violated. Not only aggression but also activities such as theft, vandalism, lying, truancy, running away from home, and oppositional behaviors are involved.

Most normally developing children will occasionally exhibit negative and disobedient behavior toward adults and engage in lying, fighting, and bullying other children. When antisocial behavior forms a pattern that goes beyond the “normal” realm and starts to have adverse effects on the child’s functioning, psychiatrists tend to make a diagnosis of conduct disorder (CD) or oppositional defiant disorder (ODD; American Psychiatric Association, 1994). These disorders are relatively common in children, with estimated prevalences ranging from 5 to 10%. The extent to which these disorders can be treated via therapy is limited, and, as a result, these children are at risk for a host of negative outcomes in adolescence and adulthood, including dropping out of school, criminality, unemployment, dependence on welfare, and substance abuse (Hill & Maughan, 2001).

There is a growing consensus that both child-specific (i.e., genetic, temperamental) and social (e.g., early adversity) factors contribute to the development and maintenance of antisocial behavior, although most research has focused on identifying specific contextual factors that impinge on the developing child. For example, negative life events, family stress, and parental relationship problems have been associated with antisocial-behavior problems in children. However, there is increasing evidence that factors organic to individual children exacerbate the risk of antisocial behavior to those who live with social adversity. Here, we review evidence relating to the role of neurobiological factors in accounting for the link between early adversity and childhood antisocial behavior and propose that consideration of biological factors underlying this stress–distress link significantly advances understanding of the mechanisms explaining individual differences in the etiology of antisocial behavior.
Research suggests that neurobiological deficits related to the functioning of the stress systems in children with CD are linked to antisocial behavior. We argue that familial factors (e.g., genetic influences, early adversity) are linked to negative outcomes through the mediating and transactional interplay with neurobiological deficits (see Fig. 1) and propose that stress hyporeactivity is an index of persistent and serious antisocial behavior.

**STRESS-RESPONSE SYSTEMS**

There are clear indications that stress plays an important role in explaining individual differences in antisocial behavior. The systems involved in the regulation of stress are the neuroendocrine hypothalamic-pituitary-adrenal (HPA) axis and the psychophysiological autonomic nervous system (ANS). Cortisol is studied in relation to HPA-axis activation, and heart rate (HR) and skin-conductance (SC) responses are used as markers of ANS (re)activity.

The starting point of our approach is that antisocial individuals are less sensitive to stress. This can be deduced from the fact that antisocial individuals engage in risky or dangerous behavior more often than other people do and seem less deterred by its possible negative consequences. There are two explanations for the proposed relationship between lower stress sensitivity and antisocial behavior. One theory claims that antisocial individuals are fearless (Raine, 1996). A lack of fear leads to antisocial behavior because individuals are less sensitive to the negative consequences of their own or other people’s behavior in general and to the receipt of punishment in particular. The implications for treatment are clear: Antisocial individuals will have problems learning the association between behavior and punishment, such that pointing out the negative consequences of behavior, or punishing unacceptable behavior, is likely to have little or no effect.

The second explanation focuses on stress thresholds and sensation-seeking behavior (Zuckerman, 1979), and argues that antisocial individuals have elevated thresholds for stress. They are more easily bored and less easily put off by situations that normal people find stressful or dangerous.

What evidence is there that dysfunctional stress systems play a role in antisocial behavior? Several studies (e.g., Virkkunen, 1985) have found that antisocial adults have low resting levels of cortisol, SC, and HR. There is also evidence of inverse relationships between these physiological variables and the severity of the behavioral problems shown. Studies investigating the relation between biological stress parameters and antisocial behavior have also been performed in children (e.g., van Goozen et al., 1998), and the predicted (inverse) relations have been found.

Stress variables can also predict antisocial behavior over time. Raine, Venables, and Mednick (1997) measured HR in more than 1,700 three-year-old children. Aggressive behavior was assessed at age 11. Raine et al. found that low resting HR at age 3 predicted aggressive behavior at age 11. In a study of criminals’ sons (who are at risk of becoming delinquent), Brennan et al. (1997) found that boys who did not become delinquent had higher HR and SC than did boys who became delinquent. The

**Fig. 1.** Theoretical model relating early social adversity to later antisocial behavior problems. It is hypothesized that this relationship is explained by the underlying mediating and moderating role of neurobiological factors. The dashed-dotted lines emanating from genetic susceptibility to neurobiological deficits and from neurobiological deficits to antisocial behavior problems represent an indirect (or mediating) pathway between these factors. The bold line emanating from genetic susceptibility to the pathway linking early social adversity to neurobiological deficits, and the dashed-dotted line from neurobiological deficits to the pathway linking early social adversity to antisocial behavior problems, represent proposed moderating influences from each source variable (i.e., genetic susceptibility and neurobiological deficits). A moderating influence is the equivalent of statistical interaction between two theoretical constructs. Bold and dashed-dotted lines in all other instances represent direct and indirect pathways linking primary theoretical constructs. For a full exposition of this model, see van Goozen, Fairchild, Snoek, and Harold (2007).
authors concluded that the boys in the former group were biologically protected by their heightened autonomic responsivity.

Studies of youths who engage in antisocial behavior show that they, like antisocial adults, have less reactive stress systems than do youths who do not engage in antisocial behavior. The question is whether the same applies to children with serious antisocial behavior who have been diagnosed with CD or ODD.

**STRESS STUDIES IN CD CHILDREN**

Most studies collect stress data under resting conditions rather than during stress exposure. Antisocial individuals might be different from normal individuals in two respects: A low resting stress level could result in failing to avoid, or even approaching, stressful situations; and low stress reactivity implies that one is more fearless and cares less about possible negative consequences.

Our studies use a paradigm in which psychosocial stress is evoked by exposing children to frustration, provocation, and competition (e.g., van Goozen et al., 1998). The participant competes against a fictitious video taped “opponent” who behaves in an antagonistic manner. The participant and opponent perform computerized tasks on which they can earn points. The participant is told that the person who earns the most points will receive an attractive prize. Some tasks are impossible to complete, which induces frustration. HR and SC are measured continuously, and cortisol is collected repeatedly in saliva.

CD children show lower HR, SC, and cortisol reactivity to stress than do normal children. Although CD children appear to be less affected at a biological level, they react more angrily and aggressively to provocation than do non-CD children and report feeling quite upset. It is known that CD children are impulsive, have hostile appraisal patterns, and engage in conflictual situations. It is striking that this pattern of appraisal and behavior is not accompanied by contextually appropriate somatic changes.

Genetic factors likely play a role in the functioning of the HPA axis and ANS. There is also evidence that stressful events—by which we mean serious stressors like neglect and traumatization—play an important role in “programming” the stress systems, particularly the HPA axis. This evidence comes mainly from nonhuman animal studies, but the neurobiological consequences of the types of severe stress that can be manipulated in animal studies also occur in humans.

**EARLY EXPERIENCE AND FAMILY ADVERSITY**

Physical and biological problems during important phases in development (e.g., birth complications, stress or illness during pregnancy), together with early adversity (e.g., malnutrition, neglect, abuse), contribute importantly to the development of personality and psychopathology. There is increasing evidence that interactions between biological and environmental factors affect the developing brain (Huizink, Mulder, & Buitelaar, 2004).

Nonhuman animal studies show that stressors in early life can have permanent effects on the functioning of the HPA axis, resulting in altered basal and stress-reactivity levels. For example, Liu et al. (1997) varied the amount of licking and grooming behavior in mothers of newborn rats. In adulthood, offspring who had been exposed to normal maternal care were more capable of handling stress than were rats that had received less care. The former also expressed more stress-hormone receptors in the hippocampus, an area important for stress regulation, than did rats that had received less care. Thus, maternal behavior had a direct and lasting effect on the development of the stress systems of the offspring.

Such conclusions are based on data from nonhuman animals, and for obvious reasons it is difficult to conduct similar studies on humans. However, evidence from a handful of studies involving institutionalized children suggests that the processes at work are similar (Carlson & Earls, 1997; Gunnar, Morison, Chisolm, & Schuder, 2001).

Antisocial children are more likely to come from adverse rearing environments involving atypical caregiver–child interactions (Rutter & Silberg, 2002). It is known that CD children are more likely to experience compromised pre- or perinatal development due to maternal smoking, poor nutrition, or exposure to alcohol and/or drugs. It is possible that these factors have affected such children’s stress-response systems and resulted in children with a difficult temperament.

**STRESS HYPOREACTIVITY AS A MEDIATING FACTOR**

We have suggested that physiological hyporeactivity may reflect an inability to generate visceral signals to guide behavior and, in particular, to regulate anger and reactive aggression (van Goozen, Fairchild, Snoek, & Harold, 2007). Evidence from nonhuman animals indicates that abolishing the hormonal response to stress may impair processing of social signals and lead to abnormal patterns of aggression (Haller, Halász, Mikics, & Kruk, 2004). These studies also showed that abnormal aggressive behavior can be prevented by mimicking the hormonal response normally seen during aggressive encounters. These findings have clear parallels with abnormal aggression in humans, in the sense that the behavior is not only excessive but also often risky, badly judged, and callous.

We have proposed an integrative theoretical model linking genetic factors, early adversity, cognitive and neurobiological regulatory mechanisms, and childhood antisocial behavior (van Goozen et al., 2007; see Fig. 1). Interactions between genetic predispositions and the environment in which they are expressed appear to be crucial in the etiology of antisocial behavior problems. A genetic predisposition toward antisocial behavior may be expressed in adverse rearing environments in which the child receives harsh or inconsistent discipline or is
exposed to high levels of interparental conflict or marital breakdown (Moffitt, 2005). It is likely that the origin of antisocial behavior in young children lies in this combination of a difficult temperament and a harsh environment in which there is ineffective socialization: A difficult child elicits harsh, inconsistent, and negative socialization behaviors, as a result of which a difficult temperament develops into antisocial behavior (Lykken, 1995). Conversely, the effects of a genetic predisposition may be minimized if the child is raised in an environment in which the parents express warmth or adopt a consistent, authoritative parenting style.

Some children are born with a more easygoing temperament than others. In cases of “hard-to-manage” children, a child’s genotype can evoke negative behavior from the environment because genetic influences lead the individual to create, seek out, or otherwise end up in environments that match the genotype (Rutter & Silberg, 2002). These active, evocative gene–environment processes are extremely important in understanding the development and continuity of antisocial behavior (Moffitt, 2005). Social factors occurring independently of the child’s genetic makeup or temperament can serve as contributory factors (Harold, Aitken, & Shelton, 2008).

We noted above that early brain development is vulnerable to the effects of environmental stress (Huizink et al., 2004), and that CD children are likely to have been exposed to early stress. A down-regulation of the stress-response system in the face of chronic stress in early life would be an adaptive mechanism, avoiding chronic arousal and excessive energy expenditure that could ultimately result in serious pathophysiological consequences. Given what we know about the background of CD children, it is plausible that these processes have occurred.

We propose that physiological hyporeactivity is a mediating and/or moderating factor for persistent and severe antisocial behavior and that the effects of variations in genetic makeup and early adversity on childhood antisocial behavior occur via this deficit. The primary pathway by which familial factors are linked to antisocial outcome is the reciprocal interplay with neurobiological deficits and resulting disinhibited cognitive (e.g., impulsivity, hostile bias) and emotional (e.g., increased anger) processing, with the latter serving as the psychological gateway through which neurobiological deficits find their expression in antisocial behavior.

CONCLUSION

Antisocial behavior in children can be persistent and difficult to treat. Although behavioral interventions have been shown to be effective in milder forms of problem behavior, they have limited effectiveness in more seriously disturbed children (Hill & Maughan, 2001).

At present, we do not know what causes the pattern of neurobiological impairments observed in antisocial children, although it is clear that genetic factors are involved (Caspi et al., 2002). An important line of research suggests that psychosocial adversity affects brain development. Knowing that many CD children have problematic backgrounds, it seems possible that exposure to severe stress has had an effect on the development of their stress systems. Longitudinal research in high-risk children is needed to shed more light on this issue.

Future interventions and treatments should benefit from a neurobiological approach: Neurobiological assessment of high-risk children could indicate whether their deficits are such that interventions involving “empathy induction” or “learning from punishment,” for example, are unlikely to work. In such cases, pharmacological interventions could be considered as a treatment option. An important line of future research is to establish whether CD children with attenuated stress (reactivity would be more effectively treated by using pharmacological therapies that reinstate normal HPA-axis functioning.

Current interventions for childhood antisocial behavior have limited success because we lack knowledge of the cognitive–emotional problems of these children and their neurobiological bases. We also fail to assess the environmental risk factors that affect individual neurodevelopment. Furthermore, available treatment options do not target the individual’s specific neurobiological vulnerabilities. It seems prudent to identify subgroups of children in whom different causal processes initiate and maintain behavioral problems. This should result in a better match between patient and treatment.

A final point is that the understandable tendency to focus on persistence of antisocial behavior runs the risk of overlooking the fact that a substantial proportion of antisocial children do not grow up to be antisocial adults (with prevalence rates for antisocial children who persist into adulthood ranging from 35 to 75%). Neurobiological factors could also account for this: Promising data from a handful of studies show that neurobiological factors differ between children who persist in and desist from antisocial behavior (Brennan et al., 1997; van de Wiel, van Goozen, Matthys, Snoek, & van Engeland, 2004). Expanding on this research base is essential if we are to reach a more adequate understanding of the causes, course, and consequences of childhood antisocial behavior and, most importantly, devise effective ways of reducing the negative consequences for society.

Recommended Reading


neurobiological basis of antisocial behavior in greater detail than
the current paper.

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