The Role of Early Emotion Impairments
in the Development of Persistent Antisocial Behavior

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Abstract

Antisocial behavior that begins in childhood predicts chronic and serious antisocial behavior in adulthood[AU: Is this right? yes]. Antisocial children are impaired in how they perceive, experience, and regulate emotion, particularly fear and sadness, and the neurobiological systems that process aversive emotional information, particularly the stress response systems in the paralimbic system, are compromised. As a result, children may have a difficult temperament, impaired cognitive abilities, or different levels of emotional reactivity. These characteristics, in turn, increase the risk that children have poor social relationships and make decisions that increase the likelihood that their antisocial behavior becomes stable and pervasive. In this article, I clarify the role of emotion impairments in children's antisocial behavior. I also argue that investigating these emotion functions can help identify which children are more likely to persist in behaving antisocially and guide the development of new interventions.

Most individuals behave aggressively or antisocially from time to time. Normally developing children occasionally disobey adults, tell lies, fight, and intimidate other children. However, when antisocial behavior goes beyond occasional occurrences and adversely affects a child's functioning, psychiatrists diagnose conduct disorder (CD) or oppositional defiant disorder (ODD; 1). Conduct disorders are the most common mental health disorder in children and young people. Among children and youth in Britain in 1999 and 2004, the prevalence of conduct disorders and associated impairment was 5% (2).

Early aggression is a risk factor for the persistence of antisocial behavior through childhood (3). The issue of why, when, and how antisocial behavior persists has fascinated researchers since Robins (4) pointed out that antisocial behavior is remarkably stable across time and circumstances for some people and decidedly unstable for others. In a seminal article, Moffit (5) argued that persistent antisocial behavior starts in childhood, originating in an interaction between a child's biological vulnerabilities and a criminogenic environment. Since Moffit's article, we have learned that children who develop persistent antisocial behavior are impaired in biologically based emotion functions, and that these impairments are linked to antisocial behavior that begins in childhood and is sustained for lengthy periods (6, 7). Deficits in these emotional functions may result in children having a difficult temperament (with high negative emotionality or poor inhibitory control), impaired cognitive abilities (e.g., poor attention, inhibition, decision making, and planning), as well as having problems with emotional reactivity and self-regulation (8). Together, these characteristics increase the risk that antisocial children not only have poor social relationships, but also make decisions that increase the likelihood of their behavior becoming stable and pervasive.

In considering the causal role of individually based neurobiological impairments that are linked to impaired emotion function, we must acknowledge the role of the social environment. Early violence breeds more violence, and the contagious nature of aggressive

behavior means that, for example, victims of bullying may become aggressors and group-based intervention programs may boost rates of aggression (9). Children with early emotional problems are also found disproportionally in disadvantaged and less supportive environments, and some of these characteristics are shared by the children and their parents, explaining the stability of antisocial behavior. Moreover, the behavior of these children can evoke a transactional process of problematic parent-child interactions that promote and maintain antisocial development over time (10). Although I focus in this article on individual-level influences on antisocial development, we must recognize that these influences combine with family-level influences (e.g., exposure to harsh parenting, interparental conflict, and maltreatment) to understand more fully how persistent and pervasive antisocial behavior develops.

In this article, I review the neurobiological systems that play a role in processing negative emotions. I demonstrate that problems in these systems are largely responsible for the severity and persistence of the behavioral problems observed in antisocial children.

Impaired Functioning of the Stress Systems

Systems Involved in Processing and Regulating Stress

Individual differences in aggression emerge in late infancy (11). Dispositional factors not only contribute to its development and maintenance, but also explain or accentuate risk to children who live with early social adversity (12). Impairments in processing and experiencing negative emotions, which are related to the functioning of the stress systems, are particularly important in antisocial behavior that starts early.

Normal variations in stress system (re-)activity play a role in temperamental differences in children, with higher activity linked to shy, inhibited, and fearful temperament, and low activity to more impulsive, fearless, and aggressive temperament. The systems involved in

processing and regulating stress in general, and negative emotions in particular, are the neuroendocrine hypothalamic-pituitary-adrenal (HPA) axis and the autonomic nervous system (ANS).

The autonomic nervous system. The ANS, through its sympathetic and parasympathetic branches, regulates critical functions on a moment-to-moment basis and governs the fight-or-flight reaction. In physiological terms, the parasympathetic nervous system conserves and restores energy, whereas the sympathetic nervous system enables the body to prepare for flight or fight *(7, 13)*.

The HPA axis. The other major system involved in regulating stress is the HPA axis. When stress is perceived, the hypothalamus releases corticotropin-releasing hormone (CRH), stimulating the pituitary to secrete adrenocorticotropic hormone (ACTH). These events, in turn, promote cortisol release from the adrenal gland, and centrally cause behavioral activation and intense arousal (14). Cortisol, via negative feedback inhibition on the hypothalamus, pituitary, and other brain structures (hippocampus), suppresses the HPA axis, leading to restored basal cortisol levels. [AU: For parallel structure with the prior paragraph, please add a sentence citing work on the correlates of HPA activity that relate to antisocial behavior. Or we could drop the sentence about the correlates of ANS activity (that work is described in greater detail in the section on stress impairments in aggressive children). Which would you prefer? I would drop the sentence about ANS correlates here]

Theories of Impaired Stress (Re-)Activity

Antisocial individuals not only experience negative emotions less intensely, they also seem to have an elevated threshold for negative events. We know this because antisocial people place themselves in risky, stressful, or dangerous situations more frequently than other people. Understanding the neurobiological pathways and systems associated with

underarousal, fearlessness, and reduced response to distress provides clues to how stress affects mood, cognition, and behavior.

Why are reduced physiological arousal and the development of aggression related? According to the stimulation seeking theory (15), low physiological arousal is aversive and motivates people to seek stimulation to raise arousal to a more optimal level. Aggressive acts increase arousal and are therefore rewarding and more likely to be committed. The fearlessness theory (16) focuses on reactions to negative events. It argues that fearless children are more likely to engage in aggression to obtain rewards and social status because they are relatively insensitive to the negative consequences of their actions. The fearlessness account, which focuses on the consequences of aggression, and the stimulation-seeking explanation, which focuses on the antecedents of getting into risky situations, are not incompatible.

Low physiological arousal in young children may also reflect a more general risk factor, because individuals with low arousal have difficulty attending and reacting to environmental stimulation (17). The prefrontal cortex allocates attention, regulates emotion, and reacts to stress, and damage to this region may lead to psychophysiological abnormalities that predispose individuals to antisocial behavior.

Stress Impairments in Aggressive Children

Measures indexing low ANS activity have been linked to antisocial, aggressive delinquent, psychopathic, and violent behavior (13). In several studies, low physiological arousal precedes the onset or predicts the persistence of antisocial behavior. In one study (18), low arousal at age 15, measured using cardiovascular and skin conductance activity (SCA), predicted criminal convictions by age 24. In another study (19), low heart rate in typically developing children at age 3 years predicted aggression at age 11.

In yet another study (7), children with CD had low SCA, which predicted persistence of CD into adolescence. Studies of baseline cortisol levels in disruptive children have yielded mixed results, but in a recent longitudinal study (21), 5- to 15-year-olds with antisocial behavior had lower cortisol levels .Low cortisol also predicts persistent aggression (22). In studies on cortisol reactivity following negative emotional challenges (23), children with CD had an impaired stress response.

Researchers have also identified associations between poor fear conditioning in typically developing 3-year-olds and later aggression and criminal behavior (24). And one study (25) identified an even earlier link: 12-month-olds with lower physiological arousal were more physically and verbally aggressive at age 3. These studies of young children suggest not only that objective physiological measures predict later aggression, because reduced biological activity could be associated with deficient emotion regulation and inhibition of aggressive behavior, but also that we can identify children who may be at risk long before problematic behavior is seen.

Neuropsychological Evidence of Emotion Dysfunction

The central tenet of the fearlessness theory is that antisocial individuals are relatively impaired in their perception and experience of fear, and that the neurobiological systems that normally process threat information are compromised structurally or functionally. The amygdala plays a key role in the perception of threat signals; in neuroimaging studies, the amygdala activates when individuals view negative facial expressions or pictures, and neuropsychological studies of people with damage to the amygdala show that these individuals are less able to recognize fear and anger, impaired in fear conditioning, and respond with less startle to acoustic probes (6).

In two studies (26, 27), children with CD or psychopathic tendencies had problems

perceiving facial and vocal expressions of fear, sadness, and empathy. In another study (6), children with CD exhibited impaired fear conditioning, reduced startle amplitudes, and autonomic reactivity to affective pictures; in functional neuroimaging studies (28, 29), children's amygdala, anterior cingulated, and orbitofrontal cortex activation was reduced while processing negatively valenced stimuli; and in structural imaging studies (30, 31), amygdala, anterior insula, and orbitofrontal cortex volumes also were reduced.

Both the prefrontal cortex and amygdala exert considerable control over the stress response systems (32). The amygdala has an excitatory influence on the paraventricular nucleus of the hypothalamus that drives the HPA axis, while the prefrontal cortex inhibits activation of the HPA axis. Deficits in the functioning of the circuitry that encompasses the prefrontal cortex–anterior cingulate–amygdala could underlie the problems in the functioning of the stress response system that are observed in antisocial individuals. Alternatively, low reactivity to distress could be due to changes in functioning as a result of (epi-)genetic factors.

Taken together, these studies show that children with severe antisocial behavior have emotional problems and that these problems have a neurobiological basis. Deficits in the function of the amygdala and closely connected systems cause problems in processing distress, rendering individuals relatively fearless and unemotional and making it difficult for them to recognize cues from the environment that signal threat or submission.

Neurobiological Effects of Early Adversity

How do these emotion impairments develop in children? Some environmental risk factors that affect children's neurobiological development and functioning are prenatal (e.g., exposure to mothers' smoking in utero, maternal psychopathology), while others are postnatal (e.g., maltreatment or adversity in the form of parental neglect, interparental

violence, and inconsistent discipline; 33). Some of the environmental risk factors most likely to affect the biological stress systems involved in psychopathology are variations in the quality of parents' care and psychopathology, poverty, and child abuse and neglect (9).

In studies of nonhuman primates and rodents, early events have long-term neurobiological consequences, including effects on the HPA axis, which persist into adulthood (13). Variations in maternal care affect the development of individual differences in neuroendocrine responses to stress so maternal behavior programs children's HPA responses to stress (34). In the few studies on the effects of early stress on neurobiological systems conducted in clinical groups (34), children with a history of abuse had long-term changes in the functioning of the HPA axis, findings that are consistent with studies of animals. Although the quality of mothers' care in humans predicts children's self-regulation, we do not yet know precisely which aspects of maternal behavior determine the unfolding of infants' stress responses or how these physiological responses map onto behavioral adjustment. And although researchers are interested in the effect of early exposure to risk on variations in DNA methylation (35), epigenetic processes and the effects on neural development and problem behavior have only been studied in animals.

Returning to the development of antisocial behavior, conduct disorders have a steep social class gradient, with a three- to fourfold increase in prevalence in the lowest social [AU: socioeconomic? Yes!] classes. Almost 40% of children between 5 and 17 years who are in care[AU: child care? foster care? Foster care], who have been abused, or who are listed on child protection/safeguarding registers have conduct disorder (2).

Knowing that children with CD are more likely to come from adverse rearing environments that involve atypical caregiver-child interactions, parents' psychopathology, or compromised pre- or perinatal development, we may surmise that early adverse influences have lasting effects on developing neurobiological systems in the brain, including systems

important in processing and regulating emotions (HPA axis, prefrontal cortex, neurotransmitter systems). These effects, in turn, may result in more difficult temperament in some children. Exposure to childhood adversity can also disrupt brain development by inhibiting the expression of genes (i.e., epigenetic mechanisms) or amplifying pre-existing vulnerabilities (36). Such an interpretation suggests that positive environments can exert protective effects. For these reasons, interventions when children are young are likely to be the most successful in achieving lasting change through their enduring effects on the developing neurobiological systems involved in emotion and cognition (37). For example, early malnutrition is linked to externalizing behavior problems; if malnutrition harms those brain areas involved in emotion regulation and impulsivity, providing at-risk families with more optimal nutrition might prevent problem behaviors (38).

Conclusion and Looking Ahead

In studies of children with antisocial behavior, the functioning of the paralimbic system, which includes the prefrontal cortex–anterior cingulate–amygdala circuitry, is impaired. As a result of the reduced (re-)activity of these systems, children's abilities to process and experience negative emotions are compromised, and they are less able to regulate emotion and inhibit aggression. Genetic factors could predispose children to more severe and persistent antisocial behavior, either by directly influencing the development of different neurobiological systems or in conjunction with early exposure to adversity (6, 34). An early deficit in activity of the stress systems is particularly crucial in this developmental sequence because it results in individuals becoming emotionally detached from their actions and unable to learn from what others might experience as negative events (e.g., punishment). If the physiological response to distress acts as a warning to restrain ongoing behavior in situations

of psychological or physical danger (39), children who fail to activate these systems are likely to behave in a more disinhibited fashion.

Investigating neurobiological functioning in antisocial children can identify which children are most likely to persist in engaging in severe antisocial behavior and guide the development of interventions. Assessing high-risk children neurobiologically could determine whether their impairments may reduce the success of certain psychosocial interventions (e.g., those involving empathy induction or learning from punishment) because these interventions rely on types of processing that are difficult for these children. In those instances, pharmacological interventions could be considered as a precursor or adjunct to traditional psychological forms of treatment. For example, in an individual who fails to recognize distress in others, oxytocin could promote positive social behavior through enhanced social attention and improved emotion recognition (40).

The success of current interventions for antisocial behavior in childhood is limited because we neither consider nor target the neurobiological bases of children's cognitive-emotional problems. We should identify subgroups of children with distinct neurobiological profiles early in life. Opportunities for changing behavior are greatest in the early years because the brain is more plastic at that time. Identifying precursors of disorder in the context of typical development can inform prevention programs and result in a more effective match between patient and treatment, thereby reducing the psychological and economic costs to society of antisocial behavior.

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