# Childhood antisocial behavior: A neurodevelopmental problem

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**Abstract**

Early onset disruptive, aggressive and antisocial behavior is persistent, can become increasingly serious as children grow older, and is difficult to change. In 2007, we (Van Goozen et al., 2007) proposed a theoretical model highlighting the interplay between neurobiological deficits and cognitive and emotional functioning as mediators of the link between genetic influences and early social adversity, on the one hand, and antisocial behavior problems in childhood, on the other. In the current paper we review the post-2007 evidence relevant to this model. We discuss research on genetics/epigenetics, stress/arousal-regulation, and emotion and executive functioning in support of the argument that antisocial children, especially those who persist in engaging in antisocial behavior as they grow older, have a range of neuropsychological characteristics that are important in explaining individual differences in the severity and persistence of antisocial behavior. Current clinical practice tends not to acknowledge these individual neuropsychological risks factors or to target them for intervention. We argue that aggressive and disruptive behavior in childhood should be regarded as a neurodevelopmental problem and that intervening at the level of mediating neuropsychological processes represents a promising way forward in tackling these serious behavioral problems.

**Introduction**

Children who exhibit aggressive and disruptive behavior from an early age are likely to persist and develop increasingly serious forms of antisocial behavior (ASB; Lahey, Loeber, Burke & Applegate, 2005). ASB established in childhood is associated with problematic relationships and poor educational outcomes, which lead to other negative outcomes later in life, such as psychiatric illness, poor physical health and substance use problems (Fergusson, Horwood & Ridder, 2005), as well as violent relationships and erratic employment patterns (Scott, Knapp, Henderson, & Maughan, 2001).

ASB is an umbrella term, covering different clinical diagnoses, judicial terms and a wide range of behaviors, meaning that antisocial individuals represent a heterogeneous group of people (Skeem, Scott & Mulvey, 2014; Stadler, Poustka, & Sterzer, 2010). Despite its numerous manifestations and heterogeneous nature, impairments in emotional and cognitive functioning have consistently been found in many antisocial populations; these impairments are thought to play a causal role in the development and maintenance of ASB (Van Goozen et al., 2007).

Evidence has been accumulating showing that children who go on to develop persistent antisocial behavior have deficits in neuropsychologically based emotion functions, and that these impairments are linked to antisocial behavior that begins in childhood and is sustained over lengthy periods of time (Fairchild, Van Goozen, Calder, & Goodyer, 2013; Moffitt 1993). Deficits in emotion functioning (e.g., in emotion recognition or empathy) can result in children having problems with emotional reactivity and self-regulation (Eisenberg et al., 2009; Van Goozen, 2015) and impaired cognitive abilities (such as poor attention, inhibition, decision making and planning), especially under emotionally arousing conditions (Thapar & Van Goozen, 2018; Zelazo, 2020). Together, these characteristics increase the risk that antisocial children not only have poor social relationships, but also make decisions that increase the likelihood that their behavior becomes stable and pervasive.

**Heterogeneity and transdiagnostic overlap**

ASB and conduct problems manifest in different ways and these different manifestations are associated with distinct etiologies (Hudziak, Achenbach, Althoff & Pine, 2007). Various attempts to reduce the heterogeneity of ASB and to differentiate between different subtypes of ASB have all employed a categorical approach. Examples include *early* versus *late age of onset*; *low* versus *high callous-unemotional* (CU) traits; *instrumental* versus *reactive aggression*; and *aggressive ASB* versus *rule-breaking ASB*. However, research suggests that antisocial individuals differ in degree rather than kind (Skeem, Scott & Mulvey, 2014; Van Goozen et al., 2007) and that dimensional rather than categorical approaches are needed to understand ASB and develop treatments and interventions (Cuthburt & Insel, 2013; Hudziak et al., 2007).

Seriousness of ASB, regardless of age of onset, is associated with neural impairments and significant psychosocial adversity (Fairchild et al., 2013). Many existing interventions aiming to reduce behavior problems in young children focus on improving parenting skills and/or the child-parent relationship. Whilst the effectiveness of parenting programs and family-based interventions has been demonstrated (Piquero et al., 2016; Scott, 2010), and such interventions are recommended for use (for example in the UK by the National Institute for Health and Care Excellence; NICE, 2017), a significant subgroup of parents will not engage with these interventions. Furthermore, individual differences in children's emotional or cognitive functioning might moderate the effectiveness of such interventions, and these factors are not routinely taken into account.

For these reasons we have argued that interventions should focus on the specific problems and needs of the individual child (Hunnikin & Van Goozen, 2018). Intervention can be done more easily in younger children, at a time when they are developing the emotional and cognitive skills that are crucial for healthy, appropriate and supportive interpersonal functioning (Herba et al., 2006; Skeem et al., 2014). A focus on the identification of the underlying emotional and cognitive processes that influence the development and severity of ASB and that are shared across diagnoses can lay the groundwork for the development of personalized and targeted intervention and treatment options that are feasible, effective and resource efficient (Marchette & Weisz, 2017).

**Antisocial children: A neurodevelopmental problem**

Most individuals engage in aggressive or antisocial behavior from time to time. Typically developing children occasionally disobey adults, tell lies, fight, and intimidate other children. However, when antisocial behavior extends beyond occasional occurrences and has significant adverse effects on a child's functioning and development, and when the abnormalities or impairments can be linked to maldevelopment of neural tissue that gives rise to emotional, cognitive and behavioral problems, we can speak of a neurodevelopmental disorder (Bishop & Rutter, 2008; Raine, 2018). Indeed, Wakschlag and colleagues (2017) have persuasively argued that early disruptive behavior is a neurodevelopmental disorder.

Building on our 2007 paper, in the present review we summarize evidence that childhood ASB is characterized by disrupted physiology and impaired emotional and cognitive functioning, explaining both the type and the severity of behavioral problems. Although it has been counter-argued that not all children with ASB go on to manifest ASB in adulthood (Fairchild et al., 2013; Tremblay et al., 2004), lack of continuity into adulthood is also true of other established neurodevelopmental disorders, particularly attention-deficit/hyperactivity disorder (ADHD; Moffitt et al. 2015; Van Lieshout et al., 2016); likewise, children who do not engage in persistent antisocial behavior may go on to display dysfunction later in life (Raine, 2018).

The model we proposed in 2007 (see Figure 1) concerns the causal role of individually based neural impairments that are linked to impaired emotion and cognitive function. It sought to explain how individual differences in aggression emerge in late infancy, how family and dispositional factors contribute to the development and maintenance of ASB, and how they attenuate or accentuate risk to children who live with early social adversity (Van Goozen et al., 2007). More specifically, the model links familial factors (e.g., genetic influences, early childhood adversity) to negative behavioral outputs (e.g., antisocial behavior problems) through the interplay between neurobiological deficits (e.g., neurotransmitter; HPA axis, ANS) and cognitive and emotional problems, and proposes that these biological, cognitive and emotional factors jointly mediate the link between early family influences and later behavioral output. Thus, rather than early childhood adversity (e.g., hostile parenting; parental conflict and violence; negative life events) exerting a direct effect on childhood ASB, the model proposes that any effect is mediated by neurobiological and neuropsychological impairments. The relationship between neurobiological and cognitive and emotional functioning is represented as reciprocal in nature, in the sense that initial disruptions in neurobiological functioning facilitate disruptions in cognitive and emotional functioning, which in turn affect further disruption at a neurobiological level. Genetic factors are emphasized in the model as a source of familial influence on children's neurobiological and neuropsychological functioning, with variation in genetic makeup interacting with early childhood adversity to adversely affect neurobiological and neuropsychological development and functioning. The model proposes that these processes unfold over time and that child behavior can also evoke a process of problematic parent-child interactions that promotes and maintains antisocial development over time.

In the present paper we review evidence that has accumulated since this model was published, including evidence that well-developed emotion skills or executive function (EF) skills can serve as a protective factor against the risk associated with early adversity.

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*Understanding mediating processes: Impaired self-regulation*

In order to better understand why children show antisocial and aggressive behaviors and be able to influence their developmental outcome effectively, we have to learn more about the mechanisms that underlie aggression. An important mechanism in the development and treatment of antisocial and aggressive behavior is self-regulation. This is the process by which people (un)consciously act to control emotions, thoughts or behaviors, and also involves processes at a neurobiological level (Heatherton, 2011). Problems in self-regulation are prominent in many forms of psychopathology (Cole & Deater-Deckard, 2009; Nigg, 2017), and aggression is often an extreme behavioral expression of self-regulation failure. Aggressive

behavior in response to frustration is quite common in young children due to insufficient self-regulation at that developmental stage, but most children develop effective regulation of behavior as they grow older (Eisenberg et al., 2009). Conversely, if they do not develop regulation and their aggressive and antisocial behavior becomes pervasive, affecting diverse domains of functioning, this can lead to symptoms that form part of the diagnosis of oppositional defiant disorder (ODD) or conduct disorder (CD) (American Psychiatric Association; APA, 2013). However, these problems are typically defined at a behavioral level, without an analysis of the mechanisms that underlie them.

Self-regulation can be captured by neurobiological, emotional and cognitive parameters that are sensitive in identifying individual differences and specific in explaining individual behavioral problems, and that can be used in the development of interventions (see Figure 1). Negative emotions are important triggers of self-regulation failure (Heatherton, 2011). When an individual encounters a stressor and experiences negative emotions, the autonomic nervous system (ANS) and the hypothalamic-pituitary-adrenal (HPA) axis are automatically activated; these processes are under regulatory control (McEwen, Gray & Nasca, 2015). At a cognitive level, executive functioning (EF) is used to control emotions, thought and behavior. Executive functions subserve emotion regulation and self-regulation. It is known that children with ASB exhibit reduced physiological response to negative events and stress, have impairments in awareness, processing and regulation of emotions, and problems in EF, especially under emotionally arousing condition (i.e., "hot" EF; Zelazo, 2020). Deficient self-regulation leads to difficulties in adapting to changes in the social environment; an inability to inhibit initial responses and resist interference from irrelevant stimuli; and difficulty in persisting with relevant tasks. This manifests in behavioral problems, in the form of temper tantrums, aggression, impulsivity, and anxiety, and in attention deficits and lack of empathy. These behavioral difficulties are evident in diagnostic categories other than ODD/CD that are also associated with difficulty in regulating emotion and behavior, such as ADHD or autism spectrum disorders (ASD) (Anastopoulos et al., 2011; Mazefsky et al., 2013).

**Familial influences: Genetic effects and the interplay between genes and environment**

Although we focus here on individual-level influences on antisocial development, it is important to acknowledge the role played by the social environment. Exposure to early violence breeds more violence, and the contagious nature of aggressive behavior means that, for example, victims of aggression may themselves become aggressors (Dishion, Bullock & Granic, 2002). Children with early emotional problems are disproportionally found in disadvantaged and less supportive environments (characterized by harsh parenting, inter-parental conflict, and maltreatment) and these characteristics are shared between the children and their parents, helping to explain the cross-generational stability of antisocial behavior (Erath, El-Sheikh & Cummings, 2009). Individual-level and family-level influences combine to explain how persistent and pervasive antisocial behavior develops (Van Goozen et al., 2007; Figure 1).

The role of genetic factors in the etiology of ASB has long been recognized (Rhee & Waldman, 2002; Polderman et al., 2015) and genetic factors contribute to the cross-generational stability of ASB. Attempts to identify specific risk variants are underway and have had some success in identifying potential candidate genes that may be associated with ASB, including those in the dopaminergic and serotonergic systems (for reviews, see Holz et al., 2018, or Salvatore & Dick, 2018). Such candidate gene methods have fallen out of favor in the field of identifying genetic risk variants for complex traits, due to the top-down nature of identifying potential candidates and variable replication across studies (Salvatore & Dick, 2018). Instead, the field is moving towards genome-wide, hypothesis-free analyses, such as Genome-wide Association Studies (GWAS). A few GWAS studies of antisocial behavior have been conducted (Salvatore & Dick, 2018; Tielbeek et al., 2018). To date there have been no variants of genome-wide significance identified, likely due to the paucity of the very large samples required for such studies, the small effect sizes of each individual variant, and the statistical power burden of such techniques (Wray et al., 2014). These studies highlight the polygenic nature of ASB and other traits/disorders (Wray et al., 2014) whereby multiple genes of small effect are likely to be of importance. One method of capturing such polygenic influences, including in smaller samples, is to use polygenic risk scores (PRS) whereby a composite risk score captured across a large number of variants in one GWAS sample is investigated in a second sample to understand the genetic architecture of a particular trait, as well as cross-disorder or transdiagnostic influences (see Wray et al., 2014 for a more detailed explanation). From the few studies to date, there is some indication that polygenic risk scores are associated with ASB, confirming the importance of genetic factors (Hamshere et al., 2013), but more work is needed to fully understand their role.

Genetic studies have also provided initial evidence concerning the relationship between ASB and emotional functioning. Previous research has demonstrated an association between a functional variant in the catechol-O-methyltransferase (COMT) gene and ASB in those with ADHD (but not CD or ADHD alone; Lee & Song, 2018). Additional studies within both general population and clinical samples have suggested that this association is partially mediated by emotional functioning, but not executive functioning (Langley et al., 2010; van Goozen et al., 2015).

Etiologically, the role of both family environment and genetic effects are recognized and it is likely that the interplay of these different factors is important through gene-environment interactions (GxE) in which the effects of genetic influences are dependent upon the presence of specific environments (or vice versa). This interaction can take various different forms, many of which have been examined in relation to ASB. The diathesis stress model hypothesizes that genetic factors lead to more adverse outcomes in some environments but not others (Belsky & Pluess, 2009). This is probably the most frequently investigated model of GxE and has been studied in relation to ASB in studies examining interactions between the

monoamine oxidase A (MAOA) gene and maltreatment, with some studies identifying a significant increase in ASB associated with the low activity MAOA alleles but only in the presence of maltreatment (Caspi et al., 2002; Ouellet-Morin et al., 2018), although not all replications have supported this (e.g., Young et al., 2006).

Another type of interaction is the bioecological or "social push" model, whereby genetic influences are only relevant when environmental conditions are favorable. For example, a twin study investigating the role of parental conflict in CD found that genetic factors were more important at low levels of conflict, whereas shared environmental factors were more relevant at high levels of conflict (Burt & Klump, 2014).

These different types of GxE for ASB have been identified using behavioral genetics via twin or adoption studies and candidate gene or PRS approaches, and by investigating a range of family factors (e.g., conflict, maltreatment, monitoring) and peer factors (e.g., deviant peers, bullying/victimization), as well as more distal factors such as socio-economic status and neighborhood risks (for a full review, see Holz et al., 2018). Despite the generally recognized importance of these interactions, investigating GxE is complex, and to date findings have been inconclusive, with inconsistent replication of findings and a lack of understanding of the specific causal mechanisms that may be involved. Additionally, behavioral genetic studies are limited by the inherent limitations of twin designs or the non-representativeness of adoption cohorts (which are more likely to have exposure to biological risk and to have less adverse experience in their adoptive families; Dick, 2011), whilst molecular genetic studies are restricted by our knowledge of relevant genetic risk factors and the limitations of candidate gene approaches, as discussed above. Finally, hypothesis-free approaches may result in extremely large numbers of analyses (Holz et al., 2018). The multiple types of possible interaction can lead to issues with multiple testing, and false positive and false negative findings, exacerbated by the fact that the statistical power to identify interaction effects is lower than when detecting main effects (Dick, 2011).

Another way in which interactions between genes and environment may arise in ASB is through epigenetic effects. Epigenetics refers to alterations in the expression of genes without changes to the DNA, for example "turning on" or "turning off" of specific genetic effects, which occur mainly via changes to DNA methylation. Because changes in methylation – and therefore gene expression – can occur due to environmental exposure, it is thought that epigenetic effects might be responsible for some gene-environment interactions (El-Sayed et al., 2013). For example, Beach et al. (2011) found that increased methylation of the serotonin transporter gene (5HTTLPR) significantly mediated an association between childhood sexual abuse and symptoms of antisocial personality disorder in a sample of adult females; there are also indications that whole genome patterns of methylation are associated with childhood aggression in both males and females (Guillemin et al., 2014; Provencal et al., 2014). Although such findings are promising, they are often small effects observed in small

samples, and more work is needed to replicate them. There are also methodological issues to be overcome. These relate to knowing which candidate genes should be investigated, understanding the impact of small effect sizes, and addressing the multiple testing burden associated with genome-wide (methylation) studies (Holz et al., 2018). There are also factors specific to epigenetic research; for example, methylation changes can be tissue-specific and most researchers are not equipped to examine changes at the level of the brain while relying on other tissue samples, such as skin or blood (Holz et al., 2018). Nevertheless, this is an area that requires further investigation and illustrates the complexities involved in improving our understanding of the etiology of ASB and the interplay between genes and the environment.

It is also the case that a child's genotype can evoke reactions in others, thereby influencing their environment (evocative gene-environment correlation or rGE; Rutter & Silberg, 2002). For example, a child's genetically influenced behavior, such as irritability or early behavioral problems, is likely to evoke negative interactions with others (such as negative parenting or family conflict). Considering the developmental pattern of ASB, such processes are likely to be extremely important in the continuation of antisocial behavioral problems (van Goozen et al., 2007). Although teasing apart such effects can be difficult, there is evidence (especially from adoption or twin studies) that associations between child aggression and poor parenting or family processes are diminished when the child's genetic propensity is taken into account (e.g., Klahr et al., 2013; Marceau et al. 2019), demonstrating the need to include this in our model. However, more work is needed to understand the extent of such processes and how they should be considered in relation to neurobiological processes.

**Fearlessness and neural features of antisocial behavior**

Individual differences in aggression emerge in late infancy (Hay et al., 2011) and impairments in the processing and experience of negative emotions, and fear in particular, are important in early onset antisocial development (Gao, Raine, Venables, Dawson, & Mednick, 2010; van Goozen, 2015).

The fearlessness theory (Raine, 2013) states that antisocial individuals are relatively impaired in the perception and experience of fear, and that the neural systems that normally process threat information are structurally and/or functionally compromised. The findings of functional and structural neuroimaging studies in young people exhibiting conduct problems show reduced amygdala, anterior cingulate and orbitofrontal cortex activation, compared to typically developing controls, during the processing of negative affective stimuli (e.g., Passamonti et al., 2010; Rubia et al., 2009), and reductions in amygdala, anterior insula and orbitofrontal cortex volumes (e.g., Fairchild et al., 2011; Hyatt et al., 2012; Raine 2018). Impairments in amygdala function and closely connected systems lead to problems in the processing of negative emotions, in particular fear, and render the individual relatively 'fearless' and unemotional.

Children exhibiting ASB engage in various types of problematic and disruptive behavior that suggest they have an elevated threshold for responding to negative events (distress, danger) and/or experience negative emotions less intensely. Understanding the neurobiological pathways and systems associated with under-arousal, fearlessness and reduced distress response provides important clues to how stress affects mood, cognition, and behavior (Van Goozen & Fairchild, 2008).

Self-regulation at a neurobiological level can be studied by examining the functioning of the two main stress regulation systems: the ANS and the HPA axis. The ANS is an instinctive and fast-acting pathway; it regulates critical life functions and consists of the parasympathetic nervous system (PNS) and the sympathetic nervous system (SNS). In times of stress, a nearly complete withdrawal of the *vagus nervus*, the main nerve of the PNS, occurs (Porges, 2001). Metabolic demands are suppressed, facilitating fight-flight reactions by accelerating heart rate and activating sweat glands, which increase skin conductance level (SCL). In times of rest, the *vagus nervus* decelerates heart rate, facilitating social engagement (Porges, 2001). Malfunctioning of the ANS system places children at risk for emotion dysregulation (Beauchaine, Gatzke-Kopp & Mead, 2007) and there is an extensive body of research on ANS correlates, particularly skin conductance and heart rate, in antisocial and violent individuals (Raine, 2013).

Being exposed to a stressor also activates the other, slower-acting stress regulation system, the HPA-axis. When a stressor is perceived and appraised, the hypothalamus starts to release corticotrophin releasing hormone (CRH) from the paraventricular nucleus (McEwen, Gray & Nasca, 2015). CRH subsequently stimulates the release of adrenocorticotropic hormone (ACTH) from the pituitary, which in turn activates the adrenal glands to release cortisol. Cortisol, via negative feedback inhibition on the hypothalamus, pituitary, and other brain structures (such as the hippocampus), suppresses the HPA axis, eventually leading to restoration of basal cortisol levels and recovery (Gunnar & Quevedo, 2007). Research on children suggests that exposure to chronic stress is correlated over time with initially elevated and subsequently blunted stress hormone levels (and other disrupted stress physiology), changes that have an impact on the children's emotions and cognitive skills (Hostinar et al. 2014; Gunnar et al., 2006).

In general, children with severe aggression problems or CD have lower ANS system activity during rest as well as during stress (e.g., Beauchaine, Gatzke-Kopp & Mead, 2007; Lorber, 2004; Scott & Weems, 2014), and a reduced cortisol stress response when challenged (e.g., Fairchild et al., 2013; Van Goozen et al., 2000). These findings indicating reduced arousal, responsiveness and regulation are consistent with the notion of impaired neurobiological regulatory function in children with ASB (Van Goozen et al., 2007), and potentially explain why children with ASB often lack fear of the negative consequences of their actions and may also be motivated to seek out and engage in risky or stressful activities.

Importantly, studies also show that low physiological arousal can precede the onset and predict the persistence of antisocial behavior, even in infants (Baker et al., 2013) and young children (Gao et al., 2010). These studies show not only that physiological measures predict later aggression through their association with deficient emotional reactivity and regulation, but also that it is possible to identify children who are potentially at risk long before problematic behavior is observable.

It should be acknowledged that there are findings that are not consistent with the fearlessness or under-arousal hypothesis (e.g., Calkins, Graziano & Keane, 2007; Scott & Weems, 2014). Such inconsistencies might reflect methodological differences, such as variations in populations sampled or types of informants or stressors used. As noted above, children with ASB are a heterogeneous group, not only with respect to behavioral phenotype (i.e., individual differences in aggression and comorbid symptoms of anxiety, attention deficits and autism), but also with respect to developmental course and outcome (Lahey et al., 2005; Stadler et al., 2010).

A focus on assessing intervening processes or underlying mechanisms should help to explain variation in type, severity, and outcome (Van Goozen et al., 2007, 2015). For example, some children with ASB may exhibit heightened autonomic activity and be more vulnerable to stressful situations because their nervous system is already 'primed' for reaction (Gatzke-Kopp et al., 2012), creating greater risk of displaying reactive aggression (Bubier & Drabick, 2009). Other children with ASB might fail to react to demanding or challenging situations, showing under-arousal, and are therefore less able to attend to and use environmental cues that are needed to adapt their behavior. Thus, within the group of children with ASB, differences in underlying neurobiological processes may explain differences in behavioral phenotype (Belsky & Pluess, 2009; Van Goozen et al., 2007).

**Emotional functioning in children with ASB**

Consistent with this latter argument, neuropsychological studies in children with ASB or psychopathic tendencies show impaired fear recognition and reduced startle and pupil amplitudes to negatively-valenced pictures or fear-arousing events (Burley & Van Goozen, 2020; Fairchild et al., 2013; Marsh & Blair, 2008; Schwenck et al., 2012), strongly suggesting that these children are relatively fearless and that their behavioral problems have a neuropsychological basis in impaired emotion processing. Impairments in amygdala function and closely connected systems (Raine, 2018) cause problems in the recognition and processing of fear and distress and render the individual relatively 'fearless' and unemotional; this makes it difficult to recognize cues from the environment that signal threat or submission, and elicit compassion or empathy (Blair, 2005 & 2013).

*Children with severe ASB have impairments in recognizing negative emotions and in empathy for other's distress*

Being able to detect, process and respond appropriately to the emotions of others is crucial for normal social interaction and helps to initiate and maintain healthy relationships (Izard et al., 2001; Herba et al., 2006). Children who demonstrate accurate facial emotion recognition engage in more prosocial behaviors, are liked more by their peers, and show more empathic responses (Denham, Bassett, Zinsser, & Wyatt, 2014). Recognition of others' emotions and responding with empathy are learned through experience and based on the gradual refinement with age of children's production and recognition of emotional signals. Caregivers, parents and teachers play a key role in the development of children's emotion recognition proficiency (Steele et al., 2008). Not only do they model appropriate emotional facial expressions; they also provide a social context in which children can learn what these expressions mean.

Conversely, research in children who have been exposed to adverse emotional environments, such as those who have experienced maltreatment, neglect or abuse, reveals impaired or biased facial emotion recognition and understanding (Kujawa et al., 2014; Pollack & Sinha, 2002). There is increasing evidence that such impairments are influenced by family factors (e.g., genetic, family relationship quality) and have profound effects on psychological outcomes and social functioning more broadly (Burley et al., 2021; da Silva Ferreira, Crippa, & de Lima Osório, 2014).

Difficulties in emotion recognition are well documented in children and adolescents with a range of mental health, neurodevelopmental and behavioral issues, indicating that impaired emotion recognition is a transdiagnostic risk factor (Collin, Bindra, Raju, Gillberg and Minnis, 2013; Cooper, Hobson & Van Goozen, 2020). In individuals with ASB, reduced emotional awareness and a failure to recognize cues from the environment that signal threat, distress or submission can lead to a lack of empathy or guilt, which may continue and intensify the problematic behavior (Blair, 2005; 2013). This impairment may affect children's developing relationships in everyday life, where proper emotion regulation and adaptation to a changing social environment are required.

Behavioral problems, including conduct problems, attention and hyperactivity difficulties, peer problems and a lack of prosocial behaviors, have repeatedly been linked to impairments in recognizing negative facial emotions (fear, anger, sadness) in childhood and adolescence (Airdrie, Langley, Thapar, & van Goozen, 2018; Hunnikin, Wells, Ash, & Van Goozen, 2019; Marsh & Blair, 2008). A specific impairment in fear recognition has been widely reported in antisocial and violent samples (Marsh & Blair, 2008), although impairments in sadness and anger recognition have also been observed (Fairchild et al., 2013; Hunnikin & van Goozen, 2018). In addition, antisocial individuals have been reported to display a 'hostile attribution bias', misinterpreting ambiguous or neutral faces as angry (Dadds et al., 2006; Penton-Voak

et al., 2013). However, there is considerable individual variability in risk factors for antisocial behavior in general, and in the nature and severity of emotion recognition ability in particular. Not all individuals who exhibit ASB have emotion recognition impairments. One study found that 41% of children who were rated by their teachers as having severe conduct problems and adverse childhood experiences were proficient in emotion recognition (Hunnikin et al., 2021). Interestingly, these children were also reported to have fewer peer problems, confirming the role of emotion recognition in social interaction.

Empathy, the ability to recognize and understand another's emotional state (cognitive empathy) and to vicariously experience or share another's emotional state (affective empathy; Bons et al., 2013; Singer, 2006) is another process important in explaining conduct problems. Some researchers equate cognitive empathy with emotion recognition (Schwenck et al., 2012) and suggest that emotion recognition impairments underlie deficient empathy (Blair, 2005). There is some evidence of a positive relation between facial emotion recognition and cognitive (but not affective) empathy, although other studies have not found this relation (Hunnikin et al., 2020).

Empathy impairments have been causally linked to ASB. Emotional facial expressions have evolved to signal distress and evoke empathic responses, which inhibit (further) aggressive acts (Blair, 2005). Although some studies have found that antisocial individuals are impaired in both cognitive and affective empathy (e.g., Dawel et al., 2012; Hunnikin et al., 2020), most have found intact cognitive but impaired affective empathy specifically in relation to others' negative emotions (e.g., Van Zonneveld et al., 2017). This pattern of findings has been observed in children with conduct problems and callous-unemotional (CU) traits (Pasalich, Dadds, & Hawes, 2014; Schwenck et al., 2012), in children with conduct disorder with or without ADHD (Van Goozen et al., 2016), and in children with psychopathic tendencies or conduct problems recruited from the community (Jones, Happé, Gilbert, Burnett, & Viding, 2010). Variations in methodology may be responsible for some of the differences in findings: whereas most emotion recognition studies assess the ability to recognize static facial expressions, more advanced studies have used dynamic affective clips to study empathy, requiring an ability to understand vocal, gestural and contextual information to demonstrate cognitive empathy (Decety & Jackson, 2004). Antisocial individuals have been found to be impaired in integrating multiple sources of emotional information (Gonzalez-Gadea et al., 2014).

Only a handful of studies have measured affective empathy in children with ASB using physiological measures. Although physiological arousal is not synonymous with affective empathy, it represents a reliable, objective, and direct measure of this aspect of empathy (Bons et al., 2013), and reduced affective arousal in response to negative emotional events has been causally implicated in the development of ASB (e.g., Gao et al., 2010; Van Goozen, 2015). Studies measuring physiological arousal in children with disruptive behavior disorders

(De Wied, van Boxtel, Matthys, & Meeus, 2012), conduct disorder (Marsh, Beauchaine, & Williams, 2008) or those at risk of future criminal behavior (through being younger siblings or children of delinquents, and failing primary school; van Zonneveld et al., 2017) have observed decreased physiological responses in response to others' negative emotions. The van Zonneveld et al. (2017) study showed that high-risk children exhibited reduced HR, SCL and SCR specifically in response to others' pain and fear, despite not differing from typical controls in social attention (assessed by eye-tracking), cognitive empathy, or affective empathy to happiness. Whilst problems in affective empathy in response to negative emotions have previously been observed in children with CD (e.g., Van Goozen et al., 2016) or CU traits (Lockwood, Bird, Bridge, & Viding, 2013), the children in the van Zonneveld et al. study were not psychopathic, and did not have a diagnosis of CD, thereby showing that affective processes play a role in a wider range of children with conduct problems.

Although social attention to emotionally charged events is required to ensure an empathic response, and some studies (Dadds et al., 2008) have suggested that fear recognition problems in children with CU-traits are partly due to a failure to attend to key areas in the face that reveal emotional information (e.g., the eyes), recent studies have appeared to rule out a role for social attention in emotion impairments in children with ASB (Airdrie et al., 2018; Hunnikin et al., 2021; van Zonneveld et al., 2017).

This review of evidence on emotional functioning in children with ASB supports the view that these children are impaired in their ability to process negative emotional information and also in the 'gut' level emotional response that is needed for affective empathy. A subgroup of children with ASB, however, may have a higher-level understanding of what the other person is feeling (and may use this to manipulate or exploit others), in line with evidence that cognitive empathy is not always impaired.

**Impaired executive functioning (EF) in children with ASB**

Executive functions are involved in controlling thought, emotions and behavior, and subserve self-regulation. There are several key executive functions: inhibition, interference control, working memory, and cognitive flexibility (Diamond, 2013). Adequate social functioning requires being able to flexibly adapt to changing environments. This requires the ability not only to perceive and process emotions, but also to adapt behavior in situations that are new, complex, unpredictable, or have high information load. Emotions can influence EF and studies have shown this by distinguishing between EF in neutral situations and EF in the context of affect, incentives and motivation, i.e., 'cool' versus 'hot' EF (Zelazo, 2020).

Cool EF facilitates cognitive regulation under non-reward conditions, and involves slow, deliberative processing and reasoning. Hot EF is defined as processes which involve top-down control to facilitate accurate problem solving and decision making in the context of situations involving a high degree of affective and motivational demand (typically involving regulating

responses under conditions of reward or loss; Zelazo & Carlson, 2012). Functional neuroimaging and lesion studies (e.g., Rubia, 2011) have demonstrated differential patterns of neural activation associated with cool and hot EF tasks: Cool EF is associated with frontal-striatal regions, whereas hot EF is associated with orbitofrontal-limbic regions. An important topic in ASB research has been to understand the relative contributions of cool and hot EF to ASB.

Due to the high comorbidity between ODD/CD and ADHD (which has been conceptualized as an EF disorder; Barkley, 1997) and to the fact that many children with ADHD do not develop 'antisocial' behavior, it is important to understand which cool and hot EF deficits are present in childhood ASB, independently of ADHD. Many antisocial acts, particularly those involving verbal or physically aggressive behaviors, could be regarded as involving a loss of inhibitory control. A recent meta-analysis of inhibitory control deficits in cool EF tasks, comparing children with ODD/CD and ADHD, pooling findings in both motor and cognitive inhibition tasks, suggests that children in 'pure' ODD/CD groups perform more poorly than typical controls in such tasks (Bonham, Shanley, Waters & Elvin, 2021). However, the authors note that these findings could be accounted for by significantly higher levels of ADHD symptoms within the 'pure' ODD/CD groups (even though these groups do not meet full diagnostic criteria for ADHD, they had significantly higher levels of ADHD symptoms across the studies). The authors of the meta-analysis therefore argue for a dimensional framework for future research.

In the few studies that have adopted dimensional analyses, there is mixed evidence that ODD/CD is associated with poorer inhibitory control when controlling for the presence of ADHD symptoms. For example, Hobson et al. (2011) found evidence for associations, when controlling for ADHD symptoms, between ODD/CD and poorer Stop task performance, but not in a Go/No-Go task or in across-task premature responding. A possible reason for these inconsistent findings is the nature of a diagnostic framework that subsumes different presentations and etiologies within supposedly discrete disorders. For example, ODD subsumes an affective/emotional component (e.g., easily annoyed, angry/resentful, being spiteful) and an impulsive/antagonistic behavior component (loses temper, actively defies, argues, deliberately annoys others, blames others for misbehavior). Interestingly, Griffith, Arnold, Rolon-Arroyo and Harvey (2019) found, when controlling for ADHD symptoms, that oppositional/antagonistic ODD symptoms were associated with poorer response inhibition, but that affect-related ODD symptoms were associated with *improved* response inhibition. Hence, if *emotional* dysregulation is considered to be an essential feature of the development of ASB in young children, this study suggests that cool inhibitory control deficits are not key to understanding the development of antisocial behavior beyond comorbid ADHD.

Other types of cool EF impairments have been found in ODD/CD samples, but the impairments that are reported are varied. The majority of studies controlling for ADHD have

found that cool EF deficits in ODD/CD samples, in the areas of planning, verbal fluency, cognitive flexibility, response variability, working memory and sustained attention, are largely accounted for by ADHD (e.g., Antonini, Becker, Tamm & Epstein, 2015; Dolan & Lennox, 2013; van Goozen et al., 2004). However, some exceptions have been found, whereby cool EF abnormalities were found in ODD/CD independently of ADHD in adequately controlled studies, for example in some aspects of working memory (Rhodes, Park, Seth & Coghill, 2012) and in intra-subject response variability (e.g., Hobson et al., 2011; Scheres, Oosterlaan & Sergeant, 2001).

Whereas the most common and consistent correlates of ADHD are measures of cool EF, some have argued for a role of hot EF difficulties, such as high delay aversion (Sonuga-Barke, 2003; Petrovic & Castellanos, 2016), which is often found in ADHD samples. However, the elevated levels of more overt antisocial behavior in ADHD samples, including those symptoms common to ODD and CD, may account for these findings. Many childhood antisocial behaviors could be seen as 'misjudgments' in terms of a child's ability to properly consider the likely rewards compared to the likely negative consequences. It is therefore relevant to consider ODD/CD performance in hot EF tasks that measure response perseveration in the face of punishment, impaired decision-making in relation to rewards, and punishment insensitivity.

Indeed, studies of hot EF have consistently found impairments in ODD/CD samples (e.g., Dolan & Lennox, 2013; Hobson et al., 2011; Syngelaki et al., 2009; Van Goozen et al., 2004; Woltering et al., 2016), and there is emerging evidence that hot EF problems are more characteristic of ODD/CD than they are of ADHD. Although it is well-established that ADHD children tend to prefer immediate rewards to delayed rewards in delay of gratification tasks, the majority of these studies have not accounted for the possible impact of ODD/CD (Luman, Tripp & Scheres, 2010), and in the studies that have done so (e.g., Griffith et al., 2019), ODD/CD was found to be a stronger predictor. Evidence has also been found in studies using card-playing paradigms that measure problems in motivational inhibition (continuing to respond for a reward despite receiving increasing punishment) that reveals deficits in pure ODD/CD groups but not in pure ADHD groups (van Goozen et al., 2004). ODD/CD has also been found to be associated with riskier behavior in reward-related decision-making tasks such as the Iowa Gambling Task and its variants, with a systematic review of risky behavior in such gambling tasks concluding that the evidence was generally mixed for ADHD, but that comorbid ODD/CD increased risky behavior in ADHD (Groen, Gaastra, Lewis-Evans & Tucha, 2013).

In line with behavioral findings, a systematic review and meta-analysis of structural (sMRI) and functional (fMRI) studies of ODD/CD children with and without ADHD found evidence of ODD/CD abnormalities in the hot EF relevant regions of the bilateral amygdala, bilateral insula, right striatum, and left medial/superior frontal gyrus, irrespective of ADHD (Noordermeer, Luman & Oosterlaan, 2016). These authors also found that abnormalities in

the amygdala, a key area involved in the processing of emotional responses, were *specific* to ODD/CD, yet there was only weak evidence of specific ODD/CD abnormalities (independently of ADHD) in structures commonly associated with cool EF, such as the cerebellum and the dorsolateral prefrontal cortex.

In summary, the evidence supports what we have argued elsewhere (Thapar & Van Goozen, 2018), namely that hot (rather than cool) EF pathways are key correlates of ASB in children, independently of ADHD, and that problems in emotion processing and experience explain these hot EF impairments. Such emotional impairments may be caused by early adversity. The toxic effects of adversity and stress on the development of the fronto-limbic system (e.g., Shonkoff et al. 2012; Figure 1 this paper) can explain impairments in EF that are observed in children with severe ASB, particularly under emotionally salient conditions.

**Implications for intervention**

*The role of the early caregiving environment in children's self-regulation problems*

How do these impairments in emotion and cognition develop in children? Heritable processes are not the only mechanisms that induce neural maldevelopment. Some environmental risk factors that impact on children's neural development and functioning are prenatal (e.g., exposure to maternal smoking in utero, maternal psychopathology), while others such as poverty, poor living conditions, inconsistent caregiving, neglect or maltreatment are postnatal (Gunnar et al., 2006). Nearly 40% of children aged between 5 and 17 years who are in care, who have been abused, or are on the child protection/safeguarding registers have a conduct disorder (NICE, 2017).

Studies consistently show that early life events have neurobiological consequences, including effects on the HPA axis, which subsequently affect the child's emotional and cognitive functioning, and may persist into adult life (Roth & Sweatt, 2011; Figure 1). Studies of non-human animals show that variations in early maternal care affect the development of individual differences in neuroendocrine responses to stress, such that maternal behavior 'programs' HPA responses to stress in the offspring (Kaffman & Meaney, 2007). The results of studies of the effects of early stress on the developing neurobiological systems in at-risk or clinical populations also show longer-term changes in HPA axis functioning (Gunnar & Quevedo, 2007). Although the quality of parental care in humans predicts child self-regulation outcomes (Blair, 2010), we do not yet know which aspects of parental behavior determine the unfolding of infants' stress responses, or how these physiological responses map onto behavioral adjustment. Exposure to early adversity can disrupt brain development by inhibiting gene expression (i.e., epigenetic mechanisms) or by amplifying preexisting vulnerabilities (Kaffman & Meaney, 2007; McEwen, Gray & Nasca, 2015). There is increasing interest in the effect of early risk exposure on variations in DNA methylation (Roth & Sweatt, 2011) and as discussed above, studying epigenetic processes and their effects on neural

development and problem behavior in humans is inherently complicated but certainly needed.

The fact that conduct disorders have a steep social class gradient, with a three- to four-fold increase in prevalence in the lowest social classes (NICE, 2017), is further evidence of the importance of the environment in which children develop. Children from low-SES families receive less support for language acquisition and have fewer opportunities to engage in activities or play games that help develop EF skills (Zelazo, 2020). These conditions, in combination with exposure to more sustained and uncontrollable stress due to poverty and other forms of psychosocial adversity, make it difficult for children to predict and anticipate sequences of events, and elicit chronic and recurring stress (e.g., Blair, 2010) — effects that are likely to impair the prefrontal–amygdala–striatal system and to affect emotion and cognitive development (Shonkoff et al. 2012; Zelazo, 2020), thereby contributing to emotion dysregulation and aggression.

*Prevention and early intervention*

A neurodevelopmental approach to intervention recognizes the need to intervene early in life to prevent or ameliorate dysfunctional brain development. Knowing that children with ASB are more likely to come from adverse rearing environments that involve atypical caregiver-child interaction, parental psychopathology, or compromised pre- or perinatal development is consistent with the view that adverse early influences have lasting effects on developing systems in the brain that are important in emotion processing and self-regulation (Leppänen & Nelson, 2006). Such an interpretation suggests that positive rearing environments have protective effects and that interventions early in life have the potential to be successful in altering antisocial trajectories (Skeem et al., 2014) and bringing about lasting change through their enduring effects on the developing neural systems that are involved in emotion and cognition (van Goozen & Fairchild, 2008).

If ASB is in part neurodevelopmentally determined, successful prevention efforts should be more effective if they begin early, given that experience-dependent neuroplasticity is at its peak during early childhood (Luby, 2012). Clinical and social services that help to improve health and wellbeing in at-risk mothers, and educational support for at-risk children, preparing them for school entry, should help to reduce neural maldevelopment, reduce disruptive behavior, and – at least to some extent – alleviate this significant public health problem. Indeed, there is evidence of long-term benefits of early interventions in the pre- and postnatal periods. For example, a biosocial prenatal program that targeted maternal health factors – focusing on reductions in smoking and alcohol use during pregnancy and better nutrition with nurse visitations – resulted in significant reductions in juvenile delinquency 15 years later (Olds et al., 1998; Raine, 2018). Another health intervention that enhanced the early environment of young children through better nutrition, more physical exercise, and cognitive stimulation was associated with a 34% reduction in criminal offending at age 23

years (Raine et al., 2003).

The importance of early intervention is increasingly recognized, and research demonstrates that early intervention is both behaviorally (Piquero et al., 2016; Skeem et al., 2014) and economically (Chowdry & Fitzsimons, 2016) more effective than later intervention. More effective strategies for targeting the relatively small group of children at high risk of persistent antisocial behavior, including those at risk of future involvement in the criminal justice system, at an early, sensitive period for intervention would provide opportunities not only to help these children attain a more positive developmental trajectory, but also to diminish the negative impact of their behavior on society. Research has challenged the notion that high-risk children inevitably mature into adult offenders, raising the possibility that well-targeted interventions could create a turning-point in antisocial behavior for high-risk children (Skeem et al., 2014). Childhood is a time when children are particularly adept at specific kinds of social and emotional learning, thereby creating a window of opportunity for intervention.

*Targeting mediating processes*

Interventions targeting parenting practices have been found to be effective in reducing antisocial and aggressive behavior (Piquero et al., 2016) but not all children with ASB respond positively to parent training programs and there is considerable variability in the amount of change achieved. We have argued in this paper that there is a need for interventions that (a) are not fully reliant on parents, (b) work in a targeted manner based on clinical need (not diagnosis), (c) draw on our knowledge of the basic science of brain-related problems and (d) can be implemented during crucial developmental periods. Schools provide an ideal setting to deliver interventions, especially to children coming from adverse backgrounds.

Focusing on self-regulation and mediating emotional and cognitive processes has the potential to enhance our understanding of childhood aggression at different ages, providing knowledge that is relevant for the design of interventions that would improve developmental outcomes. A focus on underlying processes also helps to identify those children who are most likely to persist in engaging in severe ASB and who would benefit from specific types of intervention, such as empathy training; or show that children's impairments are such that some psychosocial interventions that presuppose basic emotion skills (i.e., emotion recognition) or the ability to weigh up rewards and costs are *less* likely to work because they depend on processes that are impaired in some children (Van Goozen & Fairchild, 2008).

Impaired emotion recognition has been found in numerous antisocial populations and is thought to play a causal role in the development and maintenance of ASB (Marsh & Blair, 2008). Emotion recognition is negatively related to peer problems (Wells et al., 2020), consistent with the claim that there is a relationship between impaired emotion recognition and social relationships (Izard et al., 2001). Given that having better friendships with others is a protective factor in preventing further behavioral issues, this is further evidence of the

potential utility of emotion recognition training. Indeed, research is beginning to show the longer-term positive effects of improving emotion recognition on behavior, mental health and wellbeing in children and young people with severe ASB (Dickerson et al., 2021; Hubble, Bowen, Moore, & van Goozen, 2015; Penton-Voak et al., 2013; Wells et al., 2020). However, in line with evidence that not all children with ASB are impaired at recognizing the emotions in others (Wells et al., 2020), these interventions need to be tailored to those with emotion recognition impairments and/or peer problems.

Similarly, there is substantial evidence that EF skills mediate the relations between environmental factors and negative outcomes, supporting the use of early interventions that train these skills in order to prevent the development of psychopathology and improve antisocial trajectories (Miyake & Friedman, 2012). The heterogeneity in risk factors and symptoms suggests that a psychological approach that involves a combination of identifying specific impairments and targeting those associated with symptoms of psychopathology is likely to be more effective and represent a better use of financial and other resources.

Self-regulation has been identified as a target for transdiagnostic interventions. Early training could be used as a preventative intervention, independent of specific diagnoses, to promote resilience and protect individuals at risk of ASB (Piehler et al, 2014). In view of the evidence that hot rather than cool EF pathways are key correlates of ASB in children, and given that problems in emotion processing may explain these hot impairments, cognitive training should seek to improve executive control, frustration regulation and delay of gratification (i.e., punishment and reward sensitivity; Ip et al., 2019). Metacognitive reflection and explicitly teaching children how to practice self-regulation skills and reflect on their arousal levels and motivation could also help to reduce symptoms and promote positive development and resilience.

Although there are many cognitive interventions designed to improve cool EF skills (especially sustained attention and working memory; Rapport et al., 2013), relatively few target self-control or hot EF. Some of the more promising ones come in the form of applied games and use calming (slow paced respiration) biofeedback to lower stress/arousal levels in order to engage better and use cognitive processes more effectively to guide behavior (Sonne & Jensen, 2016). Another intervention for children (“Focus Pocus”; Johnstone et al., 2017) targets arousal via neurofeedback, alongside cognitive training (see Alegria et al. [2017] for an fMRI neurofeedback study in 12–17 years-old boys with ADHD). This intervention found clear effects on EEG measures and also reductions in ADHD symptom severity, but only minor effects on measures of inhibition and cognitive processing. This suggests that cognitive factors alone cannot explain the reductions in ADHD symptoms, and that other factors, such as emotion and motivation, play a role in the observed improvements. A limitation is that most of these interventions have been developed for children with ADHD. Another shortcoming is

that although applied games are a viable and cost-effective way to deliver interventions, very few studies have tested the effects of these games in a scientifically rigorous way.

In summary, there is a pressing need for more evidence that improving processes such as emotion recognition and emotion regulation reduces clinical symptoms of externalizing problems. The relatively few training interventions targeting intermediate processes that have been used to support children with ASB suffer from methodological limitations: significant effects have often been reported in studies using small sample sizes; active control groups are rarely used; and non-blinded raters have sometimes been used to assess outcomes (Rapport et al, 2013; Wells et al., 2020). Wherever possible, future research should use double-blind, randomized controlled trials when testing the effectiveness of interventions, and include follow-up measures to index the long-term effect on symptoms. Finally, increased precision in the tailoring of interventions should help to develop more effective interventions targeting the key mechanisms implicated in disorders associated with dysregulation.

**Implications and conclusion: The critical importance of early detection and intervention**

The theoretical model that we proposed in 2007 (Figure 1) hypothesized that genetic factors and early childhood adversity act as precursors to disruptions in neurobiological and neuropsychological functioning which, through a bidirectional interplay, affect ASB. The research reviewed here supports our model but much of that research has methodological limitations that mean that further research is needed. For example, there is variation in the quality of the tasks used to study mediating processes; current evidence is also dependent upon studies with small sample sizes, so there is a need to use larger samples and to replicate findings in additional studies or to make greater use of meta-analyses. Researchers should aim to study how factors influencing ASB differ with developmental age. Heterogeneity within ASB is an increasingly recognized issue but one that has not fully been considered in research to date; future studies should also consider the differential factors associated with ASB where it co-occurs with other neurodevelopmental or emotional difficulties, for differential developmental trajectories of ASB and for individuals with different family situations. A final point is that the majority of studies that can be regarded as 'state-of-the-art' research on the neurobiological and neuropsychological bases of childhood ASB are cross-sectional and correlational in nature. This necessarily limits any conclusions regarding the causal nature of neural influences on ASB. Although it is only experimental research that can unambiguously answer questions about causality, an important step in developing this field of research would be to conduct prospective longitudinal studies that permit tests of the mediating and moderating factors that underlie early adverse influences on ASB in childhood. Nevertheless, longitudinal studies cannot provide definitive evidence of causal relations between constructs, so it will be necessary to complement longitudinal correlational studies with carefully designed experimental studies. This will be the 'gold standard' by which future research in this area will be evaluated, with the triangulation of findings from studies using

different research designs adding strength to conclusions (Lawlor, Tilling & Davey Smith, 2016).

In reviewing research on ASB it was also our intention to inform the design of interventions that are likely to be more effective in helping children who exhibit emerging behavioral problems. The early childhood years are a crucial time for psychological development. Early experiences foster learning and brain development and help to shape emotional and cognitive skills, language and social relationships. Children who exhibit behavioral problems and early signs of neurodevelopmental problems do not make the same progress as typically developing children: They engage less well with school, are less likely to have a network of family and friends with whom they feel close, lag behind their peers and may never catch up. Our review highlights the need to intervene early because early intervention is more effective in shaping the processes responsible for ASB. Young children are also more open to influence because they are still in the process of developing the emotional and cognitive skills that are important for wellbeing and resilience (Herba et al., 2006; Luby, 2012). Sadly, many children who need such intervention do not receive it (McManus et al., 2020).

Due to the Covid-19 pandemic (with the associated lockdowns and school closures) children have experienced increased social isolation from peers and disrupted educational input, both of which are likely to negatively impact upon the emotional and executive function processes that were identified in this review as relevant to the development of ASB. Further, the pandemic has, at least in the short term, probably exacerbated the home environmental risk factors for ASB, as a result of its adverse effects on adult/parental mental health (see Adegboye et al., 2021), and higher risk of child maltreatment due to increased parental stress, domestic violence and reductions in protective health and social care services (Romanou & Belton, 2020). Although the longer-term impact of the pandemic on children's ASB is not yet known, short-term detrimental impacts have been noted for the majority of children, in both typically developing and vulnerable samples, in the areas of depression, anxiety, attention/hyperactivity, oppositionality or conduct problems (Adegboye et al., 2021; Nonweiler et al., 2020; Cost et al., 2021). Although some researchers have noted a subgroup of children for whom there have been short-term improvements in psychopathology (Adegboye et al, 2021; Cost et al., 2021), perhaps due to overall lower stress levels in the subgroup of children who find the structure or social demands of school the most challenging, there is no theoretical reason to think that these gains will be maintained upon resumption of pre-pandemic social and educational practices. Overall, then, the likely impact of the pandemic highlights the need for greatly improved access to earlier and more targeted interventions for children (Raballo et al., 2020).

Children with ASB have complex problems and needs that are difficult for parents and professionals to identify and understand. We have highlighted the role of mediating processes in explaining variations in the type, severity and persistence of ASB, and have

argued that there is a need for more research on the impact of intervening at this intermediate level, either directly or by better assessing children's individual impairments in order to inform a targeted intervention plan. Most current interventions fail to take into account the specific individual cognitive–emotional problems and needs of children with ASB, and therefore do not target these needs. It seems sensible to identify subgroups of children with distinct neuropsychological profiles early in life. By identifying possible precursors of disorder in the context of typical development, we can achieve a better match between treatments and the specific needs of individual children, and thereby reduce the psychological and economic costs of antisocial behavior to individuals, families, and society.

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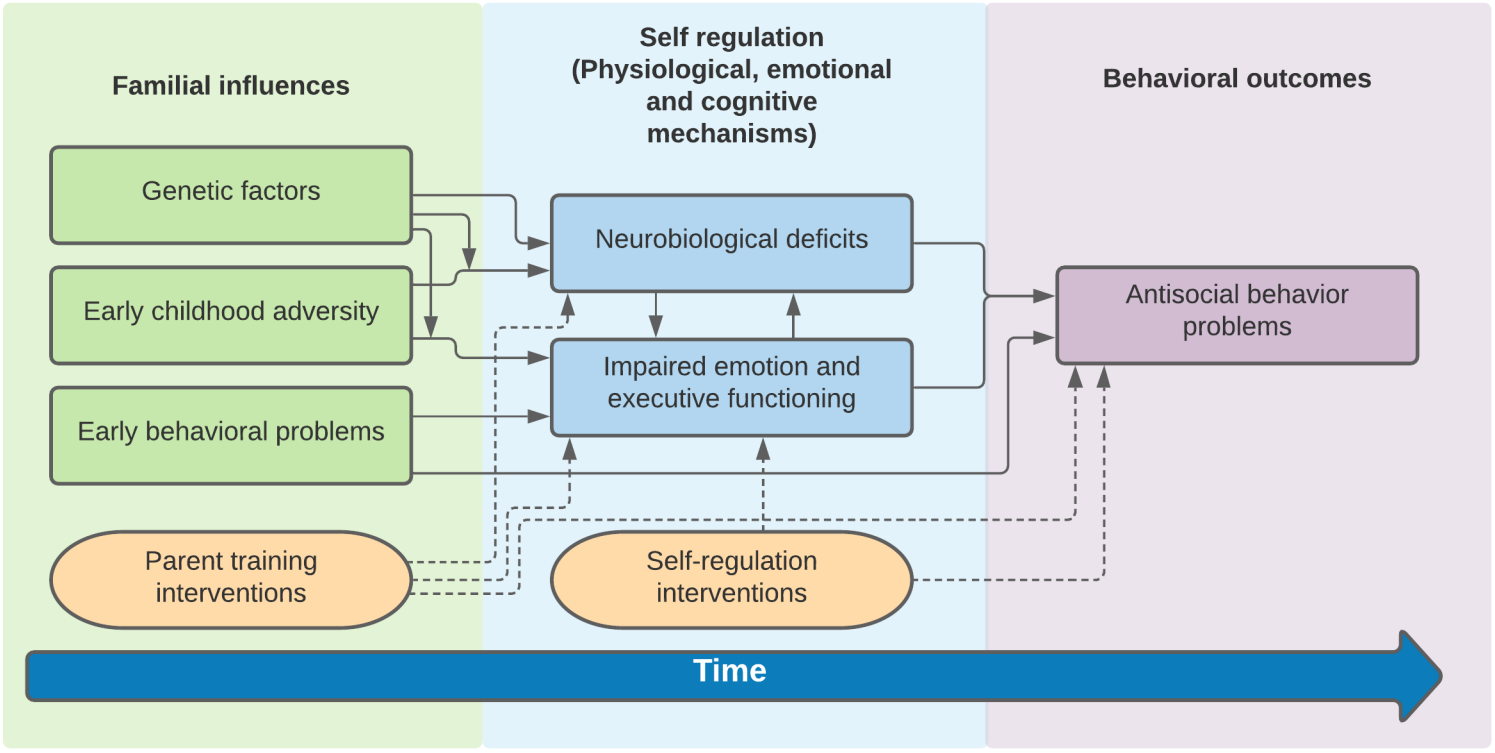
Figure Caption

Figure 1. Theoretical model based on van Goozen et al. (2007). The model represents pathways through which familial factors (genetic factors, early childhood adversity) are hypothesized to influence childhood onset antisocial behavior problems. The depicted pathways (solid arrows) are supported by empirical evidence (van Goozen et al., 2007; 2021). The dashed arrows represent the likely ways in which parent training interventions and self-regulation interventions exert an influence.


Familial influences
Self regulation (Physiological, emotional and cognitive mechanisms)
Behavioral outcomes
Genetic factors
Early childhood adversity
Early behavioral problems
Neurobiological deficits
Impaired emotion and executive functioning
Antisocial behavior problems
Parent training interventions
Self-regulation interventions
Time

**A summary list highlighting the central points**

Children with severe antisocial behavior have impaired neurobiological regulatory function and exhibit impaired recognition of negative emotions and empathy for others' distress.

Impairments in emotion processing and experience help to explain why 'hot' (rather than 'cool') executive function pathways are key correlates of antisocial behavior in children.

There is as yet only a weak link between what we know about the basic science of brain-related problems and clinical practice in this area.

Regarding childhood antisocial behavior as a neurodevelopmental problem could promote the development of interventions that focus on enhancing brain development and function in children at risk and most in need of early intervention.

Preliminary evidence in studies with children and young people indicates that emotional and cognitive skills can be enhanced, with positive effects on subsequent problematic behavior.

**Future issues**

To advance the understanding of antisocial development it will be essential to conduct more longitudinal research that simultaneously examines individual-level physiological, emotional and cognitive processes and permits tests of the factors that mediate and moderate the relations between early adverse influences and problematic development in childhood.

Prospective longitudinal research in young children with emerging emotional and behavioral problems would help to advance the argument that ASB should be regarded as a neurodevelopmental problem.

We should seek to identify children who exhibit distinct neuropsychological profiles at early developmental periods and then intervene in a targeted manner based on evidenced need, rather than clinical diagnosis.

Increasing precision in the tailoring of interventions that target key mechanisms should make interventions more effective.

**A list of up to 10 important abbreviations/acronyms**

ASB – antisocial behavior
ODD – Oppositional Defiant Disorder
CD – Conduct Disorder
ADHD – Attention Deficit Hyperactivity Disorder
HPA – Hypothalamic Pituitary Axis
ANS – Autonomic Nervous System
NICE - National Institute for Health and Care Excellence
CU trait – callous and unemotional trait
EF – Executive Function
GxE – Gene-Environment interaction