An ‘embedded brain’ approach to understanding antisocial behaviour

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Review

An 'embedded brain' approach to understanding antisocial behaviour

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Antisocial behaviour (ASB) incurs substantial costs to the individual and society. Cognitive neuroscience has the potential to shed light on developmental risk for ASB, but it cannot achieve this potential in an 'essentialist' framework that focuses on the brain and cognition isolated from the environment. Here, we present the case for studying the social transactional and iterative unfolding of brain and cognitive development in a relational context. This approach, which we call the study of the 'embedded brain', is needed to fully understand how risk for ASB arises during development. Concentrated efforts are required to develop and unify methods to achieve this approach and reap the benefits for improved prevention and intervention of ASB.

Highlights

The origins of antisocial behaviour are complex and vary between individuals.

We need to improve our understanding of the transactional and iterative unfolding of brain and cognitive development within a relational context. Without this study of the 'embedded brain', we cannot fully understand the emergence and maintenance of antisocial behaviour.

To advance the study of the 'embedded brain', the field needs to unify ways of measuring antisocial behaviour and generate more reliable and sensitive measures of brain, cognition, and social environment.

Improved understanding of different developmental pathways to antisocial behaviour can help personalise interventions and improve their effectiveness.

ASB in children and adolescents

ASB, including aggression and rule-breaking, that begins in childhood is associated with the development of a range of mental health problems, physical health problems, special educational needs, failure to complete education, and legal system involvement [1–4] – all of which incur substantial economic costs[i] [5]. Moreover, ASB has social and emotional costs that cannot be measured financially. There is a detrimental impact on the individuals who display ASB, those close to them, and those who have the misfortune of being a victim of their antisocial acts. ASB has in the past been conceptualised as a sociological phenomenon, with the focus on the role of societal, socioeconomic, and family factors in predisposing to ASB [6,7]. More recently there has been a shift away from this predominantly environmentally framed account towards models that stress the role of neurodevelopmental factors in ASB risk [8]. Fundamentally studies that focus either on the environment (often conceptualised as 'external to the child') or on individual neurodevelopment (often conceptualised as 'located within the child') fail to capture the complex and dynamic interplay between individuals and their social ecology.

Developmental psychopathology researchers have for a long time urged researchers to focus on transactional models that capture the interplay between a child and their social environment over time [9,10]. There are now a number of studies that have examined the reciprocal associations between different types of ASB and social environmental factors, with most reporting evidence of bidirectional influences [11–15]. For example, longitudinal data indicate that child ASB increases the likelihood of harsh parenting (including physical discipline), and that harsh parenting also increases the likelihood of ASB over time [13,14]. This finding has been replicated in genetically informative study designs, which have demonstrated both impact of a child's genetic endowment on parenting, as well as parenting effects on child behaviour that reflect both genetic and environmental processes [16–18]. However, there has not been an effective integration of experimental and neuroimaging work on socio-cognitive-affective processes into a transactional framework as we study the development of ASB. We suggest that in order to genuinely advance the study of ASB, it is critically important to investigate how the socio-cognitive-affective vulnerabilities unfold

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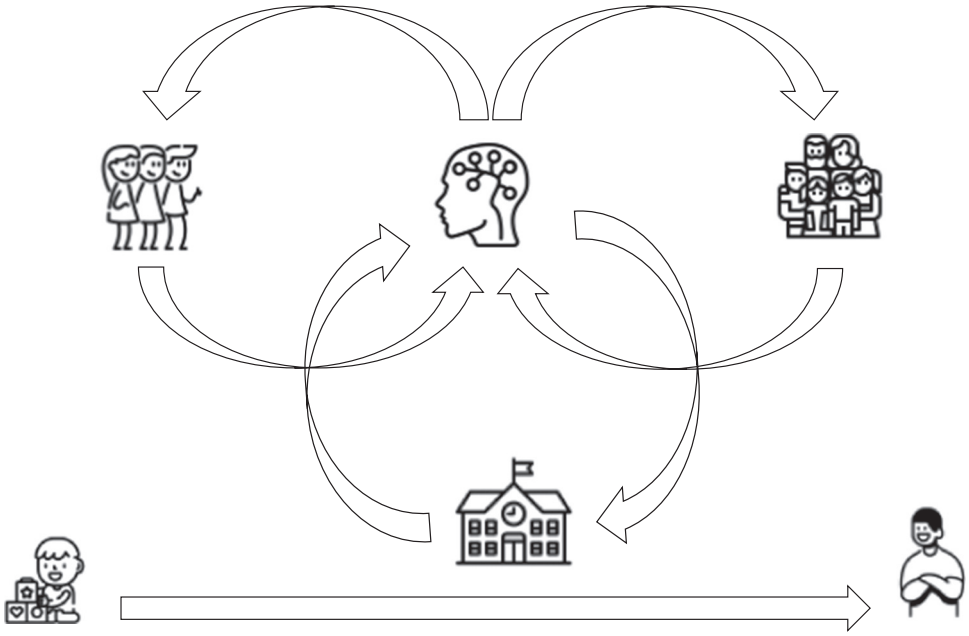

Figure 1. A schematic conceptualisation of 'The Embedded Brain'. The relationships between neurocognitive functioning and the social world are complex. Much of the past research on ASB has either focused on neurocognitive vulnerability, often conceptualised as 'located within the child,' or on social/environmental risk factors, often conceptualised as 'external to the child'. This separation is artificial and unhelpful and hampers our efforts to understand why ASB develops and how it is maintained. We need to adopt a developmental focus where the transactional mechanisms between intra- and extra-individual factors – and how these may differ between different individuals – are studied. We can consider, for example, the interplay between hypervigilance to threat and peer relationships, lack of empathy and family relationships, and poor executive functions and the learning environment. Threat-based reactive aggression is likely to lead to reduced social support from friends over time, which in turn can reinforce the threat bias. Reduced empathy is likely to result in weaker bonds with family members, which can, over time, contribute to poor family relationships and reinforce the lack of common feeling between family members. Poor executive functioning includes poor impulse control, which reduces opportunities to learn at school, which can in turn hamper future executive function development.

in a social context, where the neurodevelopment is not only impacted by the social environment, but where the child's socio-cognitive-affective propensities also shape their social interactions [19]. We term this transactional approach the study of the 'embedded brain' (Figure 1).

In this article, we provide a concise summary of how ASB in children and adolescents is characterised and what we know of its aetiology. We then briefly discuss extant experimental and neuroimaging findings, which suggest difficulties in different aspects of socio-cognitive-affective processing (emotion processing, empathy, perspective-taking, reinforcement learning, and executive functions) for children and adolescents with ASB – although the precise patterns may vary between those affected. The focus of the article will be to consider how particular socio-cognitive-affective processes may shape and be shaped by the social environment and what future research challenges must be navigated to achieve the goal of advancing the study of the 'embedded brain'. We also suggest that this approach can help the development of effective intervention approaches for ASB, beyond what can be achieved by focusing on behaviour, the brain. or the environment alone [19,20].

Characterising clinically concerning levels of ASB

Children and adolescents with persistent and concerning levels of ASB qualify for a diagnosis of conduct disorder (Box 1). However, because of referral biases and lack of clinical services, many

Box 1. Conduct disorder

Children and adolescents with significant levels of ASB may meet clinical threshold for diagnosis of conduct disorder, which is described as a repetitive and persistent pattern of ASB in which the basic rights of others or major age-appropriate societal norms or rules are repeatedly violated[ii]. These behaviours fall into four main groupings: aggression to people or animals, destruction of property, deceitfulness or theft, and serious rule violations [55,86]. There are 15 symptoms included in these four clusters that can vary greatly from serious aggression (e.g., 'has used a weapon that can cause serious physical harm to others') to violations of societal norms for behaviour expected of a certain age that do not cause great harm to others (e.g., 'often stays out at night despite parental prohibitions, beginning before age 13'). Thus, the definition of conduct disorder includes a number of requirements to restrict it to only those who show a serious and impairing pattern of ASB. To meet criteria for a diagnosis of conduct disorder, the person must show at least three of the 15 criteria behaviours during the past 12 months, (with at least one behaviour present in the past 6 months) and the behaviour must also cause clinically significant impairment in social, academic, or occupational functioning.

Even with these criteria, the severity of conduct disorder can still vary greatly across individuals, depending on the number of symptoms and how much harm the behaviours cause to others. Thus, the definition of conduct disorder includes a number of nonorthogonal specifiers to further capture some of this heterogeneity, including severity (based on number of symptoms and degree of harm to others), age of onset of the symptoms (childhood-onset type; adolescent-onset type; unspecified onset), and whether the conduct disorder symptoms are displayed with the presence of callous-unemotional (CU) traits ('limited prosocial emotions' specifier). CU traits relate to severity of ASB symptoms and also predict differences in outcome and presentation of ASB that suggest differences in the causal processes leading to conduct disorder, not captured by focusing on severity of ASB symptoms alone [55,86].

children with conduct disorder never receive a formal diagnosis. Therefore, the studies we discuss here focus both on children and young people with a formal conduct disorder diagnosis, as well as those with clinically concerning levels of ASB, as defined by standardised assessment instruments used by researchers to document conduct disorder symptoms. The umbrella term ASB will be used throughout when we talk about these studies.

The aetiology of ASB is complex, reflecting the influence of multiple genetic and environmental risk factors, as well as their interplay (Figure 2 and Box 2). Furthermore, not all children and adolescents with ASB are the same. They vary in their profile of risk factors (genetic and environmental), the age of onset (early childhood vs. adolescence), and the severity of their ASB, as well as in their comorbidities (e.g., attention deficit hyperactivity disorder, anxiety) and temperamental traits [callous-unemotional (CU) traits, irritability, and impulsivity] co-occurring with ASB [21–24]. For example, prospective, longitudinal cohort data clearly show that ASB that has an early onset (prior to adolescence) is more severe and associated with more indicators of atypical neurodevelopment, as well as social risk factors [21]. Children with early-onset ASB also often display comorbidities, which complicates their profile of difficulties and needs [21]. Research over the past 20 years has additionally examined the role of CU traits in explaining differences in the expression of ASB [19,25,26]. Individuals with ASB with high levels of CU traits show diminished empathy and guilt, have difficulty in forming meaningful relationships with others, and often engage in instrumental ASB (where there are clear, pre-meditated goals, such as gaining money or dominance, motivating their behaviour) [21]. Those with ASB and low levels of CU traits typically show empathic responses to others' distress and patterns of aggression that are not pre-meditated, but which have clear external triggers (e.g., threat, insults, frustration) [21].

Socio-cognitive-affective processes associated with ASB in children and adolescents

The harmful and affectively atypical behaviour of children and adolescents with ASB has motivated experimental and neuroimaging work to examine socio-cognitive-affective processes that may be associated with this presentation. Emotion processing, empathy, perspective-taking, executive functions, and reinforcement learning are thought to be critical, interlinked building blocks of successful socialisation [26–28]. For example, theoretical accounts and empirical research suggest that perception of own and other's emotions are key for experiencing empathy;

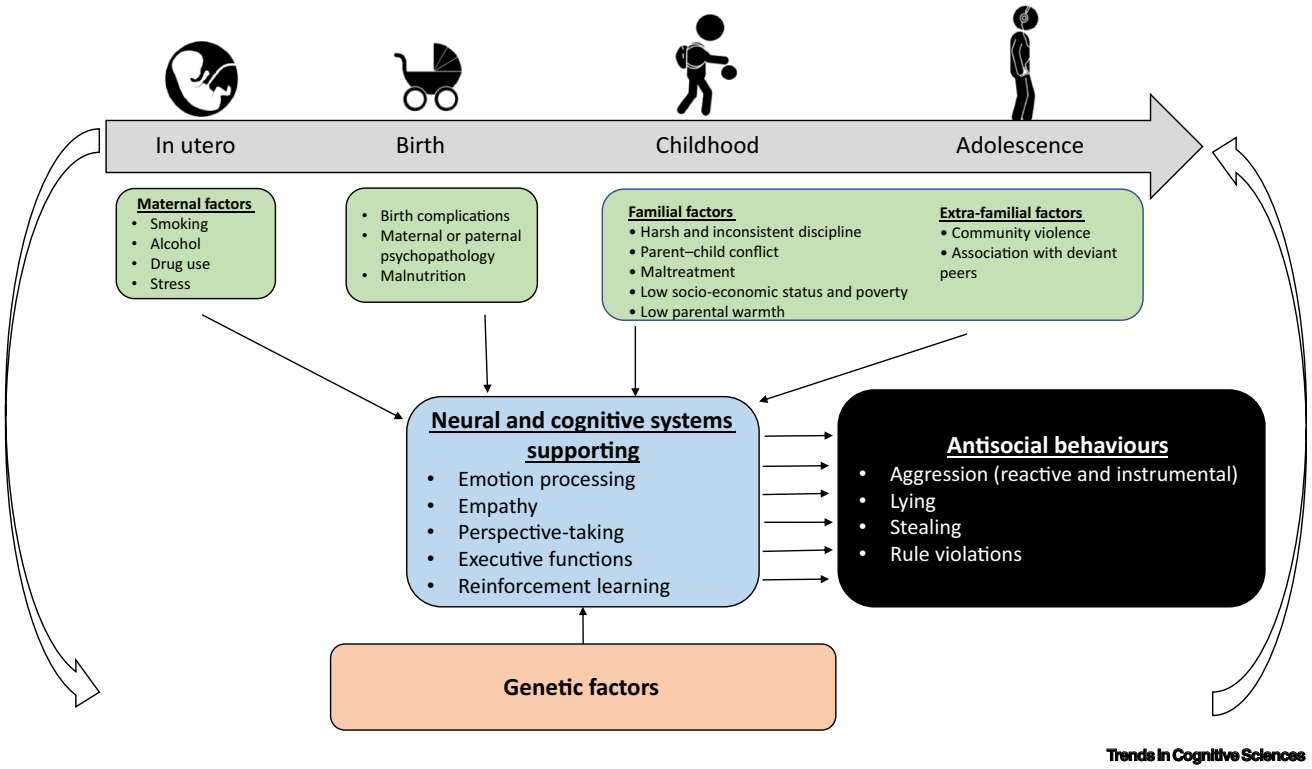

Figure 2. Complex risk factors contributing to the development of antisocial behaviour (ASB). Multiple dispositional (i.e., genetic and neurocognitive) and environmental (in green) risk factors for ASB operate across the lifespan; their hypothesised associations over time are depicted in this figure. The nature and importance of these risk factors varies depending on the developmental stage. For example, genetic risk for emotion processing and regulation (as well as other socio-cognitive-affective processes) may contribute to the risk of early behavioural problems, whereas genetic risk for low empathy could increase the likelihood of engaging in bullying during adolescence. The importance of environmental risk factors also varies by developmental stage, with low parental warmth contributing to risk behaviours during childhood and ineffective parental monitoring becoming more important during adolescence. Many of the dispositional factors also contribute to the generation of environmental risk (gene–environment correlation), as well as to susceptibility to environmental risk (gene–environment interactions). The challenge for the field is to use innovative study designs to improve our ability to integrate across multiple levels of analysis as we try to understand why ASB develops.

and empathy is in turn important for development of conscience and for moral socialisation [27,29,30]. How emotions are perceived, what intentions are attributed to another person, and how emotions are regulated is also critical for shaping our social interactions [19,30]. From an early age, harmonious social interactions in a changeable environment require good executive functions to support the control of emotions, thoughts, and behaviour [31,32]. Finally, reinforcement learning is thought to be vital for learning consequences of one's actions and for learning to behave in accordance with social norms [33].

Decades of well-replicated cross-sectional experimental studies demonstrate that children and adolescents with ASB present with atypical patterns of emotion processing, empathy, perspective-taking, executive functions, and reinforcement learning [21,34–36]. First, in terms of emotions, experimental studies document differences between children and young people with ASB and typically developing peers in recognition and attention to fear most consistently [37], but also other negative emotions (sadness and anger) [37,38] and happiness [38,39]. Second, a number of studies also report attenuated empathic processing for other people's distress and happiness in children and adolescents with ASB [21,40,41]. Third, there appear to be some differences between ASB and typically developing peers in terms of perspective-taking (sometimes called cognitive empathy), but the picture is complex. When assessed with

Box 2. Aetiology of ASB

In recent decades, we have made some progress understanding the aetiology of ASB, and the role played by genes and environment (see Figure 1 in main text). Twin and adoption studies of children and adults have shown that individual differences in ASB are heritable (estimates varying between 40% and 50%) [87,88]. There is some evidence that the heritability of ASB is particularly high for those whose ASB is early onset or coupled with CU traits [89,90]. There is also some evidence that different genetic risk factors are important for the initial risk versus the developmental course of ASB [91]. The molecular genetic research has implicated the serotonergic, dopaminergic, and neuroendocrine pathways [87], but the largest genome-wide association studies of ASB have not confirmed the associations found in candidate gene studies [92,93]. Multisite studies with bigger sample sizes are required to make advances in understanding genetic risk for ASB in its different forms.

A number of risk factors that we traditionally view as environmental have been associated with ASB. These include prenatal (maternal smoking, alcohol use, drug use, and stress during pregnancy) [94–98], perinatal (obstetric complications, parental psychopathology, malnutrition, and exposure to heavy metals) [99–103], familial (maladaptive parenting, including harsh, coercive, and inconsistent discipline, and childhood maltreatment) [104,105], and neighbourhood (deviant peers, low socioeconomic status, poverty and community violence) factors [106–108]. Genes and environments do not, of course, act in isolation. Instead, their complex interplay shapes how development canalizes over time.

There is good evidence that genes influence exposure to environments (gene–environment correlation), and that individuals with particular genetic risk factors react more strongly to specific environmental risk factors, contingent on their genotype (gene–environment interaction) [109]. For example, parents with genetic variants that predispose to ASB are more likely to engage in negative and harmful parenting practices and may also pass on some of these genetic variants to their offspring; the association between dysfunctional parenting and child's ASB may thus partly represent a genetic confound [4]. Children also evoke different reactions in people around them or actively seek particular environments, in part contingent of their genotypes [4,110]. These are examples of gene-environment correlation. As for gene–environment interaction, adoption studies have shown that the effect of parental risks (having an adoptive parent who is a criminal) on the development ASB was larger in genetically vulnerable individuals (i.e., children with criminal biological parent) than in adoptees without a genetic predisposition, while warm parenting in the adoptive mother buffered the effects of heritable risk for ASB and CU traits [111–113]. Given the heterogeneity among children and adolescents with ASB, it is reasonable to speculate that the gene–environment interplay will look different for different children with ASB.

experimental tasks that do not require perspective-taking about emotions, children and adolescents with ASB do not appear to differ from their typically developing peers [42–48]. However, difficulties can emerge when they are asked to perspective-take about emotions, particularly when these are embedded in a complex context (e.g., movies) or the judgement itself is of complex nature (e.g., second-order affective evaluations) [43]. Fourth, in terms of executive functions, three meta-analyses across pre-school [35], child [36] and adolescent [34] samples have shown that individuals with ASB exhibit a moderate impairment in overall executive functions, with two of those reporting a moderate effect for inhibition [35,36], while one found small effects for working memory and cognitive flexibility [35]. Finally, there is growing evidence showing that the impaired reinforcement learning observed in children and adolescents with ASB may stem from difficulties in accurately assessing the value of prospective behavioural outcomes, including punishment. Indeed, several studies show that this population struggle to learn how to avoid choices that lead to punishment rather than reward [38,49–51].

Functional neuroimaging studies have also contributed to our understanding of emotion processing, empathy, perspective-taking, executive functions, and reinforcement learning in children and adolescents with ASB. These studies have investigated the processing of affective information by engaging participants in tasks that involve observing emotional images, facial expressions of emotions, or stimuli designed to induce empathy (e.g., hands in painful situations). A meta-analysis that included 24 studies reported reduced activation in the dorsal and rostral anterior cingulate cortex (ACC), medial prefrontal cortex, and ventral striatum in young people with ASB [52], while another meta-analysis showed that this population also exhibits decreased response in the amygdala and striatum during emotion processing or reinforcement-related tasks [53]. Meta-analyses of structural brain imaging data have consistently reported reduced grey matter volume in an overlapping set of cortical (i.e., ventrolateral, medial prefrontal, middle temporal, superior temporal, and anterior insular cortices) and subcortical regions (i.e., amygdala, caudate, and putamen) [21].

Notably, the direction of effects in some of the socio-cognitive-affective processes related to ASB may differ depending on the age of onset or levels of CU traits [8,21,54]. For example, the executive function deficits appear most pronounced in those individuals whose ASB starts early and persist across the life course, compared with those whose ASB is limited to adolescence [55–57]. As another example, there is evidence that emotion processing may be different between those with high and low levels of CU traits. Several neuroimaging studies show reduced amygdala activity to emotional faces, in particular fear, in those with ASB and high CU traits [54] while ASB in children and adolescents with low levels CU traits may be associated with increased amygdala activity when processing fearful/emotional faces [58–61].

'Embedded brain': ASB as an emergent, transactional phenomenon

Current cross-sectional experimental and fMRI research focusing on static 'snapshots' of socio-cognitive-affective functioning associated with ASB at a group level offers a limited understanding of how different information processing patterns may confer risk for developing ASB. These studies fail to capture the dynamic interplay between social environmental factors associated with ASB (including family, peers, and neighbourhood) and brain development. We need to gain new information regarding how particular information processing biases may shape the social interactions of children with ASB, as well as how a child's social context can influence their socio-cognitive-affective functioning [19,28].

Children and adolescents with ASB play a key role in co-creating aspects of their social environment. Their pattern of information processing can influence how they interpret their social environment and the social environmental conditions may evoke particular demands on socio-cognitive-affective functioning [19,28,62]. Advancing the study of the 'embedded brain', which considers the transactional interactions between person and environment, is essential if we want to improve our understanding of how information processing differences contribute to formation and maintenance of social relationships.

We illustrate two hypothetical transactional cascades that may result from different patterns of socio-cognitive-affective vulnerability. The first involves a child who, from an early age, shows disrupted processing of other people's distress and difficulties in associating their actions with punishment outcomes – features that have been linked with ASB and high levels of CU traits in prior studies. When this child's parent tries to engage typical socialisation tools – empathy induction (making children understand how their behaviour makes others feel) and sanctions (punishing behaviour that is not desired) – their child simply does not respond in an expected way. The child does not get aroused by other people's distress, and as a consequence, has fewer opportunities to learn about emotions over development and does not respond to empathy induction. If the child also fails to make a reliable connection between actions and consequences, they will have a harder time learning social norms and are more liable to get angry and frustrated when things do not turn out as anticipated. Over time, a child with such processing difficulties fails to develop typical levels of empathy and does not internalise social norms. This can lead to repeated conflictual interactions and callous behaviours that leave the parent feeling exhausted and frightened, causes teachers to write off the child as a troublemaker with no hope of character reform, and either alienates peers or leads to dysfunctional relationships (e.g., peers are manipulated to meet the selfish needs of the child).

The second example of a transactional cascade relates to a child who may have experienced maltreatment earlier in development (for example, physical abuse or exposure to domestic violence) [63]. Such early adverse experiences are thought to lead to calibration of threat circuitry leading to a pattern of hypervigilance [64]. It has been postulated that hypervigilance, often termed as hostile attribution bias [62,65], compromises interpersonal functioning, leading to both heightened

risk of conflictual interactions and peer rejection. For example, normative experiences of being teased or pressured by peers may elicit excessive threat appraisals and poorly judged responses that simply serve to exacerbate conflict – and aggression – with peers. Repeated or serious displays of aggression will increase the risk of exclusion from school which could lead to a cascade of further social stress. Moreover, we know that repeated displays of reactive aggression in adolescents are associated with reduced popularity [66], and this may contribute in turn to an increased risk of peer rejection and social anxiety. In other words, altered threat processing can contribute to increases in interpersonal stress and a reduction in the number and quality of social supports among both peers and adults. These phenomena have been referred to as stress generation and social thinning, respectively [64]. Over time, such poorly optimised social responses – driven by heightened threat reactivity – create a cascade of maladaptive social interactions that accrue costs in terms of a child's ability to cultivate and maintain social relationships with others [64].

These two examples highlight the importance of measuring socio-cognitive-affective processes, as well as multiple aspects of children's and adolescent's environments to achieve a better understanding of the range of transactional pathways that can lead to ASB. By doing so, we are poised to improve our understanding of why social interaction and socialisation can derail. However, these examples focus on proximal relational influences. It is also important to consider the impact of and individual response to more distal/structural factors, including intergenerational poverty, racism, and community violence. By assessing the intersection of the person and their environment, we can improve the personalisation of interventions. Before the field can genuinely move forward with this endeavour, there are several challenges that need to be considered.

Challenges and opportunities for the study of the 'embedded brain'

To advance longitudinal, multimethod study of the 'embedded brain', we need to consider how we characterise our samples and improve our study protocols, with particular attention to the challenges posed by studying development. Here, we will discuss how we can build on extant psychological and neuroscience research to advance theoretical and empirical understanding of ASB in children and adolescents.

Studies to date have shown that children and adolescents with ASB display atypical socio-cognitive-affective processing. The precise nature of atypical processing varies between individuals with ASB, for example, depending on whether their ASB is early or late onset, or accompanied with high or low levels of CU traits or irritability [8,21,67]. However, between study differences in how samples are recruited, differences in approaches and instruments that are used to measure ASB, its onset and accompanying traits, and differences in cut-offs used to group participants make it challenging to interpret any inconsistencies in the current data. We provide a few illustrations from the current literature to highlight these challenges.

First, it is important to note that for some children and adolescents, ASB and CU traits may arise as a consequence of childhood maltreatment and are accompanied by anxiety symptoms [68–70]. These children's experimental task performance and psychophysiological responding are more closely related to what is typically seen in individuals with anxiety disorders, than those whose ASB and high CU traits do not occur in the context of the maltreatment [68,70]. As the extant studies have not systematically measured maltreatment histories of their participants with ASB and high CU traits, it is currently not possible to evaluate the contribution of maltreatment experience on study findings or to explaining mixed findings between studies. Second, it is interesting to note that studies that have examined the role of CU traits in socio-cognitive-affective processing in ASB tend to report different findings depending on whether CU traits are self-rated (CU traits not found to moderate findings [40,71]) or other-rated (CU traits related to diminished behavioural and

neural responses to distress and pain [26,27,72]) [73]. Third, when prospective longitudinal data are used to characterise early- versus adolescent-onset ASB, clear differences are seen between the two groups in their neuropsychological functioning and brain structure [55,56,74]. Furthermore, the early-onset group differs from typically developing peers in a much more pronounced way than their adolescent-onset peers with ASB [55,56]. When ASB trajectories are characterised using single timepoint retrospective data, the differences between early- and adolescent-onset groups are not as clear [75]. As the cognitive-affective and neural study of ASB moves forward, there should be efforts to standardise measurement and recruitment protocols in ways that makes it easier to compare findings from different studies and increases the reliability of the findings.

It also is worth noting that some of the socio-cognitive-affective differences that we see overlap with those observed in a number of other mental health conditions – in line with the notion of transdiagnostic vulnerability to poor mental health [76]. This is perhaps not surprising, given that heterotypic continuity and comorbidities are the norm in the development of psychopathology, including ASB [2,21,77]. From a research design perspective, we should invest in longitudinal studies that enable us to distinguish between problems that contribute to the emergence of ASB (e.g., problems in empathy leading to reduced threshold for instrumental aggression) from those that may represent consequences of these behaviour problems (e.g., anxiety and distress over the consequences of the behaviour problems). Such studies can also help us understand distinct trajectories of ASB and must incorporate multiple levels of analysis to identify which factors lead to ASB, and at which developmental stage they exert their influence. To progress the study of the 'embedded brain', we will also need to improve the measurement of socio-cognitive-affective processes (see Box 3 for challenges and opportunities with respect to this).

Improving our ability to study the 'embedded brain' is a challenging, but an important, task with clear implications for translation. Early emerging ASB is a risk marker not just for persistent ASB, but also for a host of other mental and physical health conditions, as well as for problems in social, educational, occupational, and legal domains [2,3]. Therefore, providing timely interventions is critical in terms of lifelong outcomes and associated societal costs. Neurocognitive findings can have a useful role to play in informing interventions.

Translational potential of psychological and neuroscience research into ASB

Better understanding of different patterns of socio-cognitive-affective vulnerability associated with ASB can be informative for personalising prevention programmes that address behaviour that may not yet have precipitated clinical referral, as well as treatment of those diagnosed with clinical levels of ASB (e.g., conduct disorder) [54,78].

Current interventions are often targeted at improving parent education and skills for child and adolescent behaviour management [20,79]. In this case, interventions are undertaken to make improvements in key social environmental areas but often do not provide children and adolescents with new scripts to interpret these changes or with skills to take advantage of opportunities. By contrast, interventions that focus on socio-cognitive-affective factors provide strategies for addressing problematic cognitions, managing emotions, and modifying ASBs. However, information processing and behavioural patterns are learned and reinforced through experiences with the environment. Therefore, participation in these interventions becomes a tug of war in which children and adolescents are expected to accomplish information processing and behavioural change without regard for the social environmental factors that vie to pull them back into the very social environments that put them at risk for ASB. Thus, although modifications in socio-cognitive-affective functioning within the person are necessary, they may be insufficient to produce lasting change (particularly if the child or adolescent themselves does not experience

Box 3. Improving measurement of socio-cognitive-affective processes

Currently, task parameters and task demands can vary considerably between studies, even when they claim to assess the same processes [19] (or worse still, questionnaire and task measures are assumed to capture the same construct, although they are typically poorly correlated [114]). Some agreement on core measures for particular constructs under study would enable better comparison between studies and contribute to more meaningful meta-analyses as data accumulate. These could include commonly agreed measures of emotion processing and empathy, as well as a set of reinforcement learning and executive function paradigms shared between laboratories. Before such measures are ready to be shared between laboratories, we also need more work on psychometric validation of experimental and functional neuroimaging measures, especially if we want to advance longitudinal study of how ASB develops. Current paradigms, for example, emotion recognition and reward learning tasks, stem from an experimental tradition where tasks are typically designed to minimise between individual variation and to reliably capture effects across all humans or within a specific group [115,116]. In other words, these paradigms have not, as a rule, been psychometrically validated to sensitively and reliably capture individual differences [116]. This currently limits their utility for inclusion in large scale, longitudinal studies or in intervention studies, and hampers our ability to relate functional neuroimaging and experimental data to behavioural (including clinical) outcomes [117]. Another challenge relates to the dearth of work validating paradigms that could be used to assess the same processes across the lifespan. For example, the tasks that have been chosen to experimentally index socio-cognitive-affective processing in ASB groups with high versus low levels of CU traits, have varied considerably between studies of younger children and adolescents [72,118]. This means that we do not currently know whether any differences in findings between younger and older age groups are due to developmental effects or due to methodological differences between studies. The studies in younger children typically necessarily include fewer stimuli and different content, than the studies conducted in adolescents. More work is needed to ensure that we can have confidence that the measures employed at different ages are indeed indexing the same underlying cognitive process and do so reliably. This would require testing age-adapted measures of the same constructs over time and studying their associations with other measures, and again promoting cross-lab collaboration to ensure that common measures and tasks are employed across samples. Furthermore, as developmental researchers, we must grapple with the reality that such processes themselves will, in almost all instances, evolve and change over time.

It is not sufficient to just pay attention to the experimental and neuroimaging paradigms that we use. We will also need sensitive measurement of social functioning over time (e.g., via observational, experience sampling, and social network measures) [119,120]. In this way, we will be better positioned to study how socio-cognitive-affective processing impacts the formation and maintenance of social relationships at different developmental stages, and how social relationships shape brain and cognitive development in ways that either increase the risk of ASB or promote more prosocial outcomes.

distress and lacks insight into the impact of their behaviour on others). Consistent with the 'embedded brain' concept, some interventions attempt to address the interplay of both social environmental and person-specific factors. These programmes arise from an understanding that children and adolescents bring views with them that are grounded in life experiences shaped by social environmental processes. Such programmes tend to be multisystemic and target structural conditions that are specific to a child or adolescent's environment, expand individual skills, and change the ways youth make sense of and react to their life. Some existing individualised programmes that focus both on the child and the child's psychosocial context are Functional Family Therapy (FFT) [80] and Multi-Systemic Therapy (MST) [81], both of which have proven effective in reducing ASB even in children with elevated CU traits [82]. Here, we suggest that further improvement in the measurement of socio-cognitive-affective functioning can refine the targets for these interventions at both the social environmental and individual levels. Intervention targets could be aimed at: (i) changing information processing styles that confer different types of risk for ASB; (ii) cultivating compensatory strategies that motivate the likelihood of prosocial engagement; and (iii) integrating actors from the social environment into the intervention process.

Given their difficulty with empathising with other people, reduced prosocial motivation and difficulty in learning from punishments, individuals with ASB and high CU traits might particularly benefit from warm and responsive parenting. Unfortunately, their behaviour often evokes the opposite types of responses from parents. For these individuals, interventions that reflect a transactional understanding could include empathy training and perspective-taking for the child/adolescent and caregivers, combined with explicit motivational strategies for encouraging prosocial behaviour by caregivers/teachers. One such intervention is an enhancement of a standard parenting intervention [Parent-Child

Interaction Therapy (PCIT)] [83] that integrates such strategies in order to break the maladaptive transactional cycles. There are positive early data suggesting that such skill-building approaches tailored to the specific socio-cognitive-affective vulnerability of the child can increase the effectiveness of the standard intervention [82,84]. These results are quite promising, given that this group of children has often left more traditional treatments with significant and impairing ASB still present [79].

For individuals with low CU traits, interventions aimed at reducing hostile biases, combined with approaches to help the child better manage emotions in interpersonal contexts, consider the consequence of their behaviour, and affiliate with prosocial peers, could be most helpful for targeting the complex socio-cognitive-affective functioning related to this expression of ASB. One example of such a skill-building programme is the Coping Power programme [85] which has been demonstrated to be effective in reducing ASB in many children and may be even more effective than suggested by past research when it is individualised to the specific deficits of the child. In other words, a more precise understanding of the socio-cognitive-affective processes that contribute to a particular child's ASB could help clinical formulation by fostering individualised and tailored interventions that take account of and target specific patterns of socio-cognitive-affective processing of a particular child.

Outstanding questions

How does socio-cognitive-affective processing impact formation and maintenance of social relationships at different developmental stages, and how do social relationships shape brain and cognitive development in ways that either increase risk of ASB or promote more prosocial outcomes?

What are the best tasks for measuring socio-cognitive-affective processing associated with ASB across development?

What are the neurodevelopmental (as opposed to cross-sectional neuroimaging) signatures of different types of ASB?

How do socio-cognitive-affective processing features associated with risk for ASB develop over time?

Concluding remarks

Our understanding of ASB is poised for transformation. We are now in a position to integrate insights from experimental, neural, developmental, and social research in ways that allow us to better understand how and why some children grow up to display ASB (see Outstanding questions). To fully capitalise on the translational potential of the experimental and neuroscience research into ASB, longitudinal samples with sensitive measures of behaviour, socio-cognitive-affective processing as well as the environment are needed to capture the complexity of the phenomenon under study. At the heart of this approach is the central notion of the 'embedded brain'. Child and adolescent ASB is not simply located in the brain or the environment. Rather it is the transactional and iterative unfolding of brain and cognitive development (including alterations in socio-cognitive-affective processes) within a relational context that will be the key to understanding the emergence and maintenance of ASB.

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Declaration of interests

No interests are declared.

Resources

[i] https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/732110/the-economic-and-social-costs-of-crime-horr99.pdf

[ii] www.psychiatry.org/File%20Library/Psychiatrists/Practice/DSM/APA_DSM-5-Conduct-Disorder.pdf

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