the brain works when facing such 'adversarial' choices.

Linking directly with biology, there is a large undergoing effort to combine data from many studies with socioeconomic outcomes and genome-wide association data to greatly improve the statistical power needed to identify genuine associations. Economists also use the language of evolutionary selection to describe and explain aspects of human behavior and institutions, but they have not done so with both formal discipline and careful observation. More direct communication with biologists would be useful in such a synthesis.

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HSS 228-77 Caltech, Pasadena CA 91125, USA.
E-mail: camerer@hss.caltech.edu

Psychopathy

Essi Viding[1,2,*], Eamon McCrory[1], and Ana Seara-Cardoso[1]

Psychopathy is a condition that has long captured the public imagination. Newspaper column inches are devoted to murderers with psychopathic features and movies such as *No Country for Old Men* and *We Need to Talk About Kevin* focus on characters who are exceptionally cold and callous. Psychopathy is in fact a personality disorder characterised by lack of empathy and guilt, shallow affect, manipulation of other people and severe, premeditated and violent antisocial behaviour. Individuals with psychopathy generate substantial societal costs both as a direct financial consequence of their offending behaviour and lack of normal participation in working life, but also in terms of the emotional and psychological costs to their victims.

Manifestations of psychopathic traits and behaviours are evident from early childhood and, although it is entirely inappropriate to label children as psychopaths, it is clear that callous-unemotional traits that characterise adults with psychopathy can also be reliably observed in children and predict increased risk for persistent antisocial behaviour. In this primer, we will provide an overview of the research on the manifestation and causes of psychopathy. We will start by describing a 'case history' — an amalgamation of cases we have encountered during our research and clinical careers — that aims to capture what an individual with psychopathy is like. Throughout, we take a life-course perspective describing findings from studies of children at risk for developing psychopathy — those with antisocial behaviour and callous-unemotional traits — and adults with psychopathy.

Mark was the second-born child of Lisa and Tom. Following the unplanned birth of their first child, Tom had to leave college and take a job at the local storage facility. As a result, the family struggled financially and Lisa was often depressed. Lisa recalls that Mark was a difficult baby, often 'screaming in rage' and rarely returning affection for his parents. He did not respond to Lisa's attempts to engage with him, often looking away when Lisa was talking to him. Lisa says that Mark always struck her as very different from his sister. As a toddler, he was frequently violent towards other children, trying to hurt them when he thought grown-ups were not looking. He was also cruel to the family pet and could not be left alone with it. He would deliberately break his big sister's toys and there were many instances when he hit, kicked and bit her very hard. He appeared fearless and immune to any punishment, such as being made to sit on the 'naughty step' to have a time out. He showed little empathy for others and when he was asked to imagine how his behaviour might make other people feel, he simply looked blank.

At school, Mark's problems escalated and he was eventually transferred to a special school for children with behavioural difficulties. This was in sharp contrast to his sister, who did well at school and had many friends. By adolescence, Mark's behaviour at school was characterised by aggression, bullying, blackmail of other boys, attempts to intimidate members of staff and lack of regret for his actions. No sanctions imposed by the school staff seemed to have any effect on Mark. Eventually, Mark started to skip school and by his mid-teens got involved in a number of burglaries and robberies. His parents were not able to monitor his activities, and Mark began to routinely hang out with delinquent peers, but did not seem to have firm friends. Acquaintances seemed to come and go depending on whether they were of use to him. He would often implicate his peers if he was caught doing something in order to save his own skin.

Eventually, in his late teens, he received a prison sentence for a violent robbery. When he was released from prison he continued his criminal lifestyle. He never settled down with a family but had a string of girlfriends, two of whom became pregnant. Mark showed little interest in his children and failed to provide any financial support for them. He was engaged in supplying drugs, was involved in several financial scams, and ended up jailed for a second time for killing

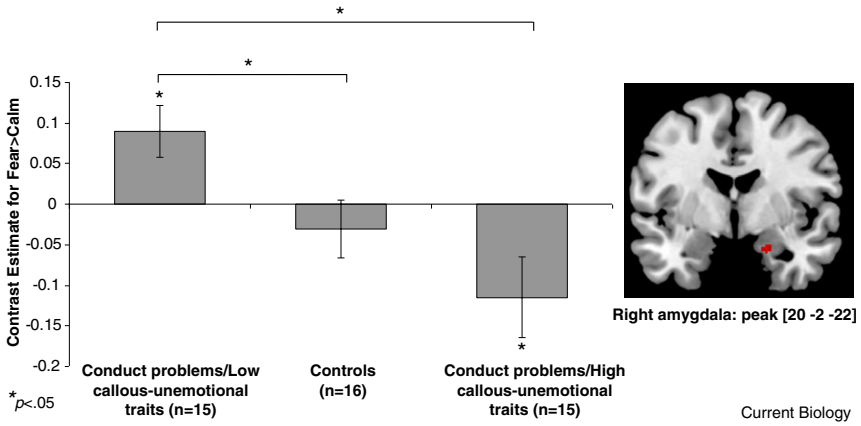

Figure 1. Amygdala response to fearful faces.
Amygdala response to fearful faces (in comparison to calm faces) for boys with conduct problems and low levels of callous-unemotional traits, typically developing comparison boys, and boys with conduct problems and high levels of callous-unemotional traits. The boys with conduct problems and callous-unemotional traits have the weakest amygdala response to fearful faces. (Adapted with permission from Viding *et al.* 2012; copyright ©2012 American Psychiatric Association; all rights reserved.)

his criminal partner following a disagreement about money. As an adult, Mark's prison file was reviewed and he was interviewed by the prison psychologist. The psychologist noted that Mark rarely expressed any guilt or remorse for killing his friend, seemed incapable of feeling empathy, was happy to manipulate other people, did not fulfil his obligations to his children and engaged in a wide variety of criminal behaviours. Although Mark's psychopathic traits have manifested in different ways at different ages, at each point in development his behaviours were striking in so far as they demonstrated a profound lack of empathy or concern for the rights of others.

Prevalence and causes

No epidemiological data exist that directly quantify the prevalence rates of psychopathy across the population; however, people have used data from forensic and clinical samples to estimate that approximately 0.75–1% of the population may be psychopaths. A similar percentage of children present with both severe antisocial behaviour and callous-unemotional features. There are more males than females presenting with these traits, although the precise gender ratio is unclear.

Several twin studies to date have demonstrated that psychopathic personality traits are moderately to highly heritable in children and adults. Furthermore, antisocial behaviour in the presence of psychopathic traits appears more heritable than antisocial behaviour in the absence of psychopathic traits. In short, genetic differences between individuals can explain why some individuals are at increased risk of developing psychopathy. These genetic differences are likely to also encompass differential susceptibility to environmental risk factors between individuals.

Only a handful of molecular genetic investigations have focused on psychopathic traits. These have tentatively implicated variants in the serotonin and oxytocin genes as increasing risk of psychopathy, but the current candidate gene studies need to be replicated in larger samples to evaluate whether they report true associations. Genome-wide association studies of psychopathic traits have not unearthed any genetic variants with a large effect. Studies of gene–gene and gene–environment interactions — which more than likely account for a proportion of the heritability estimate for psychopathic traits — will be important in the future, as will be whole-genome sequencing to detect rare variants.

Genetic research is likely to advance greatly in the coming decade, including studies using novel epigenetic approaches that may help uncover mechanisms of gene–environment interaction, but it is of critical importance to keep in mind that *there are no 'genes for psychopathy'.* Genes code for proteins that influence characteristics such as neurocognitive vulnerabilities that may in turn increase risk for developing psychopathy. Genetic variants that are implicated as risk genes for psychopathy are likely to include several common polymorphisms that confer advantages, as well as disadvantages, depending on the environmental context. The neurocognitive vulnerabilities associated with psychopathy are at least partially distinct from those associated with antisocial behaviour/conduct problems in general (see sections on cognitive and neuroimaging findings). This suggests that the risk polymorphisms for psychopathy may not always be the same as risk polymorphisms for antisocial behaviour in the absence of psychopathic traits.

Because genetic risk may only become manifest in the presence of environmental risk, there is a pressing need for genetically informative studies to collect data on environmental risk factors (including peri-natal and post-natal, as well as social and neighbourhood measures). One recent study reported that the long allele of a serotonin transporter polymorphism, the allele known to confer attenuated emotional reactivity, was associated with increased callous-unemotional traits, but only in those adolescents from low socio-economic status backgrounds. This finding of gene–environment interaction highlights the real possibility that genetic vulnerability to callous-unemotional traits only becomes expressed under unfavourable environmental conditions.

Several risk factors, typically considered to represent 'environmental' risk, such as parenting and parent mental health, have also been studied in relation to psychopathic traits. However, longitudinal, genetically informative studies are required to establish whether these proposed risk factors represent true environmental causal factors. For example, it has been shown that harsh parenting is related to higher levels of psychopathic traits. However, only one longitudinal, genetically informative study has investigated this association, and

findings from this study suggest that the association may be in part accounted for by genetic vulnerability within families — parents who parent harshly may pass on genotypes that predispose to callous-unemotional traits. In other words, the association between parenting and these traits may simply reflect shared genetic risk. Studies of gene–environment correlation, in other words, how individuals' environments are in part shaped by genetic propensities, will be important when seeking to understand how psychopathic traits develop.

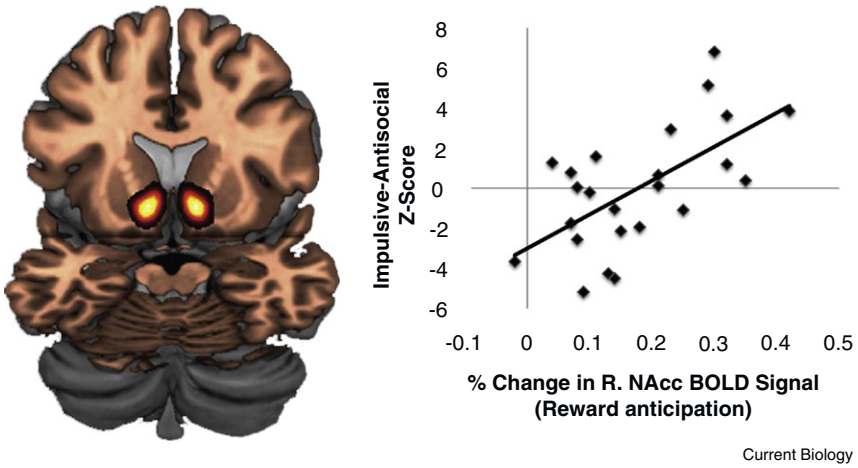

Figure 2. Ventral striatum response during reward anticipation in adults.
Plot shows ventral striatum nucleus accumbens (NAcc) response during reward anticipation as a function of levels of impulsive-antisocial psychopathic traits in adults. (Adapted with permission from Buckholtz *et al.*, Macmillan Publishers Ltd: copyright 2010.)

Cognitive deficits

Behaviourally, individuals with high psychopathic traits show marked lack of empathy and guilt. They often engage in proactive, instrumental aggression, seem impervious to sanctions and do not appear to share the affiliative needs and goals that typically characterise us human beings. Given this behavioural profile, many of the experimental studies on adults with psychopathy and children with callous-unemotional traits have focused on how they process emotions, whether they empathise with others and whether they change their behaviour following punishment.

Experimental studies have, in particular, focussed on the processing of facial cues of emotion. For example, in one well-known paradigm, individuals with psychopathy are asked to observe a facial expression morphing from neutral to an emotional one and are required to name the emotion. Compared with typical individuals or individuals with other forms of psychopathology, those with high levels of psychopathic traits take longer to recognise emotional facial expressions, particularly those indexing distress in other people. Other paradigms have documented blunted empathy towards others, reduced startle responses to negative stimuli and a relative immunity to punishment. Remarkably, however, individuals with psychopathy are perfectly adept in understanding other people's thoughts, in contrast to individuals with autism spectrum disorders. This may help explain why individuals with psychopathy are often so good at manipulating other people — they know what others are thinking, but do not resonate with the distress their actions will inflict on others.

What might be at the root of the psychopath's difficulty in recognising other people's emotions and empathising with them? One possibility is that individuals with psychopathy have attenuated emotional reactions to situations that typically generate fear and sadness in other people; over time, as an individual develops, this is likely to lead to a reduced ability to process and recognise distress cues in other people. Emotional contagion, which is necessary for empathy to occur, develops through repeated pairing of an emotional state with cues to that state in another (expressions, postures, vocalisations). Mothers typically mimic ('mirror back') their infants' emotional expressions when they observe the infant experiencing an emotion. If the infant experiences distress less often than is typical, there will be reduced opportunity for the infant to learn which cues reliably signal distress as their mothers will have fewer opportunities to mirror emotions back to them.

Recent data have also emerged suggesting that children with callous-unemotional traits do not seek out eye contact with their mothers (although the mothers themselves do not differ from mothers of typically developing children in seeking out eye contact with their children). It is unclear what underlies this reduced drive to seek eye contact, but this is likely to further reduce learning opportunities about emotions over development for children with callous-unemotional traits. In other words, over time children with callous-unemotional traits are likely to develop relatively impoverished representations of their own emotions, which in turn makes it more difficult for them to reliably detect and empathise with other people's emotional experience. They cannot effectively 'anchor' other people's displays of distress to a robust representation of the same state in themselves.

The cognitive deficits seen in individuals with psychopathy offer a preliminary explanatory framework for understanding their callous and violent behaviour. Non-psychopathic individuals are inclined to desist carrying out callous and violent behaviours as they find distress in other people aversive. Individuals with psychopathy have an increased propensity for antisocial behaviour, likely because of their diminished experiential understanding of distress, their relative lack of empathy and insensitivity to punishing consequences of their actions.

Neuroimaging studies

Several neuroimaging studies to date have investigated how the brains of those individuals with high levels of psychopathic traits differ from the brains of typical individuals or even of the brains of individuals with antisocial behaviour who do

not have psychopathic traits. These studies have used many different types of tasks and have focused on criminal populations, individuals from the community and children with varying levels of callous-unemotional traits. The emerging evidence suggests that individuals with high levels of psychopathic traits show lower activity in a number of brain areas, including the amygdala and anterior insula, when processing emotional/empathy inducing/salient stimuli. Most notably, individuals with high levels of psychopathic traits are different from other individuals with antisocial behaviour who do not show this atypically low brain activation to such stimuli (Figure 1).

Research also implicates an aberrant neural response in regions typically associated with reward processing, cognitive control and emotional regulation, including the orbitofrontal cortex and striatum, when individuals with high levels of psychopathic traits perform tasks involving decision-making and reward (Figure 2). It is not entirely clear, however, whether aberrant neural responses during decision-making and reward are unique to individuals with high levels of psychopathic traits or whether such response patterns are shared with other individuals with antisocial behaviour. Atypical activity in brain areas such as orbitofrontal cortex and striatum during decision-making and reward processing gives us clues about why individuals with high levels of psychopathic traits do not plan ahead, make poor, impulsive decisions, and seem to be so driven by the potential of immediate reward to themselves.

The findings from the neuroimaging literature fit with the cognitive-experimental data on psychopathy and provide clues regarding the neural basis of the disorder. The current data suggest that although individuals with psychopathy typically have an attenuated brain response in regions implicated in affective processing, we also now know that the degree of the brain response may depend on the precise content of the task and the kind of instructions given to the participants. In other words, it appears that individuals with high levels of psychopathic traits are not entirely 'emotionally flat', but they seem to be considerably less likely to spontaneously respond to other people's distress.

Future outlook

The precise causal mechanisms leading to the development of psychopathy are still not understood. By combining information across different levels of analysis, it is possible to gain a more complete picture of how and why psychopathy develops and how affected individuals may be helped to change. Research in the coming decades will no doubt shed more light onto the genetic variants associated with psychopathy, as well as isolate the most potent environmental risk factors associated with the disorder. A longitudinal, genetically informative approach is our best chance of understanding how the developmental vulnerability to psychopathy unfolds.

From a clinical perspective, the challenge for the field is now to translate the emerging body of basic research into more sophisticated and tailored approaches to intervention. We already know that interventions can be effective with children and adults with callous and unemotional traits, but the clinical outcomes remain modest. Experimental psychology and neuroimaging work indicate that individuals with psychopathy have an attenuated response to other people's distress and do not readily learn from punishment. These basic science findings suggest that some socialization techniques, such as empathy induction and punishment, may have limited success with those who suffer from psychopathy.

Further research determining the degree of malleability in how individuals with psychopathy process affective information, as well as possible compensatory cognitive-affective functions that could be harnessed to scaffold any atypical processing, will be important next steps in informing how traditional programmes of intervention can be modified and enhanced. Furthermore, we are now better placed than ever before to identify those young people at most risk of developing psychopathy in adulthood and providing the support and intervention needed to reduce their risk of future antisocial behaviour. Such an approach would, in our view, move beyond the notion of conduct disorder as a homogenous category, and help tailor interventions to children in ways that are matched with their developmental vulnerabilities. There is every reason to be optimistic in this regard, but it will require a step-change in how basic science and clinical practice researchers collaborate if we are to make genuine progress.

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[1]Division of Psychology and Language Sciences, University College London, 26 Bedford Way, London WC1H 0AL, UK. [2]Institute of Cognitive Neuroscience, University College London, 17 Queen Square, London WC1N 3AR, UK.
*E-mail: e.viding@ucl.ac.uk