Psychopathic traits in young children

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Callous and unemotional (CU) traits provide researchers and clinicians with an additional dimension when assessing children displaying early-onset conduct problems. Evidence from genetic, brain and cognitive studies to date suggests that antisocial children with psychopathic personality traits are genetically more vulnerable to antisocial behaviour than their antisocial peers. Neuroimaging studies in adults with psychopathy have demonstrated amygdala hyporeactivity to emotional stimuli, while there is some suggestion that adults with antisocial behaviour but no psychopathy may show the opposite pattern. Child neuroimaging data in this area are still thin on the ground; however, behavioural data support the hypothesis that antisocial children with callous-unemotional traits may have some amygdala abnormality. When compared with each other, antisocial children with callous-unemotional traits demonstrate hyposensitivity to others' distress, while other antisocial children appear hypersensitive to anger directed towards them. New research combining different levels of analyses will no doubt provide further insight about the distinct developmental patterns associated with psychopathy, and help to inform methods of intervention, allowing treatment of antisocial behaviour to be targeted according to whether elevated levels of CU traits are present. (*Netherlands Journal of Psychology,* 63, 117-125.)

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Psychopaths create major costs for society, both in terms of their criminality and emotional cost to the people who cross their paths. Although they form only a minority of the prison population (approximately 15 to 30%), they commit a disproportionate amount of crimes (50% more than nonpsychopathic criminals), are more likely to commit a violent offence, and more likely to commit a wide variety of offences (Hart & Hare, 1997). Two rather different approaches

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for defining psychopathy exist. The personality-based approach views psychopathy as a specific form of antisocial personality disorder (APD) defined by a distinctive conjunction of affective and interpersonal features as well as overt antisocial behaviour. Cleckley (1976) first proposed the classic definition of psychopathy as a constellation of deviant personality traits such as lack of empathy, lack of guilt, shallow affect and manipulation of others. Building on this tradition, Hare developed a diagnostic instrument, the Psychopathy Checklist Revised (Hare, 1991), which indexes not just the extreme antisocial behaviour seen in psychopaths, but also the above-mentioned personality markers at the core of the disorder. The PCL-R is by far the most commonly used instrument to assess psychopathy in the prison setting and has both good reliability and validity. Youth and screening versions of PCL have additionally been developed for use in incarcerated populations (Forth, Kosson & Hare, 2003; Hart, Cox & Hare, 1995). Instruments also exist for self-report of psychopathic traits in both prison and wider populations (e.g. Levenson's Self-Report Psychopathy Scale (LSRP; Levenson, Kiehl & Fitzpatrick, 1995), Psychopathic Personality Inventory (PPI; Lilienfeld & Andrews, 1996) and Youth Psychopathic Traits Inventory (YPTI; Andershed, Gustafson, Kerr & Stattin, 2002). Hare and colleagues emphasise that an adequate diagnosis of psychopathy must be based on the full range of relevant symptomatology, including the personality component (Hart & Hare, 1997).

Others have argued that psychopathy should be operationalised in terms of a history of chronic antisocial behaviours, which do not require a rater to infer personality variables from behaviour (e.g. Cloninger, 1978; Spitzer, Endicott & Robins, 1975). However, a focus on the behavioural symptoms, such as irresponsibility and delinquency, to the exclusion of inferred interpersonal and affective symptoms, such as callousness and shallow affect, could lead to the overdiagnosis of psychopathy in criminal populations and underdiagnosis in non-criminals. The distinct pattern of offending (e.g. more premeditated crime and recidivism) seen in psychopaths defined by the personality-based approach, as well as the growing body of evidence for a characteristic neurocognitive profile attached to psychopaths as diagnosed in this way, attest to the success of this approach to defining psychopathic personality disorder. Knowing about the interpersonal features of psychopathy also has relevance for decisions about developing and dispensing treatment.

The findings from the adult psychopathy literature appear to find resonance in research on children with psychopathic tendencies. Frick and colleagues have highlighted the importance of charting psychopathic personality markers (callous and unemotional traits) in childhood (Frick & Marsee, 2006). The Antisocial Process Screening Device (APSD; Frick & Hare, 2001) was designed to extend assessment of psychopathy to children, with the view that the callous and unemotional (CU)[1] traits would mark one important risk factor for life course persistent antisocial behaviour. Callous and unemotional traits include such characteristics as lack of guilt and empathy, which are also considered primary in clinical descriptions of adult psychopathy (Hare & Neumann, 2005). Like PCL-R, APSD indexes CU traits at the core of psychopathic personality as well as overt antisocial acts. Children with psychopathic (CU) traits show a specific behavioural and neurocognitive profile that is similar to the profile found in adult psychopaths. This finding raises the possibility that psychopathy may be a developmental disorder with particular personality markers that can be delineated successfully in children (Blair, Peschardt, Budhani, Mitchell & Pine, 2006). Data from our own group indicate that antisocial behaviour in children with elevated levels of CU traits (AB/CU+) is strongly heritable in childhood, suggesting that children with CU traits may be particularly genetically vulnerable to antisocial behaviour (Viding, Blair, Moffitt & Plomin, 2005; Viding, Jones, Frick, Moffit & Plomin, in press).

This article will selectively review work on the development of psychopathy that has relied on the personality-based approach for the characterisation of the disorder. We will compare and contrast data from individuals with psychopathy with those with antisocial behaviour, but without psychopathy. Various different levels of analyses from behavioural to genetic will be focussed on in this review.

Behaviour

In a longitudinal study conducted a few years ago, CU traits emerged alongside depression and marijuana use as the strongest predictor of later antisocial behaviour (Loeber, Burke & Lahey, 2002). The available evidence indicates that CU traits index a relatively stable characteristic that predicts future antisocial behaviour and particularly poor outcome (Forth et al., 2003; Frick & Marsee, 2006; Frick, Stickle, Dandreaux, Farrell & Kimonis, 2005).

Frick et al. (2005) followed up a group of children from a community sample who were displaying elevated levels of antisocial behaviour (AB) with and without CU traits (ABCU+ and AB/CU- respectively). At each of the four annual follow-up assessments, the AB/CU+ group

1 Please note that we use the terms CU and psychopathic personality interchangeably in this review.

showed the highest rates of conduct problems, delinquency and police contact. In fact, this group accounted for at least half of all police contact for the sample in the last three annual assessment points. Furthermore, AB/CU+ delinquency was not limited to aggressive acts, but also showed the highest levels of most types of delinquent behaviour (e.g. substance misuse and property offences). In contrast the children who were initially designated to the AB/CU- group were indistinguishable from controls on the trajectory of self-reported delinquency. Although the AB/CU- group did show elevated levels of aggressive conduct problems compared with controls, they were less severe than the AB/CU+ group. AB/CU- children showed a significant increase in police contact only at the last time point of the study. Although it was not possible to infer a trend from this one time point, it may indicate that AB/CU- children begin their involvement in criminal activities later than their AB/CU+ counterparts.

The presence of CU traits has also been shown to be associated with aggression and attitude to punishment. Pardini, Lochman and Frick (2003) demonstrated that a group of AB/CU+ adolescents were more likely to focus on the positive aspects of aggression (i.e. rewards, social dominance) and less likely to be concerned with the negative consequences of committing antisocial acts (i.e. subsequent punishment following the transgression) than their AB/CU- peers. These findings held even after controlling for delinquency severity, cognitive ability and demographic characteristics. In contrast, AB/CU- traits are associated with hostile attribution biases; that is, these children tend to interpret ambiguous situations as being hostile, and respond aggressively (Crick & Dodge, 1994, Frick et al., 2003). Furthermore, children with AB/CU- also tend to get distressed about the consequences of their antisocial behaviour (Barry, Frick, DeShazo, McCoy, Ellis & Loney, 2000).

Research investigating the effect of parental characteristics contrasting AB/CU+ and AB/CU- groups is scarce. However, two studies have suggested that antisocial behaviour in AB/CU+ children may be less strongly associated with poor parental practices than it is for AB/CU- children (Oxford, Cavell, & Hughes, 2003; Wootton, Frick, Shelton & Silverthorn, 1997). In addition, Hawes and Dadds (2005) have demonstrated that the use of 'time-out' as a method of behaviour modification is less effective in those children with AB/CU+ as compared with AB/CU-.

In summary, CU traits can be used to distinguish two different subtypes of conduct problems at a behavioural level. AB/CU+ is associated with a poorer long-term outcome (Frick et al., 2005), increased severity of antisocial behaviour (Dadds, Fraser, Frost & Hawes, 2005) and decreased focus on and response to punishment (Pardini et al., 2003; Hawes & Dadds, 2005). AB/CU- is associated with less severe conduct problems, a more favourable response to discipline, and distress at the consequences of one's own antisocial actions.

Cognitive profile in AB/CU+ and AB/CU-

Although all children with early onset AB may engage in particular antisocial acts, there are also transgressions and behavioural patterns that differentiate between the AB/CU+ and AB/CU- subgroups. Just as we can differentiate children with AB/CU+ and AB/CU- at the behavioural level, we can also see some differences in terms of the cognitive-affective difficulties these children experience.

The cognitive deficit associated with psychopathic antisocial behaviour is postulated to be related to a reduction in the salience of punishment information (see Blair, 2006, for a causal model of this sub-type of antisocial behaviour). Blair's Integrated Emotion Systems (IES) model works on the assumption that children with AB/CU+ have diminished ability to form stimulus-punishment associations. In childhood, the ability to be able to form associations between moral transgressions and the aversive outcome (e.g. other's distress) is vital for successful socialisation. Individuals with psychopathic traits find the distress cues in others less aversive and therefore are less likely to learn to avoid actions that bring about a negative response. In addition socialisation by punishing consequences also relies on ability to form stimulus-punishment associations. Children with AB/CU+ are poor at performing tasks relying on stimulus-punishment learning. In contrast to AB/CU+ children, Blair et al. (2006) propose that AB/CU- children have elevated levels of anxiety and threat-related reactive aggression and show hyperreactivity to threat, e.g. angry faces (Pollak & Sinha, 2002). In line with this suggestion, Viding and Frith (2006) recently proposed a causal model where at the cognitive level AB/CU- children have an over-reactive emotional intent encoder, so these children are hyper-reactive to others' displays of negative emotions. Also at the cognitive level, AB/CU- children possess an emotional memory database of maltreatment and hostility and in combination these cognitive vulnerabilities will result in a fight response bias. This fight response bias is thought to be triggered in response to acute environmental stressors and to result in reactive aggression, impulsive violence and increased propensity to make hostile attributions to ambiguous situations.

To date, there are only few direct comparisons of the cognitive profile of AB/CU+ and AB/CU- children. Frick et al. (2003) compared groups of

non-referred AB/CU+ and AB/CU- children and found poor processing of punishment information in AB/CU+ and a hostile attribution bias in AB/CU-. Differences have also been observed in emotional reactivity (Loney, Frick, Clements, Ellis & Kerlin, 2003). Loney et al. (2003) demonstrated a slower recognition rate for negative emotional words in AB/CU+ adolescents, compared with a faster recognition time for the same words in an AB/CU- group. Finally, Dadds et al. (2006) report that CU traits are uniquely related to poor recognition of fearful expressions, while AB/CU- children tended to be hypersensitive to hostility. In addition, a large body of research by Blair and colleagues has repeatedly demonstrated deficits in processing fear, sadness and punishment in AB/CU+ individuals, as compared with institutionalised (although not specifically AB/CU-) controls (see Blair et al., 2006 for a review).

'Antisocial' brains

Individual differences in several brain areas and cognitive functions associated with perception and regulation of emotions have been found to correlate with antisocial and violent behaviour (Davidson, Putnam, & Larson, 2000). In particular, the orbitofrontal cortex, cingulate cortex, amygdala, and interconnected regions have shown both structural and functional abnormalities in antisocial populations. Conversely, neuropsychological functions associated with these brain regions, such as perception of threat and distress as well as modulation of affective response, are compromised in antisocial individuals (Blair et al., 2006). Toxic environments are likely to contribute to these abnormalities in brain function in some, but not necessarily all antisocial individuals. Unfortunately, most of the sparse number of reported brain imaging studies have not subtyped individuals according to their CU profile and as such are sometimes difficult to interpret. Given the proposed contrasting cognitive profile of AB/CU+ vs. AB/CU- it would be informative to study these two subtypes separately.

The IES model proposes that for AB/CU+ individuals, various aspects of amygdala functioning are impaired (e.g. the formation of stimulus-punishment associations). Early amygdala dysfunction may also have a negative impact on the development of empathy (Baron-Cohen, 1995; Baron-Cohen, Leslie & Frith, 1985; Blair, 2006). The IES model postulates that the cognitive and behaviour profile described above for AB/CU+ individuals is a consequence of amygdala hyporeactivity. In other words, the disability of children with AB/CU+ to form associations between moral transgressions and the aversive outcome is a result of amygdala dysfunction. In contrast, AB/CU- individuals are proposed to show amygdala hyperreactivity potentiated by early environmental stressors. Blair et al. (2006) suggest that this amygdala hyper-reactivity leads to the fight response bias and concomitant reactive aggression, but relatively unimpaired social cognition profile observed in AB/CU- individuals.

A handful of functional magnetic resonance imaging (fMRI) studies have studied the brain responsivity to emotional stimuli in callous-unemotional individuals. The most conclusive study to date compared psychopaths (adults with AB/CU+) with other incarcerated individuals and demonstrated that psychopaths show less amygdala activation when performing an emotional memory task (Kiehl et al., 2001). Another study compared psychopaths and controls matched for age and educational level and reported deficient amygdala activation during fear conditioning (Birbaumer et al., 2005). Finally, a recent study used visual cortex activation as a proxy for amygdala, and demonstrated decreased visual cortical response to fearful as compared with neutral faces. However, this study used an unsuitable control group that was not matched on educational level (Deeley et al., 2006).

Only one fMRI study on children with antisocial behaviour has investigated brain reactivity to emotional stimuli. Sterzer, Stadler, Krebs, Kleinschmidt and Poutska (2005) used emotionally significant stimuli and demonstrated amygdala activation in normal children and children with AB. Compared with the control children, children with AB showed less amygdala activation to threat, as long as anxiety/depression was controlled for in the analyses. No fMRI studies looking at AB/CU+ and AB/CU- children separately have been published to date. We are currently conducting a large-scale fMRI study investigating neural response to emotional stimuli in typically developing children, as well as children with AB/CU+ and AB/CU-.

In summary, studies looking at the specific neural profile of AB/CU+ and AB/CU- are scarce, but there is considerable research interest in this area. While adult psychopath neuroimaging studies indicate abnormality in amygdala activity, child neuroimaging studies with AB subjects can only suggest decreased amygdala activation at the present time. In the future, as well as examining brain activity in AB/CU+ and AB/CU- children separately, it will also be particularly important to study how genetic vulnerabilities may manifest at the level of the brain.

In their genes?

The first step in establishing whether genetic influences are important for individual differences in any given behaviour is to consult twin

and adoption studies. As twin studies are the more common of the two, the logic of these studies is discussed briefly here, before reviewing some new data regarding heritability estimates for AB by CU subtype.

The twin method is a natural experiment that relies on the different levels of genetic relatedness between MZ and DZ twin pairs to estimate the contribution of genetic and environmental factors to individual differences, or extreme scores in a phenotype of interest. Phenotypes include any behaviour or characteristic that is measured separately for each twin, such as twins' scores on an antisocial behaviour checklist. Statistical model fitting techniques and regression analyses methods incorporating genetic relatedness parameters are used to investigate the aetiology of the phenotype of choice. These techniques will not be covered in this article and an interested reader should read a textbook on Behavioural Genetics (Plomin, DeFries, McClearn & McGuffin, 2000). The basic premise of the twin method is this: If identical twins, who share 100% of their genetic material, appear more similar on a trait than fraternal twins, who share on average 50% of their genetic material (like any siblings), then we infer that there are genetic influences on a trait. Identical twins' genetic similarity is twice that of fraternal twins'. If nothing apart from genes influences behaviour, then we would expect the identical twins to be twice as similar with respect to the phenotypic measure than fraternal twins. Shared environmental influences – environmental influences that make twins similar to each other – are inferred if fraternal twins appear more similar than is expected from sharing 50% of their genes. Finally, if identical twins are not 100% similar on a trait (as would be expected if only genes influenced a trait), non-shared environmental influences are inferred – in other words environmental influences that make twins different from each other. The non-shared environmental estimate also includes measurement error.

A wealth of twin studies confirms that individual differences in antisocial behaviour and callous-unemotional traits are heritable (Blonigen, Hicks, Krueger, Patrick & Iacono, 2005; Larsson, Viding & Plomin, in press; Rhee & Waldman, 2002; Taylor, Loney, Bobadilla, Iacono & McGue, 2003). Shared environmental influences (environmental influences that make twins similar to each other) play some role for individual differences in AB, but not CU. To our knowledge, only two twin studies to date have investigated whether the aetiology of antisocial behaviour differs as a function of CU traits. Although previous research had strongly suggested that children with AB/CU+ form a distinct subtype (Blair et al., 2006; Frick & Marsee, 2006), possible aetiological differences between these children and others with AB/CU- had not been studied until recently.

To address this question of possible aetiological differences in AB, we first studied teacher ratings of callous-unemotional traits and antisocial behaviour in approximately 7500 7-year-old twins from the Twins Early Development Study (TEDS). We separated children with elevated levels of antisocial behaviour (in the top 10% for the TEDS sample) into AB/CU+ and AB/CU- groups based on their CU score (in the top 10% or not). Antisocial behaviour in children with AB/CU+ was under strong genetic influence (heritability of 0.81) and no influence of shared environment. In contrast, antisocial behaviour in children without elevated levels of callous-unemotional traits showed moderate genetic influence (heritability of 0.30) and substantial environmental influence (shared environmental influence = 0.34, non-shared environmental influence = 0.26). We have recently replicated the finding of different heritability magnitude for the AB/CU+ and AB/CU- groups using the 9-year teacher data (Viding et al., in press). This difference in heritability magnitude holds even after hyperactivity scores of the children are controlled for, suggesting that the result is not driven by any differences in hyperactivity between the two groups. In summary, our research with pre-adolescent twins suggests that while the CU subtype is genetically vulnerable to antisocial behaviour, the non-CU subtype manifests a more strongly environmental aetiology to their antisocial behaviour (Viding et al., 2005; Viding et al., in press).

Common behavioural disorders are currently proposed to be the quantitative extreme of the same genetic effects that operate throughout the distribution (Plomin, 1994; Plomin, Owen & McGuffin, 1994). In this Quantitative Trait Loci (QTL) model many genes are hypothesised to be involved in the development of any behaviour pattern and these genes are thought to act in a probabilistic manner. There has been slow progress in identifying QTLs, as they are neither sufficient, nor necessary to cause extreme behavioural outcome. They can be said to act together with other risk or protective genes to increase or reduce the risk of disorder. Furthermore, risk genes may have to be combined with environmental risk before a clinically significant outcome is produced (Moffitt, Caspi, & Rutter, 2005).

Genes regulating serotonergic neurotransmission, in particular monoamine oxidase A (MAOA), have been highlighted in the search for a genetic predisposition to antisocial behaviour (Lesch, 2003). The MAOA gene is a well-characterised functional polymorphism consisting of a variable number of tandem repeats in the promoter region, with high-activity (MAOA-

H) and low-activity variants (MAOA-L). The MAOA-H variant is associated with lower concentration of intracellular serotonin, whereas the MAOA-L variant is associated with higher concentration of intracellular serotonin. Recent research suggests that genetic vulnerability to antisocial behaviour conferred by the MAOA-L may only become evident in the presence of an environmental trigger, such as maltreatment (Caspi et al., 2002; Kim-Cohen et al., 2006). This research highlights the possibility that increased serotonin availability (often associated with anxiety) in the MAOA-L carriers may serve to increase an individual's vulnerability to environmental risk. The MAOA-L findings appear to be more relevant for the AB/CU- subtype. No molecular genetic studies on CU type of antisocial behaviour exist to date.

Despite the demonstration of genetic influences on individual differences in antisocial behaviour, it is important to note that no genes for antisocial behaviour exist. Instead genes code for neurocognitive vulnerability that may in turn increase risk for antisocial behaviour. Thus, although genetic risk alone may be of little consequence for behaviour in favourable conditions, the genetic vulnerability may still manifest at the level of brain and cognition. Imaging genetics studies attest to genotype differences being evident in the brain structure and function in non-clinical samples (Meyer-Lindenberg & Weinberger, 2006). We can think of this as the neural fingerprint, ready to translate into disordered behaviour in the presence of unfortunate triggers. Meyer-Lindenberg and colleagues recently provided the first demonstration of the MAOA-L genotype being associated with a pattern of neural hypersensitivity to emotional stimuli (Meyer-Lindenberg et al., 2006). Specifically they reported increased amygdala activity coupled with lesser activity in the frontal regulatory regions in MAOA-L as compared with MAOA-H carriers. A recent paper provides further support for the view that a link between the MAOA-L allele and aggression is partly mediated by this pattern of neural hypersensitivity to emotional stimuli (Eisenberger, Way, Taylor, Welch & Lieberman, 2007).

New directions: imaging genetics of AB/CU+ and AB/CU-

Meyer-Lindenberg et al. (2006) speculate that their brain imaging findings of poor emotion regulation in MAOA-L carriers relate to threat reactive and impulsive, rather than CU type antisocial behaviour. This conclusion is based on the observed amygdala hypo- rather than hyper-reactivity in AB/CU+ individuals (Birbaumer et al., 2005; Kiehl et al., 2001). It is thus important to address the potential moderator role of CU on the brain reactivity associated with antisocial behaviour.

A small number of studies have reported increased vulnerability to antisocial behaviour in the presence of the MAOA-H allele (e.g. Manuck, Flory, Ferrell, Dent, Mann & Muldoon, 1999). These may reflect false-positive findings, but it is also possible to speculate that the amygdala hypo- as opposed to hyper-reactivity seen in CU individuals could be influenced by MAOA-H, rather than -L genotype. This suggestion remains highly speculative, and as for any behaviour, the genetic influences will not be limited to a single candidate gene.

As imaging genetic work on antisocial behaviour is currently in its infancy, it is a great opportunity to incorporate lessons learned from the causal modelling tradition (Blair, 2006). We argue that it will be important to employ imaging and cognitive genomics strategies to study how genes to cognition pathways look for different subtypes of antisocial children. Currently such work is being undertaken by our own group (using both twin design to measure heritability and measured genotype to estimate the contribution of individual gene effects) and others.

Practical implications

The research reviewed above suggests that there may be a particularly genetically vulnerable group of youngsters for whom early intervention is likely to be extremely crucial to prevent life course persistent antisocial outcome. We would also like to highlight that prevention and treatment strategies should take into account the different aetiologies of subgroups of antisocial and violent children. Aetiologically heterogeneous samples may explain why intervention programmes can sometimes have mixed results on their success (Frick, 2001; Hawes & Dadds, 2005). Some children seem to respond to well-timed, early prevention and treatment while others do not. We would suggest that the root of this may lie in aetiological differences, particularly differences in cognitive profile of different conduct problem sub-types. The modest to moderate success of intervention programmes may actually reflect a high success rate for affecting the outcome for a particular subtype. Frick (2001) has emphasised that while there are prevention programmes available that address the needs of primarily impulsive antisocial behaviour, less is known about possible prevention and treatment of antisocial behaviour in the callous-unemotional subtype.

Research on environmental risk factors within behavioural genetic designs has highlighted a number of important issues. It is more than likely that for children with vulnerable genotype, this genotype will react with risk environ-

ments. Furthermore, at least one of the parents will share the risk genes for antisocial behaviour and is thus more likely to either directly or indirectly contribute to a less-than optimal rearing environment. As the parent or parents with the antisocial genotype are not often willing or capable to engage in efforts for prevention and treatment, these families present a particular challenge for professionals engaged in preventing a future, on-going cycle of violence. However, recent successes with nurse visit programmes in breaking the association between maltreatment and antisocial behaviour suggest that genetic risk can be effectively moderated by environmental intervention (Eckenrode et al., 2001; Olds et al., 1997). Some children may only require 'milder' environmental risk factors to go down the antisocial path, perhaps due to genetic vulnerability. It is particularly challenging to map out the cognitive profile of these children and make predictions about treatment approaches that capitalise on what is known about cognitive strengths and weaknesses. For example, children with psychopathic tendencies are strong on self-interest and get motivated by rewards, but do not characteristically process others' distress or react to punishment. These are cognitive strengths and limitations that have to be worked with to produce change in behaviour.

As a final note of caution, behavioural genetic research should caution against entertaining ideas of gene therapy for antisocial behaviour. Genes that have variants that are common in the population are more than likely to have multiple functions, some of which are desirable, others not. Hence, a risk gene may have many functions over and above increasing risk for disorder. When this information is combined with the fact that genes interact in complex systems, as well as with environmental risk factors, it seems pertinent to conclude that removing the effects of one gene via gene therapy is unlikely to be effective (Nuffield Council on Bioethics, 2002).

This does not mean that genotype information will be irrelevant for therapeutic intervention. For example, demonstration of genetically (and consequently cognitively) heterogeneous subtypes of early-onset antisocial behaviour suggests the possibility of subtype-specific risk gene variants that index a risk for different cognitive deficits. An early knowledge of such risk genes may come to guide prevention efforts prior to the emergence of clear, overt behavioural markers for the disorder. As cognitive-behavioural approaches are likely to feature strongly in the antisocial behaviour intervention, developing better understanding of the genes-brain-cognition-behaviour pathways for particular subtypes – especially within longitudinal, developmental framework – could provide crucial insights for intervention.

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