Emerging severe personality disorder in childhood

Eileen Vizard

Abstract

Personality disorder does not suddenly emerge at age eighteen years old. There are obvious links between early childhood temperament and the development of personality traits in later childhood. Nevertheless, clinicians do not routinely assess aspects of a child's personality as part of a mental health examination. This may stem from a fear of 'labelling' a child should any problems in personality development be noted, particularly those likely to progress to a personality disorder in adult life. At present, there is little research evidence informing the development of the most commonly presenting adult personality disorders such as borderline and narcissistic personality disorders. There is also a dearth of information about normative personality development in childhood. In contrast, research evidence and clinical experience show that there are behavioural and neuro-cognitive markers for emerging antisocial personality disorder in early childhood. Brain imaging studies have suggested that early childhood trauma may adversely affect the development and functioning of the child's brain. Research has also shown a genetic component in children with psychopathic (callous-unemotional) CU traits. Because adult individuals with psychopathic traits are over-represented in populations of the most dangerous incarcerated offenders, early identification of children with psychopathic traits is clearly important.

Keywords antisocial; callous-unemotional; imaging; neuro-cognitive; psychopathy

Background

Recent government initiatives have invested in secure facilities for adult offenders with Dangerous Severe Personality Disorders (DSPD) whose high levels of psychopathy may make them refractory to treatment.[1] Government has set up the Social Exclusion Task Force to intervene with children and families deemed to be 'high risk/high harm', many of whom also have risk factors for developing antisocial personality disorder.[2,3] The National Academy for Parenting has been set up to provide evidence-based parenting input for high-risk families with young children. For the middle age band of childhood and adolescence (11–18 years old), multi-systemic treatment (MST) sites have been set up to provide treatment for offending children and young people. Research has shown that this type of intervention is more effective in reducing offending behaviour and also cheaper than treatment as usual.[3] Therefore, there is current interest from government, across the life span, in identifying the early childhood indicators of later personality disorder, making effective parenting inputs to high-risk families, providing treatment for adolescent antisocial youth and trying to find some way of containing and treating some of the most dangerous adult offenders in the country. As yet, little research has been done on the most effective way to intervene with the most high-risk children showing psychopathic, callous–unemotional (CU) traits, although they may be unlikely to respond to more traditional parenting approaches.[4–6]

Eileen Vizard ***FRCPsych*** *is Consultant Child and Adolescent Psychiatrist and Honorary Senior Lecturer at University College London. She has been working in the field of child abuse and sexual offending since 1980. She is currently working in an NSPCC funded service, which supports her academic work.*

Epidemiology

Studies of adults with antisocial personality disorder show that they share common childhood risk factors for delinquency.[7–9] Conduct disorder (CD) in childhood has been robustly linked to the later development of antisocial behaviour and psychiatric disorder. Nearly 40% of children with a diagnosis of CD have been noted to develop serious psychosocial disturbance in adult life.[7,10–13] A connection between multiple symptoms of CD and adult antisocial behaviour at age 18 years has been shown to indicate a possible 'dose' relationship.[14]

There is also an association between genotype, childhood maltreatment and the risk of developing antisocial behaviour. New research evidence and a meta-analysis showed that, across studies, the association between maltreatment and mental health problems was significantly stronger in the group of males with the genotype linked to low versus high monoamine oxidase A activity.[15]

There is some evidence that antisocial behaviour arises in individuals with childhood histories of attention deficit hyperactivity disorder (ADHD) rather than CD, but this is controversial. It has been posited that alone ADHD is not a precursor of antisocial personality but, rather, that it increases the risk of antisocial personality when it co-occurs with CD.[16]

A diagnosis of Oppositional Defiant Disorder (ODD) in preschool children might be thought to overlap with CD criteria and identify treatment needs for these children. However, in a review of the criteria for Diagnostic and Statistical Manual of Mental Disorders (DSM)-V CD,[6] it was noted that 'very few studies of pre-schoolers have examined both ODD and CD in the same samples, and no conclusion can yet be reached regarding whether ODD is sufficient to identify treatment at this age or whether a CD diagnosis would improve clinical practice and service delivery'.

The possible disadvantages of preschool CD diagnosis include the fact that aggressive behaviour is common and developmentally normative in pre-schoolers, that conduct problems will decrease across the first 10 years of life and that 'down-aging' diagnostic criteria validated for older adolescents to younger children may promote over-diagnosis.[6] Of the estimated 15% of 5 year olds who display signs of ODD, there are subsequent 'in-flows' and 'out-flows' of individuals from a trajectory towards adolescence. The result is that one-fifth can be expected to escape from this high-risk group by age 8 years, and by age 17 years fewer than half of those in the original ODD group of 5 year olds will fulfil criteria for conduct disorder.[3]

Similarly, desistence from an initial conduct disorder diagnosis is well known. In Robins's 30-year follow-up of a child guidance sample of conduct disordered children, 60–70% did not go on to become adults with antisocial personality disorder.[10] Longitudinal delinquency studies show heterotypic continuity with a range of offence types and early and late onset, desistent and persistent, potential pathways towards adult antisocial behaviour.[6,17,18]

Moffitt's 2008 review also examined the role of subgroups and concluded that the two main age-of-onset subtypes (childhood onset persistent and adolescent onset) conveyed differential information about individuals' characteristic problems, were reliable and had clinical utility. Clear distinctions could be made about the family backgrounds, course and prognosis of the two subtypes of CD. In summary, the childhood onset persistent type showed a wide range of problems, including parental antisocial behaviour, greater genetic liability, neuro-cognitive deficits, low IQ, hyperactivity and peer difficulties, whereas the adolescent group tended to score within normal limits on these measures.[6]

CU traits also distinguish a subgroup of CD children. These CU children show extreme behaviour problems, a stronger genetic risk, more severe and pro-active aggression,[19] more heritability of antisocial traits[20] and at-risk neuro-cognitive profiles.[6] The neuro-cognitive profile of children with CU traits is suggestive of amygdala/orbitofrontal dysfunction, shown by insensitivity to punishment and distress cues[21,22] and is similar to that seen in adult psychopaths.[23] In a large community sample (n = 5770) of young people in the UK, CU traits independently predicted the number and intensity of conduct, emotional and hyperactivity symptoms at follow-up.[24] CD children with CU traits differ from 'CD alone' children in that they show punishment insensitivity and are indifferent to conventional parenting programmes, which tend to use 'time out' interventions.[5]

A very similar profile of children with CU traits was noted in a recent study of conduct disordered children (n = 280), which identified a high-risk subgroup with psychopathic traits. The subgroup, described as having emerging severe personality disorder (ESPD) traits, shared many known risk factors for antisocial behaviour, with multiple CD symptoms, an at-risk neuro-cognitive profile, an early onset of persistent physical aggression towards others and twice as many convictions for violent offences than the non-ESPD group. The study also identified an early onset, delinquent developmental trajectory towards late adolescence and adult life for the ESPD group, in line with Moffitt's 2008 review.[26,6]

Normal personality development and psychopathology

Personality traits have been noted in humans since ancient times and the four body 'humours' (black and yellow bile, blood and phlegm) have been linked to particular personality styles over the centuries. It has long been recognized by researchers (and by parents) that differences in infant temperament can be detected at an early stage in life. Temperament in children was first studied fully in the New York Longitudinal Study (NYLS), which resulted in a nine-trait classification of infant temperament.[25] These traits were clustered into three clinically useful categories of temperament – 'easy', 'difficult' and 'slow to warm up'. Early 'difficult' temperament has subsequently been shown to be a well-known risk factor for a range of adverse outcomes in later childhood.[7,26] Some childhood temperamental traits (e.g. shyness/inhibition) have been linked to biological markers of physiological arousal such as higher salivary cortisol levels and larger eye pupils than non-shy/uninhibited children, with evidence for the stability of the classification inhibition/non-inhibition during a 14 month–7 year follow-up period.[27]

A recent study tested whether temperament and psychophysiology serve as very early childhood indicators (at 3 years) of those with psychopathic personalities in adulthood (at 28 years). Behavioural measures of temperament and skin conductance measures were taken from a sample of 335 children aged 3 years and compared with controls. These individuals were then tested at age 28 years with the Hare Self-Report Psychopathy scale II. Results showed that high scorers on the Hare SRP-II were less fearful and inhibited, more sociable and had longer skin conductance recovery times than controls at 3 years, but they also showed increased autonomic arousal and skin conductance orienting, contrary to expectations.[28] These findings have been interpreted as showing both the stability over time and the capacity for change of the child psychopathy construct. They also appear to suggest a link between temperament, early childhood psychophysiology and later psychopathic personality. The implication is that psychopathic characteristics appear to capture a temperamental pattern which is relatively stable.[29]

An inherited temperament theory of personality disorder was earlier described in which four independent factors of temperament were identified and said to form the foundation of later personality traits.[30] More recently, a unified 'biopsychosocial' theory of personality development has been proposed.[31] However, the question of whether and to what extent there is a continuum between early childhood temperament and the later development of personality has also been raised.[32]

Linked to personality development is the physical development of the child and adolescent brain with particular emphasis on the pre-frontal cortex. Two main changes have been shown in the child's brain before and after puberty.[33] Firstly, increasing myelinization of axons in the frontal cortex continues well into adolescence with a concurrent increase in the speed of neuronal transmission. Secondly, two waves of synaptogenesis and synaptic pruning occur in childhood and after puberty. The second wave of synaptic pruning is thought to be essential for fine tuning the frontal cortex neural networks.[33] MRI studies have now confirmed that there are linear increases in white matter and non-linear decreases in grey matter during adolescence, and recent studies have indicated that the brain may not reach full maturity until well after adolescence.[34] Functional MRI (fMRI) studies allow the adolescent brain to be seen in action undertaking experimental tasks. Risk taking and poor decision-making when risk is involved are common features in adolescents. Two recent fMRI studies showed that adolescents were less efficient than adults in performing the relevant tasks primarily because their brain circuitry was less mature and hence less effective.[35,36] Brain studies of clinical populations differ from these results. Emerging findings from a structural brain imaging study comparing the brains of CD/CU boys with normal controls show increases in grey matter concentration and volume in the brains of the CD/CU boys and not in the controls. The increased grey

