

This section follows on from Chapter D.2, oppositional defiant disorder, by focusing on conduct disorder, which tends to occur in older children and teenagers.

CLASSIFICATION

ICD-10

ICD-10 has a category for conduct disorders, F91. The *clinical descriptions and diagnostic guidelines* state:

“Examples of the behaviours on which the diagnosis is based include the following: excessive levels of fighting or bullying; cruelty to animals or other people; severe destructiveness to property; firesetting; stealing; repeated lying; truancy from school and running away from home; unusually frequent and severe temper tantrums; defiant provocative behaviour; and persistent severe disobedience. Any one of these categories, if marked, is sufficient for the diagnosis, but isolated dissocial acts are not.”
(p267)

An enduring pattern of behaviour should be present, but no time frame is given and there is no impairment or impact criterion stated.

The ICD-10 *diagnostic criteria for research* differ, requiring symptoms to have been present for at least 6 months, and the introductory rubric indicates that impact upon others (in terms of violation of their basic rights), but not impairment of the child, can contribute to the diagnosis. The research criteria take a menu-driven approach whereby a certain number of symptoms have to be present. 15 behaviours are listed to consider for the diagnosis of *conduct disorder*, which usually but not exclusively apply to older children and teenagers. They can be grouped into four classes:

- *Aggression to people and animals*
 - Often lies or breaks promises to obtain goods or favours or to avoid obligations
 - Frequently initiates physical fights (this does not include fights with siblings)
 - Has used a weapon that can cause serious physical harm to others (e.g., bat, brick, broken bottle, knife, gun)
 - Often stays out after dark despite parenting prohibition (beginning before 13 years of age)
 - Exhibits physical cruelty to other people (e.g., ties up, cuts or burns a victim)
 - Exhibits physical cruelty to animals.
- *Destruction of property*
 - Deliberately destroys the property of others (other than by fire-setting)
 - Deliberately sets fires with a risk or intention of causing serious damage).
- *Deceitfulness or theft*
 - Steals objects of non-trivial value without confronting the victim, either within the home or outside (e.g. shoplifting, burglary, forgery).

- *Serious violations of rules*
 - Is frequently truant from school, beginning before 13 years of age
 - Has run away from parental or parental surrogate home at least twice or has run away once for more than a single night (this does not include leaving to avoid physical or sexual abuse)
 - Commits a crime involving confrontation with the victim (including purse-snatching, extortion, mugging)
 - Forces another person into sexual activity
 - Frequently bullies others (e.g., deliberate infliction of pain or hurt, including persistent
 - Intimidation, tormenting, or molestation)
 - Breaks into someone else's house, building or car.

To make a diagnosis, three symptoms from this list have to be present, one for at least six months. There is no impairment criterion. There are three subtypes: *conduct disorder confined to the family context* (F91.0), *unsocialised conduct disorder* (F91.1, where the young person has no friends and is rejected by peers), and *socialised conduct disorder* (F91.2, where peer relationships are normal). It is recommended that age of onset be specified, with *childhood onset type* manifesting before age 10, and *adolescent onset type* after. Severity should be categorised as *mild*, *moderate*, or *severe* according to number of symptoms *or* impact on others, e.g., causing severe physical injury; vandalism; theft.

Where there are sufficient symptoms of a comorbid disorder to meet diagnostic criteria, the ICD-10 system discourages the application of a second diagnosis, and instead offers single, combined categories. There are two major kinds: mixed disorders of conduct and emotions, of which *depressive conduct disorder* (F92.0) is the best researched; and *hyperkinetic conduct disorder* (F90.1). There is modest evidence to suggest these combined conditions may differ somewhat from their constituent elements.

DSM-IV

The DSM IV-R system follows the ICD-10 research criteria very closely and does not have separate clinical guidelines. The same 15 behaviours are given for the diagnosis of conduct disorder (312.8), with almost identical wording. As for ICD-10, 3 symptoms need to be present for diagnosis. Severity, and childhood or adolescent onset are specified in the same way. However, unlike ICD-10, there is no division into socialised/unsocialised, or family context only types, and there *is* a requirement for the behaviour to cause clinically significant impairment in social, academic, or social functioning. Comorbidity in DSM IV-R is handled by giving as many separate diagnoses as necessary, rather than by having single, combined categories.

Differential diagnosis

Making a diagnosis of conduct disorder is usually straightforward but comorbid conditions are often missed. The differential diagnosis may include:

Hyperkinetic syndrome/attention deficit hyperactivity disorder

These are the names given by ICD-10 and DSM IV-R respectively for

similar conditions, except that the former is more severe. For convenience the term *hyperactivity* will be used here. It is characterised by impulsivity, inattention, and motor overactivity. Any of these three sets of symptoms can be misconstrued as antisocial, particularly impulsivity, which can also be present in conduct disorder. However, none of the symptoms of conduct disorder are a part of hyperactivity so, excluding conduct disorder should not be difficult. A frequently made error however, is to miss comorbid hyperactivity when conduct disorder is definitely present. Standardised questionnaires are very helpful here, such as the Strengths and Difficulties Questionnaire, which is brief, and just as effective at detecting hyperactivity as much longer alternatives.

Adjustment reaction to an external stressor

This can be diagnosed when onset occurs soon after exposure to an identifiable psychosocial stressor such as divorce, bereavement, trauma, abuse or adoption. The onset should be within one month for ICD-10, and three months for DSM IV-R, and symptoms should not persist for more than six months after the cessation of the stress or its sequelae.

Mood disorders

Depression can present with irritability and oppositional symptoms but, unlike typical conduct disorder, mood is usually clearly low and there are vegetative features; also more severe conduct problems are absent. Early manic depressive disorder can be harder to distinguish, as there is often considerable defiance and irritability combined with disregard for rules, and behaviour which violates the rights of others. Low self-esteem is the norm in conduct disorder, as is a lack of friends or constructive pastimes. Therefore it is easy to overlook more pronounced depressive symptoms. Systematic surveys reveal that around a third of children with conduct disorder have depressive or other emotional symptoms severe enough to warrant a diagnosis.

Autistic spectrum disorders

These are often accompanied by marked tantrums or destructiveness, which may be the reason for seeking a referral. Enquiring about other symptoms of autistic spectrum disorders should reveal their presence.

Dissocial/antisocial personality disorder

In ICD-10 it is suggested a person should be 17 or older before dissocial personality is considered. Since at age 18 most diagnoses specific to childhood and adolescence no longer apply, in practice there is seldom difficulty. In DSM IV-R conduct disorder can be diagnosed over 18, so there is potential overlap. A difference in emphasis is the severity and pervasiveness of the symptoms of those with personality disorder, whereby all the individual's relationships are affected by the behaviour pattern, and the individual's beliefs about his antisocial behaviour are characterised by callousness and lack of remorse. Coexistent with conduct disorder there may be the personality *trait* of *psychopathy*. The characteristics of the psychopath include grandiosity, callousness, deceitfulness, shallow affect and lack of remorse. These traits, as assessed by the Hare Psychopathy Checklist, have

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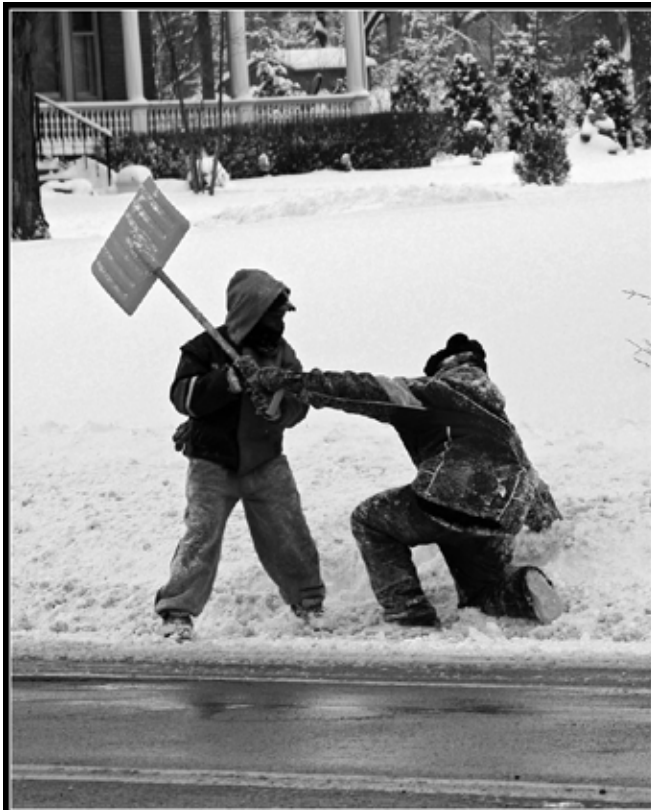
been shown to predict which individuals will engage in the most serious and violent crime careers. Can the “fledgling psychopath” be identified in childhood, as a high priority target for prevention? Callous unemotional traits such as lack of guilt, absence of empathy, and shallow, constricted emotions can be observed in children. A number of reliable instruments are now available for the clinical assessment and diagnosis of psychopathic traits in juvenile patients (Salekin & Lynam, 2010).

Subcultural deviance

Some youths are antisocial and commit crimes but are not particularly aggressive or defiant. They are well adjusted within a deviant peer culture that approves of recreational drug use, shoplifting, etc. In some localities a third or more teenage males fit this description and would meet ICD-10 diagnostic guidelines for socialised conduct disorder. Some clinicians are unhappy to label such a large proportion of the population with a psychiatric disorder. Using DSM IV-R criteria would preclude the diagnosis for most youths like this due to the requirement for significant impairment.

Multiaxial assessment

ICD-10 recommends that multiaxial assessment be carried out for children and adolescents, while DSM IV-R suggests it for all ages. In both systems, axis one is used for psychiatric disorders, which have been discussed above. The last three axes in both systems cover general medical conditions, psychosocial problems, and level of social functioning respectively; these topics will be alluded to below under aetiology. In the middle are two axes in ICD-10, which cover specific (*Axis two*) and general (*Axis three*) learning disabilities respectively; and one in DSM IV-R (*Axis two*) which covers personality disorders *and* general learning disabilities.



Conduct disordered children often become involved in physical fights.

Photo: Tony Fischer

Both *specific and general learning disabilities* are essential to assess in individuals with conduct problems. Fully a third of children with conduct disorder also have specific reading retardation, defined as having a reading level two standard deviations below that predicted by the person's IQ (see also Chapter C.3). While this may in part be due to lack of adequate schooling, there is good evidence that the cognitive deficits often precede the behavioural problems. General learning disability (mental retardation) is often missed in children with conduct disorder unless IQ testing is carried out. The rate of conduct disorder rise several-fold as IQ gets below 70.

EPIDEMIOLOGY

Between 2% and 8% of children and adolescents have conduct disorders. With respect to historical period, A modest rise in diagnosable conduct disorder over the second half of the twentieth century has also been observed comparing assessments of three successive birth cohorts in Britain. There is a marked social class gradient. With respect to ethnicity, youth self-reports of antisocial behaviours and crime victim survey reports of perpetrators' ethnicity show an excess of offenders of black African ancestry. Importantly, Hispanic Americans in the USA and British Asians in the UK do not tend to show an excess of offending compared to their white counterparts, indeed the latter have lower rates than the native white population.

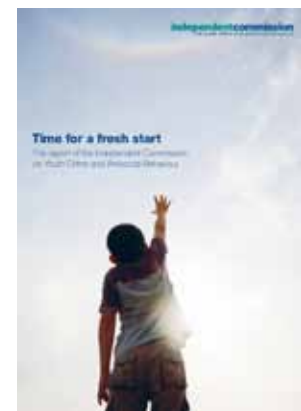
Gender

The sex ratio is approximately 4 to 10 males for each female overall, with males further exceeding females in the frequency and severity of behaviours. On balance, research suggests that the causes of conduct problems are the same for both sexes, but males have more conduct disorder because they experience more of its individual-level risk factors (e.g., hyperactivity, neuro-developmental delays). However, recent years have seen concern among clinicians about increasingly treating antisocial behaviour among girls.

Developmental subtypes

Life-course persistent versus adolescence-limited

There has been considerable attention paid to the distinction between aggressive and disruptive behaviors that are first seen in early childhood versus those that start in adolescence (Moffitt, 1993a; Patterson & Yoerger, 1993), and these two subtypes are encoded in the DSM-IV diagnostic system for conduct disorder. Early onset is a strong predictor of persistence through childhood – and early onset delinquency is more likely to persist into adult life. Findings from the longitudinal Dunedin study, following a 1972-73 birth cohort, have shown that those with early onset differ from those with later onset in that they have lower IQ, more attentional and impulsivity problems, poorer scores on neuropsychological tests, greater peer difficulties and are more likely to come from adverse family circumstances (Moffitt et al, 2001). Those with later onset, by contrast are thought to become delinquent predominantly as a result of social influences such as association with other delinquent youths, or seeking social status through delinquent behaviors. Moffitt (1993a) termed the early-onset group “life



Click on the picture to access the UK Independent Commission on Youth Crime and Antisocial Behaviour' 2010 report *Time for a fresh Start*.

course persistent”, and the later-onset group “adolescence-limited” thus linking developmental course to the differences in underlying deficits. The distinction between the two groups has been broadly supported in longitudinal studies of several cohorts from a dozen countries (Moffitt, 2006). Findings from the follow up of the Dunedin cohort support relatively poorer adult outcomes for the early-onset group in the domains of violence, mental health, substance abuse, work and family life (Moffitt et al, 2002). Follow-up to age 32 revealed that the early-onset life course persistent group had compromised physical health relative to other cohort men, as shown by increased injuries, primary-care physician and hospital visits, and clinical tests of sexually transmitted infections, systemic inflammation, periodontal disease, decayed teeth, and chronic bronchitis.

However the ‘adolescence-limited’ group were not without adult difficulties (Moffitt et al, 2002). As adults they still engaged in self-reported offending, and also had problems with alcohol and drugs. The Cambridge Study in Delinquent Development, a longitudinal study of 411 London males from age 8 to 46, also found that those with antisocial behaviors starting in adolescence were likely to continue to commit undetected crimes in adult life, although their work performance and close relationships were not impaired (McGee & Farrington, 2010). Thus, the age-of-onset subtype distinction has strong predictive validity, but adolescent-onset anti-social behaviors may have more long-lasting consequences than previously supposed and thus both, childhood-onset and adolescent-onset conduct problems, warrant clinical attention.

Childhood-limited conduct problems

Robins (1966) first pointed out that one half of conduct-problem children do not grow up to have antisocial personalities. Longitudinal studies aiming to document the continuity of antisocial behavior from childhood to adolescence to adulthood have repeatedly revealed the existence of an exceptional group of children who lack such continuity. These are often termed “childhood-limited” conduct problems (Moffitt, 2006). Some studies define this childhood-limited group broadly (as a large group of children having any elevated disruptive behavior), and these draw our attention to the ubiquity of temporary conduct problems in the healthy population of children, and show that so long as mild conduct problems do not persist they need not portend poor prognosis (Tremblay, 2003). In contrast, other studies define this childhood-limited group more narrowly (as a small group of children exhibiting extreme, pervasive, and persistent antisocial behavior problems only during childhood). These studies report that such childhood-limited antisocial boys develop into adult men who are depressed, anxious, socially isolated, and have low-paid jobs (Farrington et al, 1988; Moffitt et al, 2002). Thus, boys whose conduct problems are severe and persistent enough to warrant a clinical diagnosis may not later develop antisocial personality, but they will suffer other forms of maladjustment as adults. Thus, all conduct-disorder children warrant clinical attention.

When a young child presents for assessment, the clinician’s task is to make a differential diagnosis between childhood-onset CD that will be only childhood-limited, versus childhood-onset CD that will in future have a life-course persistent course and pathological prognosis. DSM-IV’s age of onset distinction cannot help with this task because all child patients, by definition, have childhood onset.



Lee N Robins (1922-2009) made a significant contribution to the understanding of the natural history of conduct disorder in her seminal book *Deviant Children Grown Up*.

Researchers have tried to distinguish life-course persistent versus childhood-limited trajectory groups by using childhood risk factors, without much success (Moffitt, 2006). However, initial evidence indicates that comorbid ADHD, as well as family psychiatric history, characterize the persistent subtype, but not the childhood-limited subtype.

AETIOLOGY

Individual-level characteristics

Genotypes

The search for specific genetic polymorphisms associated with conduct problems is a very new scientific initiative and little has yet been accomplished. One genome-wide linkage study has identified chromosomal regions that are good bets for harbouring conduct problem-related polymorphisms, but the polymorphisms have not been specified and the regions have not been replicated (Stallings et al, 2005). The most-studied candidate gene in relation to conduct problems is the MAOA promoter polymorphism. The gene encodes the MAOA enzyme, which metabolizes neurotransmitters linked to aggressive behavior by previous research in mice, and among men in a Dutch family pedigree. Thus, MAOA was selected as the candidate gene to test a hypothesis that genetic vulnerability might moderate the effect of child maltreatment on later conduct problems in the cycle of violence (Caspi et al, 2002). Maltreatment history and genotype interacted to predict four different measures of antisocial outcome: diagnosed adolescent conduct disorder, a personality assessment of aggression, symptoms of adult antisocial personality disorder reported by informants who knew the study member well, and adult court conviction for violent crime. Replication of this study was of utmost importance because reports of associations between measured genes and disorders are notorious for their poor replication record. Positive and negative replication studies have appeared and a meta-analysis of these studies showed the association between MAOA genotype and conduct problems is modest but statistically significant (Kim-Cohen et al, 2006). Findings of specific genetic polymorphisms associated with antisocial behavior will probably not be applied for genetic diagnosis purposes because of the inherent complexity of gene-behavior connections. Rather, gene-environment research will benefit efforts to understand how brain mechanisms connect external risk factors and genomic variation to the conduct disorders (Meyer-Lindenberg et al. 2006).

Perinatal complications

Birth complications might be a contributory factor to neuropsychological deficits that are associated with conduct problems (Moffitt, 1993). The evidence regarding this was mixed but recent reports from large-scale general population studies have found associations between life-course persistent type conduct problems and perinatal complications, minor physical anomalies, and low birth weight (Brennan et al, 2003). Most studies support a biosocial model in which obstetric complications might confer vulnerability to other co-occurring risks such as hostile or inconsistent parenting (Arseneault et al, 2002; Kratzer & Hodgins, 1999; Tibbetts & Piquero, 1999; Raine et al, 1997). Studies have further indicated that smoking in pregnancy increases the risk of conduct problems in the offspring (Brennan et al, 2003), but a causal link between smoking and conduct problems has not been established (Fergusson, 1999).

Temperament

Individual differences in infancy that might contribute to subsequent risk of psychopathology were conceptualised by Thomas and Chess in terms of “temperament”, which they viewed as inherited and not significantly influenced by experience (Thomas et al, 1968). Several prospective studies have shown associations between temperament and conduct problems (Keenan & Shaw, 2003), and also predicted antisocial personality disorder and criminal offending into adulthood (Caspi et al, 1996). Temperament as originally conceived, should be strongly heritable and experience-free. However measures of temperament are only moderately heritable and a child’s engagement with the social world from birth means that temperament measures inevitably assess the outcome of social processes. It may be that the contributions of temperament will be seen most consistently in combination with environmental risk factors (Nigg, 2006).

Neurotransmitters

Neurotransmitters have been linked to antisocial behavior in adult samples and in non-human animal models (Nelson, 2006). It would be a major advance if it were possible to link neurotransmitter levels and activity to conduct problems in children. However, in general, the findings with children have not been consistent (Hill, 2002). For example, in the Pittsburgh Youth cohort, boys with longstanding conduct problems showed downward changes in urinary epinephrine level following a stressful challenge task, whereas prosocial boys showed upward epinephrine responses to the challenge (McBurnett et al. 2005). However other studies have failed to find an association between conduct disorder and measures of noradrenaline in children (Hill, 2002). Some limited evidence supports the view that, as in adults, serotonin is linked with aggression in children, but findings for indices of serotonin function in children are also markedly inconsistent (Pine et al, 1997). It should be borne in mind that neurotransmitters in the brain are only indirectly measured – most measures of neurotransmitter levels are crude indicators of activity – and little is known about neurotransmitters in the juvenile brain.

Verbal deficits

Children with conduct problems have been shown consistently to have increased rates of deficits in language-based verbal skills (Lynam & Henry, 2001; Nigg et al, 2003). Conduct disordered children, delinquent adolescents, and adult antisocial individuals show poor performance on standardised tests of verbal ability and in tests of IQ, with poor verbal and performance scores. These associations hold after controlling for potential confounds such as race, socioeconomic status, academic attainment, and test motivation (Lynam et al, 1993). Longitudinal studies show that persistence in antisocial behavior over periods of years is predicted by low verbal IQ in childhood (Farrington & Hawkins, 1991; Lahey et al, 1995; Lynam & Henry, 2001). Deficits in verbal capacities are found also with oppositional defiant disorder among preschool-aged clinic-referred boys (Speltz et al, 1999). Several possible ways in which poor verbal ability might influence behavior can be drawn from Luria’s theory of the role of verbal memory and verbal abstract reasoning in the development of self-control (Luria, 1961). The abilities to recall oral instructions and to use language to think through the consequences of actions contribute to the effective control of actions. Children who cannot reason



To access the US Surgeon Generals’ thorough report on youth violence, click on the picture.

or assert themselves verbally may attempt to gain control of social exchanges using aggression (Dodge, 1993). It is likely that there are also indirect effects in which low verbal IQ contributes to academic difficulties, which in turn mean that the child's experience of school becomes unrewarding rather than a source of self-esteem and support.

Executive dysfunction

Children and adolescents with conduct problems have been shown consistently to have poor tested executive functions (Ishikawa & Raine, 2003; Lynam & Henry, 2001; Moffitt 1993b; Nigg & Huang-Pollock, 2003; Hobson et al, 2011). Executive functions comprise those abilities implicated in successfully achieving goals through appropriate, effective actions. Specific skills include learning and applying contingency rules, abstract reasoning, problem solving, self-monitoring, sustained attention and concentration, relating previous actions to future goals, and inhibiting inappropriate responses. These mental functions are largely, although not exclusively, associated with the frontal lobes (Pennington & Ozonoff, 1996). Important data was generated from a Montreal cohort studied from the age of six years (Séguin et al, 1999). The study used executive function tests, which have been shown to be associated with different anatomical structures in the brain, on the basis of lesion and functional imaging studies. Chronic aggression was associated with lower scores on tests tapping executive functions of the frontal brain region, and the findings held after controlling for general memory, IQ, and ADHD. Although most studies of executive deficits involve adolescents, such deficits have also been linked with disruptive behaviors in preschool children (Hughes et al, 1998; Speltz et al, 1999).

Information processing & social cognition

Dodge (1993) proposed the leading information-processing model for the genesis of aggressive behaviours within social interactions. The model hypothesises that children who are prone to aggression focus on threatening aspects of others' actions, interpret hostile intent in the neutral actions of others, and are more likely to select and to favour aggressive solution to social challenges. Several studies have demonstrated that aggressive children make such errors of social cognition. An extensive review of the many studies of social cognitions among conduct-problem children has been presented elsewhere. Dodge (1993) hypothesised that the tendencies to encode hostile aspects of situation and to attribute hostile intent to ambiguous social cues and to access and favour aggressive responses to social challenges are the result of repeated exposure to physical maltreatment. This prediction was tested prospectively (Dodge et al, 1995). Physical abuse documented in kindergarten was strongly associated with conduct problems in primary school; 28% of the abused group developed conduct problems compared with 6% of the non-abused. Encoding errors, hostile attributions, and biases toward accessing and favouring aggressive responses were each associated with conduct problem outcome and with having experienced physical abuse. Encoding errors and accessing aggressive responses mediated the link between physical abuse and conduct problems, but hostile attributions and positive evaluation of aggressive responses did not. This prospective study thus provided some support for the social cognition model.

Executive functions

The term "executive functions" is used to describe cognitive processes that activate, organize, integrate and manage other functions such as planning, problem solving, verbal reasoning, inhibition, mental flexibility and multi-tasking.



Adolescents with conduct disorder are responsible for a large part of the crime in most communities.

Risks within the family

Genetic liability

There is now solid evidence from twin and adoption studies that conduct problems assessed both dimensionally and categorically are substantially heritable (Moffitt, 2005a; Rhee & Waldman, 2002). However, knowing that conduct problems are under some genetic influence is less useful clinically than knowing that this genetic influence appears to be reduced, or enhanced, depending on interaction with circumstances in the child's environment. Several genetically sensitive studies have allowed interactions between family genetic liability and rearing environment to be examined. Adoption studies have reported an interaction between antisocial behavior in the biological parent and adverse conditions in the adoptive home that predicted the adopted child's antisocial outcome (Bohman 1996; Cadoret et al, 1995). The genetic risk was modified by the rearing environment. A twin study also yielded evidence that family genetic liability and environmental risks interact (Jaffee et al, 2005). In this study, the experience of maltreatment was associated with an increase of 24% in the probability of diagnosable conduct disorder among children at high genetic risk, but an increase of only 2% among children at low genetic risk. Thus, awareness of a familial liability toward psychopathology increases the urgency to intervene to improve a child's social environment.

Low income

There is an association between severe poverty and early-childhood conduct problems (Murray & Farrington, 2010). Early theories proposed direct effects of poverty related to strains arising from the gap between aspirations and realities and from lacking opportunity to acquire social status and prestige. Subsequent research has indicated that the association between low income and childhood conduct problems is indirect, mediated via family processes such as marital discord and parenting deficits (Maughan, 2001). As one example of this research, the Iowa longitudinal study of 378 rural families found that family economic stress was associated with adolescent conduct problems, but this was mediated via parental depression, marital conflict and parental hostility (Conger et al, 1994). Another study took advantage of a naturally occurring experiment (Costello et al, 2003). Native American families in North Carolina, formerly living below the poverty line,

benefited from increased income from newly opened casinos. In many families, the children's behavior problems decreased markedly as a result. However, the effect of increased income was mediated through better parent-child relationships. This mediation is not limited to poverty in recent times. The Glueck's study of delinquency from the historical period of economic depression also found that harsh discipline, low supervision, and weak parent-child attachments accounted for the effects of poverty on children's antisocial behaviors in the 1930's (Sampson & Laub, 1984).

Parent-child attachment

Early studies of low-risk samples, using the secure-insecure attachment classification, failed to find robust associations with externalising problems, but subsequent studies of higher-risk samples using the disorganised classification report that disorganised attachment can predict conduct problems (Van Ijzendoorn et al, 1999). Disorganisation is identified in Ainsworth's Strange Situation Test if the child shows bizarre or contradictory behaviors with the caregiver when reunited after separation (Main & Solomon, 1986). However, low rates of infants with disorganized attachment in study samples mean that findings should be viewed with caution. Although it seems obvious that poor parent-child relations in general predict conduct problems, it has yet to be established whether attachment difficulties, as measured by observational paradigms, have an independent causal role in the development of behavior problems. Attachment classifications could be markers for other relevant family risks. However, Futh et al (2008) used a doll play task with six-year-olds showing an independent association between insecure attachment and conduct problems and Scott et al (2011) found that in adolescents insecure attachment (measured using the Child Attachment Interview) predicted conduct problems even after taking into account current parenting quality, suggesting it may have at least a maintaining role.

Discipline and parenting

Parents of conduct disordered children are more inconsistent in their use of rules, issue more and more unclear commands, are more likely to respond to their children on the basis of their own mood rather than the characteristics of the child's behavior, are less likely to monitor their children's whereabouts, and are less responsive to their children's prosocial behavior. Patterson proposed a specific mechanism for the promotion of oppositional and aggressive behaviors in children. A parent responds to mild oppositional behavior by a child with a prohibition, to which the child responds by escalating his behavior and mutual escalation continues until the parent backs off, thus negatively reinforcing the child's behavior. The parent's inconsistent behavior increases the likelihood of the child showing further oppositional or aggressive behavior. In addition to specific tests of Patterson's reinforcement model (Gardner, 1989; Snyder & Patterson, 1995) there is ample evidence that conduct problems are associated with hostile, critical, punitive and coercive parenting (Rutter et al, 1998); conduct disordered children elicit more negative reactions from all groups of parents than non-conduct disorder children.

The fact that children's behaviors can evoke negative parenting does not mean that negative parenting has no impact on children's behavior. One study reported that negative maternal control at age four was significantly associated

"All in all, the most marked difference between the disciplinary practices of the parents of the delinquents and those of the non-delinquents is found in the considerably greater extent to which the former resorted to physical punishment and the lesser extent to which they reasoned with the boys about their misconduct." (Glueck & Glueck, 1950)

with conduct problems at age nine, after controlling for children's initial behavior problems at age four (Campbell et al, 1996). The E-Risk longitudinal twin study of British families examined the effects of fathers' parenting on young children's aggression (Jaffee et al, 2003). As expected, a prosocial father's *absence* predicted more aggression by his children. But in contrast, an antisocial father's *presence* predicted more aggression by his children, and his harmful effect was exacerbated the more time each week he spent taking care of the children. In another report, the E-risk study evaluated the hypothesis that because depressed mothers provide inept parenting, maternal depression promotes children's aggression (Kim-Cohen et al, 2005). Children of depressed mothers often develop conduct problems, but it has not been clear that this correlation represents environmental transmission. Although the connection between mothers' depression and children's conduct problems decreased somewhat after stringent control for familial liability to psychopathology, it remained statistically significant. Further, depressed women might exaggerate their ratings of their children's problem behaviors, but the pattern of findings remained the same when teachers rated the children's behavior. A temporal analysis showed that if E-risk mothers experienced depression only before their children's birth, the children were not unusually aggressive. In contrast, only if mothers suffered depression while rearing their children were the children likely to develop aggression. Finally, the possibility that the association was spurious because children's aggression provoked their mothers' depression was ruled out by documenting that children exposed to an episode of maternal depression between ages five and seven became even more aggressive by age seven than they had been at age five. Taken together these and other findings provide good evidence for the role of discipline in conduct problems (Moffitt, 2005b).

Exposure to adult marital conflict and domestic violence

It is likely that family processes other than parenting skills and quality of parent-child attachment relationships have a role. Many studies have shown that children exposed to domestic violence between adults are subsequently more likely to themselves become aggressive (Moffitt & Caspi, 1998). Davies and Cummings (1994) proposed that marital conflict influences children's behavior because of its effect on their regulation of emotion. For example, a child may respond to frightening emotions arising from marital conflict by down-regulating his own emotions through denial of the situation. This in turn may lead to inaccurate appraisal of other social situations and ineffective problem solving. Repeated exposure to family conflict is thought to lower children's thresholds for psychological dysregulation, resulting in greater behavioral reactivity to stress (Cummings & Davies, 2002). Children's aggression may also be increased by marital discord because children are likely to imitate aggressive behavior modelled by their parents (Bandura, 1977). Through parental aggression children may learn that aggression is a normative part of family relationships, that it is an effective way of controlling others, and that aggression is sanctioned, not punished (Osofsky, 1995).

Maltreatment

Physical punishment is widely used, and parents of children with conduct problems frequently resort to it out of desperation. Links with conduct problems are not however straightforward. One study found that physical punishment was clearly associated with behavior problems in white American children, but not in African-American ones (Deater-Deckard et al, 1996). Furthermore, the risk for



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Birching (19th century Russia). Kaluga Art Museum.

conduct problems does not apply equally to all forms of physical punishment. The E-risk longitudinal twin study was able to compare the effects of corporal punishment (smacking, spanking) versus injurious physical maltreatment using twin-specific reports of both experiences (Jaffee et al, 2004). Results showed that children's genetic endowment accounted for virtually all of the association between their corporal punishment and their conduct problems. This indicated *a child effect* – in which children's bad conduct provokes their parents to use more corporal punishment, rather than the reverse. Findings about injurious physical maltreatment were the opposite. There was no child effect provoking maltreatment and, moreover, significant effects of maltreatment on child aggression remained after controlling for any genetic transmission of liability to aggression from antisocial parents.

Overall, associations between physical abuse and conduct problems are well established (Hill, 2002). In the Christchurch cohort, child sexual abuse predicted conduct problems after controlling for other childhood adversities (Ferguson et al, 1996). In a large prospective study of court-substantiated cases of abuse and neglect, 26% of abused and neglected adolescents were antisocial, contrasted with 17% in a well-matched comparison group, implying a modest but long-lasting effect of abuse and neglect (Widom, 1997). Investigating the relationship of child maltreatment to psychopathology is particularly difficult for ethical reasons. Little is known about the possible mechanisms linking maltreatment to conduct problems, although threats to security of attachment, difficulties in affect regulation, distortions of information processing and self-concept reviewed elsewhere in this chapter are likely to be relevant.

Risks outside the family

Neighbourhood

It has long been assumed that bad neighbourhoods have the effect of encouraging children to develop conduct problems. Parents strive to secure the best neighbourhood and school for their child that they can afford. Although it is obvious that some local areas have higher crime rates than others, it has been difficult to document any direct link between neighbourhood characteristics and child behavior for a number of reasons. For example, neighbourhood characteristics were conceptualized in overly simple structural-demographic terms, such as percentage of non-white residents or percentage of single-parent households. Moreover, research designs could not rule out the alternative possibility that families whose members are antisocial tend to selectively move into bad neighbourhoods. A new generation of neighbourhood research is addressing these challenges (Beyers et al, 2003; Caspi et al, 2000; Sampson et al, 1997). New research suggests that the neighbourhood factors that are important go beyond structural-demographic characteristics. Neighbourhood-level social processes such as “collective efficacy” and “social control,” do influence young children’s conduct problems, probably by supporting or failing to support parents in their efforts to rear children.

Peers

Children with conduct problems have poorer peer relationships than non-disordered children in that they tend to associate with children with similar antisocial behaviors, they have discordant interactions with other children, and experience rejection by non-deviant peers (Vitaro et al, 2001). Three principal explanations have been tested and evidence found for all three. Either children’s antisocial behaviors lead them to have peer problems or deviant peer relationships lead to antisocial behaviours or some common factor leads to both.

Regarding the possibility that conduct problems lead to peer difficulties, there is ample evidence that children with established conduct problems are more likely to have more conflict with peers and to be rejected by non-deviant peers (Coie, 2004). This peer rejection has been shown to contribute to declines in academic achievement and increases in aggression across the first year of primary schooling (Coie, 2004). One consequence of rejection by healthy peers is that, from as young as five years of age, aggressive-antisocial children are obliged to associate with other deviant children (Farver, 1996; Fergusson et al, 1999).

In light of the limited evidence that peer difficulties prompt the onset of childhood conduct problems and the rather more substantial evidence that children’s peer difficulties are a consequence of their conduct problems, is there any reason to think that peer processes influence the long-term course of conduct problems? Regarding the possibility that peers lead to conduct problems, this has been shown to come about in several ways. Youth who are aggressive are attracted to each other and deviant youth reinforce each others’ antisocial behaviors and attitudes (Boivin & Vitaro, 1995). Evidence that peer influences do increase antisocial behaviors applies primarily to the adolescent developmental stage (Warr, 2002). Strong evidence comes from treatment experiments: in two controlled clinical trials, boys treated in groups did worse than untreated controls; treatment was followed by increased adolescent problem behaviors and poorer outcomes (Dishion et al, 1999). After group-level treatment brought the boys together they

mutually reinforced each other's antisocial activities, a finding which argues for individual treatment approaches. A natural-experiment study tracked change in antisocial behavior among boys who joined a gang to reveal that joining a gang increased each adolescent's individual offending over his pre-gang baseline, whereas leaving the gang decreased each individual's personal offending rate (Thornberry et al, 1993). Overall, we must consider the dynamic and reciprocal manner in which children's conduct problems influence who their friends are and in which those friends later promote the young person's conduct problems (Vitaro et al, 2001).

From risk predictor to evidence for causation

Associations have been documented above between conduct problems and a wide range of risk factors. A variable is called a *risk factor* if it has a documented predictive relation with antisocial outcomes, whether or not the association is causal. The causal status of most of these risk factors is unknown; we know what statistically predicts conduct-problem outcomes but not how or why (Kraemer, 2003). Establishing a causal role for a risk factor is by no means straightforward, particularly as it is unethical to experimentally expose healthy children to risk factors to observe whether those factors can generate new conduct problems. There is no one solution to the problem, although the use of genetically sensitive designs and the study of within-individual change in natural experiments and treatment studies have considerable methodological advantages for suggesting causal influences on conduct problems (Moffitt, 2005b; Rutter, 2000; Rutter et al, 2006). This chapter has emphasized risk factors that have research evidence to support a causal role in conduct problems. For example, above we have cited research that supports causation by depressed mothers' poor discipline (Kim-Cohen et al, 2005), child maltreatment (Dodge et al, 1995; Jaffee et al, 2004), family poverty (Costello et al, 2003), familial genetic liability (Moffitt, 2005a), and affiliating with delinquent peers (Dishion et al, 1999; Thornberry et al, 1993). These studies' designs either took advantage of natural experiments or otherwise were able to rule out alternative explanations to causation (Moffitt, 2005b). Other risk factors described here have not been decisively tested for causation yet but they do have evidence that they are robust predictors of conduct problems across many studies carried out in different contexts (e.g., perinatal complications, temperament, verbal and executive deficits, slow heart rate, social cognitions, exposure to parental conflict). Still other risk factors benefit from strong causal theory, warranting inclusion in this chapter, but the evidence base to show reliable association with conduct problems is not yet

TABLE D.3.1 Factors predicting poor outcome

Onset	Early onset of severe problems, before age 8
Phenomenology	Antisocial acts which are severe, frequent, and varied
Comorbidity	Hyperactivity and attention problems
Intelligence	Lower IQ
Family History	Parental criminality; parental alcoholism
Parenting	Harsh, inconsistent parenting with high criticism, low warmth, low involvement and low supervision.
Wider environment	Low income family in poor neighbourhood with ineffective schools.

strong (e.g., attachment, neurotransmitters, MAOA genotype, smoking during pregnancy, neighbourhood context).

COURSE AND PROGNOSIS

Of those with early onset conduct disorder (before eight years of age) about half persist with serious problems into adulthood. Of those with adolescent onset, the great majority (over 85%) desist in their antisocial behaviour by their early twenties. Factors which predict poor outcome are shown in Table D.3.1.

To detect protective factors, children who do well despite adverse risk factors have been studied. These so-called *resilient* children, however, have been shown to have lower levels of risk factors, for example a boy with antisocial behaviour and low IQ living in a rough neighbourhood but living with supportive, concerned parents. Protective factors are mostly the opposite end of the spectrum of the same risk factor, thus good parenting and high IQ are protective. Nonetheless there are factors which are associated with resilience independent of known adverse influences. These include a good relationship with at least one adult, who does not necessarily have to be the parent; a sense of pride and self-esteem; and skills or competencies.

Adult outcome

Studies of groups of children with early onset conduct disorder indicate a wide range of problems not only confined to antisocial acts, as shown in Table D.3.2.

What is clear is that not only are there substantially increased rates of antisocial acts in adulthood but that the general psychosocial functioning of children with conduct disorder grown up is strikingly poor. For most of the characteristics shown in Table D.3.2, the increase compared to controls is at least double for community cases who were never referred, and three to four times for referred children.

Table D.3.2 Adult outcome

Antisocial Behaviour	More violent and non-violent crimes, e.g. mugging, grievous bodily harm; theft, car crimes, fraud.
Psychiatric problems	Increased rates of antisocial personality, alcohol and drug abuse, anxiety, depression and somatic complaints, episodes of deliberate self-harm and completed suicide, time in psychiatric hospitals
Education and Training	Poorer examination results, more truancy and early school leaving, fewer vocational qualifications
Work	More unemployment, jobs held for shorter time, jobs low status and income, increased claiming of benefits and welfare
Social network	Few if any significant friends, low involvement with relatives, neighbours, clubs and organisations
Intimate relationships	Increased rate of short lived, violent cohabiting relationships; partners often also antisocial
Children	Increased rates of child abuse, conduct problems in offspring, children taken into care
Health	More medical problems, earlier death

Pathways

The path from childhood conduct disorder to poor adult outcome is neither inevitable nor linear. Different sets of influences impinge on the individual growing up and shape the life course. Many of these can accentuate problems. Thus a toddler with an irritable temperament and short attention span may not learn good social skills if he is raised in a family lacking them and where he can only get his way by behaving antisocially and grasping for what he needs. At school he may fall in with a deviant crowd of peers, where violence and other antisocial acts are talked up and give him a sense of esteem. His generally poor academic ability and difficult behaviour in class may lead him to truant increasingly, which in turn makes him fall further behind. He may then leave school with no qualifications, fail to find a job and resort to drugs. To fund his drug habit he may turn to crime and, once convicted, find it even harder to get a job. From this example, it can be seen that adverse experiences do not only arise passively and independently of the young person's behaviour; rather, the behaviour predisposes them to end up in increasingly risky and damaging environments. Consequently, the number of adverse life events experienced is greatly increased. The path from early hyperactivity into later conduct disorder is also not inevitable. In the presence of a warm supportive family atmosphere it is far less likely than if the parents are highly critical and hostile.

Other influences can however steer the individual away from an antisocial path. For example, the fascinating follow-up of delinquent boys to age 70 by Laub and Sampson showed that the following led to desistance: being separated from a deviant peer group; marrying to a non-deviant partner; moving away from a poor neighbourhood; military service which imparted skills.

PRINCIPLES OF TREATMENT

1. Engage the family

Any family coming to a mental health service is likely to have some fears about being judged as bad and possibly mad. Families of conduct problem children are more likely to be disadvantaged and disorganised, to have had arguments with official agencies such as schools and welfare officers and to be suspicious of officialdom. Dropout rates in treatment for conduct problem families are high – often up to 60% (Kazdin, 1996). Practical measures, such as assisting with transportation, providing childcare and holding sessions in the evening or at other times to suit the family are all likely to facilitate retention. Forming a good alliance with the family is especially important, and Prinz and Miller (1994) showed that adding engagement strategies during the assessment, such as showing parents that the therapist clearly understood their viewpoint, led to increased attendance at treatment sessions. Once engaged, the quality of the therapist's alliance with the family affects treatment success, accounting for 15% of the variance in outcome in the meta-analysis by Shirk and Carver (2003).

2. Select which treatment type to use and who should deliver it

If possible, interventions need to address each context specifically, rather than assuming that successful treatment in one area will generalise to another. Thus improvements in the home arising from a successful parent

training programme will not necessarily lead to less antisocial behaviour at school (Scott, 2008). If classroom behaviour is a problem and a school visit shows that the teacher is not using effective methods, then advice to the teacher and other school staff can be very effective. Where there are pervasive problems including fights with peers, then individual work on anger management and social skills should be added. Medication is controversial and generally best avoided; possible indications are discussed below. Generally speaking, due to the strong evidence for its effectiveness, the first line of treatment should be parent training.

Most countries have insufficient resources to treat all antisocial behaviour in childhood, so a decision will need to be made as to whether other agencies can be involved. Thus a number of voluntary sector bodies now provide parent training and schools may also be able to set up suitable behavioural programmes.

3. **Develop strengths**

Identifying the strengths of the young person and the family is crucial. This helps engagement and increases the chances of effective treatment. Encouragement of abilities helps the child spend more time behaving constructively rather than destructively – e.g., more time spent playing football is less time spent hanging round the streets looking for trouble. Encouragement of prosocial activities – for example to complete a good drawing or to play a musical instrument well – also increases achievements and self-esteem and hope for the future.

4. **Treat comorbid conditions**

Child antisocial behaviour often affects others so strongly that comorbid conditions can easily be missed. Yet, comorbidity is the rule rather than the exception in clinical referrals. Common accompaniments are depression and ADHD; a number will have PTSD, for example in the context of violence inflicted on themselves by a father, or witnessing beatings received by their mother from a partner.

5. **Promote social and scholastic learning**

Treatment involves more than the reduction of antisocial behaviour – thus stopping tantrums and aggressive outbursts, while helpful, will not lead to good functioning if the child lacks the skills to make friends or to negotiate: positive behaviours need to be taught too. Specific learning disabilities such as reading retardation, which is particularly common in these children, need treatment, as do more general difficulties such as planning homework.

6. **Use guidelines**

The American Academy of Child and Adolescent Psychiatry has published sensible practice parameters for the assessment and treatment of conduct disorder (AACAP 1997); the UK National Institute for Health and Clinical Excellence (NICE) has published a “technology appraisal” of the clinical and cost effectiveness of parent-training programmes (2006) and is due to publish in 2013 a guideline on assessment and treatment of conduct disorders.



Click on the picture to access the US Federal Government's site National Youth Violence Prevention Resource Center which has recent research findings and provides online training for prevention.

7. Treat the child in their natural environment

Most of the interventions described below are intended for outpatient or community settings. Psychiatric hospitalization is very rarely necessary; there is no evidence that inpatient treatment leads to gains that are maintained after the child is returned to their family.

Specific interventions

Family based

The best known in the context of delinquency is Functional Family Therapy (FFT), brought into being in 1969 by James Alexander and colleagues (Alexander et al, 2000). It is designed to be practicable and relatively inexpensive; 8-12 one hour sessions are given in the family home to overcome attendance problems common in this client group. For more intractable cases, 26-30 hours are offered, usually over three months. The target age range is 11-18. There are four phases to treatment. The first two are the *engagement* and *motivation* phases. Here the therapist works hard to enhance the perception that change is possible and to minimise perceptions that might signify insensitivity or inappropriateness (e.g., poor programme image, difficult to access, insensitive referral). The aim is to keep the family in treatment, and then to move on to find what precisely the family wants. Techniques include *reframing*, whereby positive attributes are enhanced (e.g., a youth who offends a lot but doesn't get caught is labeled as bright) and the emotional motivation is brought out (e.g., a mother who continually nags may be labelled as caring, upset and hurt).

Families are encouraged to see themselves as doing the best they can under the circumstances. *Problem-solving* and *behaviour change* are not commenced until motivation is enhanced, negativity decreased, and a positive alliance established. Explicit attempts are made to reduce negative spirals in family interactions by interrupting and diverting the flow of negative, blaming speeches. Reframes do not belittle the impact of the negative behaviour, but each family member should feel at the end of these two initial stages that:

- They are not inherently bad, it is the way they have done things that hasn't worked
- Even though they have made mistakes, the therapist *sided* with them as much as with everybody else
- Even though they experience the problems differently, each family member must contribute to the solution
- Even though they may have a lot to change, the therapist will work hard to protect them and everyone else in the family
- They want to come back to the next session because it finally seems that things might get better.

The third phase of FFT targets *behaviour change*. There are two main elements to this, communication training and parent training. The success of this stage is dependent on the first two having been achieved and is not commenced unless they have been (this differs from some programmes where a predetermined number of sessions is allocated to each topic irrespective of the rate of family progress). This stage is applied flexibly according to family needs. Thus if there are two parents who continually argue and this is impinging on the adolescent, the



Click on the picture to view *Preventing Violence and Related Health-Risking Behaviors in Adolescents*, a NIH State of the Science Consensus Report.

“marital subsystem” will be addressed, using standard techniques, These include:

1. Using the first person voice rather than the second (instead of “you are a lazy slob”, “I find it upsets me when you leave your socks on the floor”)
2. Being direct (instead of complaining to partner “he never...”, say it directly to the youth)
3. Brevity instead of long speeches
4. Behavioural specificity about what is desired
5. Offering alternatives to the young person, and
6. Active listening.

Parent training techniques are similar to those found in standard approaches and include praise, rewards (called contracting in FFT – e.g., if you come home by 6pm each night, I will take you to the cinema on Saturday), limit setting, consequences and response-cost (e.g., losing TV time for swearing).

The fourth and final phase of FFT is *generalisation*. Here the goal is to get the improvements made in a few specific situations to generalise to other similar family situations, to help the youth and family negotiate positively with community agencies such as school, and help them get the resources they need. Sometimes this latter goal may require the therapist to be a case manager for the family. Therefore, to do this requires that the therapist knows the community agencies and how the system works.

Effectiveness

The effectiveness of FFT is well established, there have been over 10 replication studies (Alexander et al, 2000), of which over half have been independent of the developers, and four are underway in Sweden. The trials published to date all have been positive, with typical recidivism rates being 20% to 30% lower than in controls.

Multiple component interventions

Multisystemic Therapy

The example of Multisystemic Therapy (MST) will be taken as it is one of the best treatments of this kind. MST was developed by Henggeler and colleagues in the US (Huey et al, 2000). There are nine treatment principles:

1. An assessment should be made to determine the fit between the problems and the wider environment: difficulties are understood as a reaction to a specific context, not seen as necessarily intrinsic deficits
2. Therapeutic contacts emphasize the positive and use systemic strengths as levers for change. Already the assessment will have identified strengths (such as being good at sports, getting on well with grandmother, the presence of prosocial peers in grandmother’s neighbourhood). The implementation of this principle means that each contact should acknowledge and work on these.
3. Interventions are designed to promote responsible behaviour and decrease irresponsible behaviour.
4. Interventions are focused in the present, are action oriented, and have specific, well-defined goals. The approach is what can be done in the

here and now, in contrast to some therapies that emphasise the need to understand the family and the youth's past.

5. Interventions target sequences of behaviour in multiple systems that maintain problems.
6. Interventions are developmentally appropriate. They should fit the life stage and personal level of the family members.
7. Interventions require daily or weekly effort by family members. This enables frequent practice of new skills and frequent positive feedback for efforts made. Non-adherence to treatment agreements rapidly becomes apparent.
8. The effectiveness of the intervention is evaluated continuously from multiple perspectives with the intervention team assuming responsibility for overcoming barriers to successful outcomes.
9. Interventions are designed to promote treatment generalization by empowering parents to address the youth's needs across multiple contexts.

The way the therapy is delivered is closely controlled. Due to the weekly monitoring of progress; if there are barriers to improvement, these should be rapidly addressed and the hypotheses of what is going on in the family and systems around the youth should be revised in the light of progress. Clinicians only take on 4-6 cases since the work is intensive. There is close attention to quality control by weekly supervision along prescribed lines and parents and youths themselves fill in weekly questionnaires on whether they have been receiving therapy as planned. Therapy is given for three months and then stopped.

Effectiveness

The first raft of outcome studies by the programme developers was positive. Thus the meta-analysis of papers up to and including 2002 by authors that include one of the programme developers, Charles Borduin, found that in 7 outcome studies comparing MST to treatment as usual or an alternative with 708 youths by 35 therapists, the mean overall effective size across several domains was 0.55 (Curtis et al, 2004). Outcome domains ranged from offending (arrests, days in prison, self-reported criminality, self reported drug-use) where the mean effect size (ES) was 0.50, peer relations (ES: 0.11), family relations (self-reported ES: 0.57, observed ES: 0.76), and individual youth and parent psychopathology symptoms (ES: 0.28). However, the three studies using the developers own graduate students as therapists achieved noticeably larger effect sizes (mean 0.81) than when the developers were supervising local community therapists, where the effect size mean was down to 0.26.

Long-term follow-up 14 years later (when participants' mean age was 29 years) by the developers of one of the first trials (with 176 cases allocated to MST or usual individual therapy), gave recidivism rates of 50% vs 81% respectively. However, in the process of evaluation, the next test of any therapy is its effectiveness when carried out by teams who have no financial or employment ties with the developers (although they may pay the developers for materials and supervision) and with an independent evaluation team (Littell, 2005). The only independent evaluation was also the only one to use proper intention to treat analyses (rather than exclude treatment refusers etc.) in a large sample (n=409) in Ontario, Canada. It found that MST resulted in no improvement compared



with treatment as usual on any outcome, either immediately or by the three year follow up (Lescheid & Cunningham, 2002). A smaller (n=75) independent study in Norway (Ogden & Hagen, 2006) was more positive, founding ESs of 0.26 for self-reported delinquency, 0.50 for parent-rated, and 0.68 for teacher-rated, though here there was 40% missing data.

Interventions that do not work

Harsh, military style shock incarceration, so-called “boot camps”, are still popular for young offenders in the US and were promoted by the Office of Juvenile Justice and Delinquency Prevention in 1992 when 3 pilot programmes were set up. However, several reviews have concluded they are ineffective (Tyler et al, 2001; Stinchcomb, 2005; Benda, 2005; Cullen et al, 2005). A randomized control trial by the California Youth Authority that included long-term arrest data found no difference between boot camp and standard custody and parole (Bottcher & Ezell, 2005). In contrast, a meta-analysis of 28 studies of wilderness programmes found an overall effect size of 0.18, with recidivism rates of 29% vs 37% for controls (Wilson & Lipsey 2000). Programmes with intense physical activity and a distinct therapeutic component were the most effective. Another approach is to seek to frighten young delinquents with visits to prisons in an attempt to deter them, for example in the “Scared Straight” programme. However, a meta-analysis of nine controlled trials found that the intervention is on average more harmful than doing nothing; it led to worse outcomes in the participants (Petrosino et al, 2003).

Medication

At present, there are no pharmacological interventions approved specifically for conduct disorder. Nonetheless, in the US, medications are used relatively frequently and increasingly in this population (Steiner et al, 2003; Turgay, 2004). Primary care physicians are often placed in the position of managing such medications. Concerns have been raised because primary care clinicians often lack training in developmental psychopathology and adequate time for thorough assessment and monitoring (Vitiello, 2001). In the UK medication would not

At present, there are no pharmacological interventions approved specifically for conduct disorder.

generally be supported as good practice because, as discussed below, well-replicated trials of effectiveness are limited, particularly for children without ADHD.

The best-studied pharmacological interventions for youth with conduct problems are psychostimulants (methylphenidate and dexamfetamine), as used with children with co-morbid ADHD and conduct disorder. In these circumstances, there is evidence that reduction in hyperactivity/impulsivity will also result in reduced conduct problems (Connor et al, 2002; Gerardin et al, 2002). There is insufficient reliable evidence to decide whether stimulants reduce aggression in the absence of ADHD; one study by Klein et al (1997) found that improvements in conduct disorder symptoms were independent of ADHD symptom reduction, but this needs replication.

Other pharmacological approaches for antisocial behaviour targeted reactive aggression and over-arousal, primarily in highly aggressive and psychiatrically hospitalised youth. Medications used in these conditions include those purported to target affect dysregulation (e.g., buspirone, clonidine) and mood stabilizers (e.g., lithium, carbamazepine). While Campbell et al found that lithium reduced aggression and hostility in psychiatrically hospitalized youth (Campbell et al, 1995; Malone et al, 2000), others failed to show effectiveness in outpatient samples (e.g., Klein, 1991) and in studies of shorter treatment intervals (i.e., 2-weeks or less) (Rifkin et al, 1997). Carbamazepine failed to outperform placebo in a double-blind placebo controlled study (Cueva et al, 1996). In children with aggression and hyperactivity, Hazell and Stuart (2003) in a placebo controlled, randomized trial of stimulants plus placebo versus stimulants plus clonidine found the latter was more effective. However, it should be noted that polypharmacy carries the risk of increased side effects (Impicciatore et al, 2001).

In the last few years, the use of antipsychotics such as risperidone, and other drugs such as clonidine in outpatient settings has been increasing. However, there is only modest evidence for their effectiveness in conduct disorder in normal IQ children without ADHD. The review by Pappadopoulos et al (2006) found that ESs were larger where ADHD or intellectual disability were present. Findling et al (2000), in a small (n=10 per group), double blind, placebo-controlled study, found significant short-term reduction in aggression. The Risperidone Disruptive Behaviour Study Group used a placebo-controlled, double blind design to examine the effects of risperidone in 110 children with subaverage IQ and conduct problems. Results suggest that risperidone resulted in significant improvements in behaviour versus placebo (Aman et al, 2002; Snyder et al, 2002) but it remains unclear whether the same findings would apply to children with normal IQ. Newer antipsychotics, while not especially sedating, have substantial side-effects, for example risperidone typically leads to considerable weight gain and the prevalence of long-term movement disorders in the long-term is unknown (Reyes et al, 2006). When antipsychotics might be contemplated? Clinical experience suggests they can lead to dramatic reductions in aggression in some cases, especially where there is poor emotional regulation characterized by prolonged rages. Prescribing antipsychotics for relatively short periods (for example, up to 4 months) in low doses (e.g., no more than 1 mg to 1.5 mg of risperidone per day) can help families cope. During this time it is crucial to introduce more effective psychological management. However, antipsychotics are not recommended in anything other than unusual circumstances.

REFERENCES

- Alexander J, Pugh C, Parsons B et al (2000). *Blueprints for Violence Prevention: Functional Family Therapy*. University of Colorado: Institute of Behavioral Science.
- Aman MG, De Smedt G, Derivan A et al (2002). Double-blind, placebo-controlled study of risperidone for the treatment of disruptive behaviors in children with subaverage intelligence. *American Journal of Psychiatry*, 159:1337-1346.
- American Academy of Child and Adolescent Psychiatry (1997). Practice parameters for the assessment and treatment of children and adolescents with conduct disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36:122S-139S.
- Arseneault L, Tremblay RE, Boulerice B et al (2002). Obstetric complications and adolescent violent behaviors: Testing two developmental pathways. *Child Development*, 73:496-508.
- Bandura A (1997). *Social Learning Theory*. Englewood Cliffs NJ: Prentice-Hall.
- Benda BB (2005). Introduction: boot camps revisited: issues, problems, prospects. In BB Benda & NJ Pallone (eds), *Rehabilitation Issues, Problems, and Prospects in Boot Camp*. New York, NY: Haworth Press, pp1-25.
- Beyers JM, Bates JE, Pettit GS et al (2003). Neighborhood structure, parenting processes, and the development of youths' externalizing behaviors: A multi-level analysis. *American Journal of Community Psychology*, 31:35-53.
- Bohman M (1996). Predisposition to criminality: Swedish adoption studies in retrospect. In GR Bock, JA Goode (eds), *Genetics of Criminal and Antisocial Behaviour*. Ciba Foundation Symposium no 194. Chichester: Wiley, pp99-114.
- Boivin N, Vitaro F (1995). The impact of peer relationships on aggression in childhood: inhibition through coercion or promotion through peer support. In J McCord (ed), *Coercion and Punishment in Long-term Perspectives*. Cambridge, UK: Cambridge University Press, pp183-197.
- Bottcher, J. & Ezell, M. E. (2005). Examining the Effectiveness of Boot Camps: A Randomized Experiment with a Long-Term Follow Up. *Journal of Research in Crime and Delinquency*, 42, 309-332.
- Brennan PA, Grekin ER, Mednick SA (2003). Prenatal and perinatal influences on conduct disorder and serious delinquency. In B Lahey, TE Moffitt, A Caspi (eds), *Causes of Conduct Disorder and Delinquency*. New York, NY: Guilford Press, pp319-344.
- Cadoret RJ, Yates WR, Troughton E et al (1995). Genetic-environmental interaction in the genesis of aggressivity and conduct disorders. *Archives of General Psychiatry*, 52:916-924.
- Campbell M, Adams PB, Small AM et al (1995). Lithium in hospitalized aggressive children with conduct disorder: a double-blind and placebo-controlled study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 34:445-453.
- Campbell SB, Pierce EW, Moore G et al (1996) Boys' externalising problems at elementary school age: pathways from early behaviour problems, maternal control, and family stress. *Development and Psychopathology*, 8:701-719.
- Caspi A, McClay J, Moffitt T et al (2002). Evidence that the cycle of violence in maltreated children depends on genotype. *Science*, 297:851-854.
- Caspi A, Moffitt TE, Newman DL et al (1996). Behavioural observations at age 3 predict adult psychiatric disorders: Longitudinal evidence from a birth cohort. *Archives of General Psychiatry*, 53:1033-1039.
- Caspi A, Taylor A, Moffitt T et al (2000). Neighborhood deprivation affects children's mental health: Environmental risks identified using a genetic design. *Psychological Science*, 11:338-342.
- Coie JD (2004). The impact of negative social experiences on the development of antisocial behaviour. In JB Kupersmidt, KA Dodge (eds). *Children's Peer Relations: From Development to Intervention*. Washington, DC: American Psychological Association, pp243-267.
- Conger RD, Ge X, Elder GH et al (1994). Economic stress, coercive family process and developmental problems of adolescents. *Child Development*, 65:541-561.
- Connor DF, Glatt SJ, Lopez ID et al (2002). Psychopharmacology and aggression. I: A meta-analysis of stimulant effects on overt/covert aggression-related behaviors in ADHD. *Journal of the American Academy of Child & Adolescent Psychiatry*, 41:253-261.
- Costello EJ, Compton SN, Keeler G et al (2003). Relationships between poverty and psychopathology: A natural experiment. *Journal of the American Medical Association*, 290:2023-2029.
- Cueva JE, Overall JE, Small AM et al (1996). Carbamazepine in aggressive children with conduct disorder: A double-blind and placebo-controlled study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 35:480-490.
- Cullen FT, Blevins KR, Trager JS et al (2005). The rise and fall of boot camps: a case study in common-sense corrections. *Journal of Offender Rehabilitation*, 40:53-70.
- Cummings EM, Davies P (2002). Effects of marital conflict on children: recent advances and emerging themes in process-oriented research. *Journal of Child Psychology & Psychiatry*, 43:31-64.

- Curtis NM, Ronan KR, Borduin CM (2004). Multisystemic treatment: a meta-analysis of outcome studies. *Journal of Family Psychology*, 18:411-419.
- Davies PT, Cummings EM (1994). Marital conflict and child adjustment: an emotional security hypothesis. *Psychological Bulletin*, 116:387-411.
- Deater-Deckard K, Dodge KA, Bates JE et al (1996). Physical discipline among African American and European American mothers: links to children's externalising behaviours. *Developmental Psychology*, 32:1065-1072.
- Dishion TJ, McCord J, Poulin F (1999). When interventions harm. Peer groups and problem behaviour. *American Psychologist*, 54:755-764.
- Dodge KA (1993). Social-cognitive mechanisms in the development of conduct disorder and depression. In LW Porter, MR Rosenweig (eds), *Annual Review of Psychology*, 44:559-584.
- Dodge KA, Pettit GS, Bates JE et al (1995). Social information – processing patterns partially mediate the effect of early physical abuse on later conduct problems. *Journal of abnormal psychology*, 104:632-643.
- Farrington DP, Gallagher B, Morley L et al (1988). Are there any successful men from criminogenic backgrounds? *Psychiatry*, 51:116-130.
- Farrington DP, Hawkins JD (1991). Predicting participation, early onset, and later persistence and official recorded offending. *Criminal Behaviour and Mental Health*, 1:1-33.
- Farver JAM (1996). Aggressive behaviour in pre-schoolers' social networks: do birds of a feather flock together? *Early Childhood Research Quarterly*, 11:333-350.
- Fergusson DM (1999). Prenatal smoking and antisocial behaviour: commentary. *Archives of General Psychiatry*, 56:223-224.
- Fergusson DM, Horwood LJ, Lynskey MT (1996). Childhood sexual abuse and psychiatric disorder in young adulthood: II psychiatric outcomes of childhood sexual abuse. *Journal of the American Academy of Child and Adolescent Psychiatry*, 35:1365-1374.
- Fergusson DM, Woodward LJ, Horwood LJ (1999). Childhood peer relationship problems and young people's involvement with deviant peers in adolescence. *Journal of Abnormal Child Psychology*, 27:357-370.
- Futh A, O'Connor TG, Matias C et al (2008). Attachment narratives and behavioural and emotional symptoms in an ethnically diverse, at-risk sample. *Journal of the American Academy of Child & Adolescent Psychiatry*, 47:709-718.
- Gardner FEM (1989). Inconsistent parenting: is there evidence for a link with children's conduct problems? *Journal of Abnormal Child Psychology*, 17:223-233.
- Gerardin P, Cohen D, Mazet P et al (2002). Drug treatment of conduct disorder in young people. *European Neuropsychopharmacology*, 12:361-370.
- Glueck S, Glueck E (1950). *Unraveling Juvenile Delinquency*, New York, NY: The Commonwealth Fund, p133.
- Hazell PL, Stuart JE (2003). A randomized controlled trial of clonidine added to psychostimulant medication for hyperactive and aggressive children. *Journal of the American Academy of Child & Adolescent Psychiatry*, 42:886-894.
- Hill J (2002). Biological, psychological and social processes in the conduct disorders. *Journal of Child Psychology and Psychiatry*, 43:133-164.
- Hobson C, Scott S, Rubia K (2011). Cool and hot executive function deficits are associated with ODD/CD symptoms independently of ADHD in adolescents with early onset conduct problems. *Journal of Child Psychology and Psychiatry*, 52:1035-1043.
- Huey SJ, Henggeler SW, Brondino MJ et al (2000). Mechanisms of change in multisystemic therapy: Reducing delinquent behavior through therapist adherence and improved family and peer functioning. *Journal of Consulting and Clinical Psychology*, 68:451-467.
- Hughes C, Dunn J, White A (1998). Trick or treat? Patterns of cognitive performance and executive dysfunction among hard to manage preschoolers. *Journal of Child Psychology and Psychiatry*, 39:981-994.
- Impicciatore P, Choonara I, Clarkson A et al (2001). Incidence of adverse drug reactions in paediatric in/out-patients: a systematic review and meta-analysis of prospective studies. *British Journal of Clinical Pharmacology*, 52:77-83.
- Ishikawa SS, Raine A (2003). Prefrontal deficits and antisocial behaviour: A causal model. In B Lahey, TE Moffitt, A Caspi (eds), *Causes of Conduct Disorder and Delinquency*. New York, NY: Guilford Press, pp277-304.
- Jaffee SR, Caspi A, Moffitt TE et al (2004). The limits of child effects: Evidence for genetically mediated child effects on corporal punishment, but not on physical maltreatment. *Developmental Psychology*, 40:1047-1058.
- Jaffee SR, Caspi A, Moffitt TE et al (2005). Nature x nurture: Genetic vulnerabilities interact with physical maltreatment to promote conduct problems. *Development and Psychopathology*, 17:67-84.
- Jaffee SR, Moffitt TE, Caspi A et al (2003). Life with (or without) father: The benefits of living with two biological parents depend on the father's antisocial behavior. *Child Development*, 74:109-126.
- Kazdin AE (1996). Dropping out of child therapy: Issues for research and implications for practice. *Clinical Child Psychology and Psychiatry*, 1:133-156.
- Keenan K, Shaw DS (2003). Starting at the beginning: Exploring the etiology of antisocial behaviour in the first years of life. In B Lahey, TE Moffitt, A Caspi (eds), *Causes of Conduct Disorder and Delinquency*. New York, NY: Guilford Press, pp153-181.
- Kim-Cohen J, Caspi A, Taylor A et al (2006). MAOA, early adversity, and gene-environment interaction predicting children's mental health: New evidence and a meta-analysis. *Molecular Psychiatry*, 11:903-913.

- Kim-Cohen J, Moffitt TE, Taylor A et al (2005). Maternal depression and child antisocial behavior: Nature and nurture effects. *Archives of General Psychiatry*, 62:173-181.
- Klein R (1991). *Preliminary Results: Lithium Effects in Conduct Disorders*. In CME Syllabus and Proceedings Summary, 144th Annual Meeting of the American Psychiatric Association, New Orleans, 119-120.
- Klein RG, Abikoff H, Klass E et al (1997). Clinical efficacy of methylphenidate in conduct disorder with and without attention deficit hyperactivity disorder. *Archives of General Psychiatry*, 54:1073-1080.
- Kraemer HC (2003). Current concepts of risk in psychiatric disorders. *Current Opinion in Psychiatry*, 16:421-430.
- Lahey BB, Loeber R, Hart EL et al (1995). Four-year longitudinal study of conduct disorder in boys: patterns and predictors of persistence. *Journal of Abnormal Psychology*, 104:83-93.
- Leschied AW, Cunningham A (2002). *Seeking Effective Interventions for Serious Young Offenders: Interim Results of a Four-Year Randomized Study of Multisystemic Therapy in Ontario, Canada*. London, Ontario: Centre for Children and Families in the Justice System.
- Littell JH (2005). Lessons from a systematic review of effects of multisystemic therapy. *Children and Youth Services Review*, 27:445-463.
- Luria AR (1961). *The Role of Speech and the Regulation of Normal and Abnormal Behaviour*. New York: Basic Books.
- Lynam DR, Henry W (2001). The role of neuropsychological deficits in conduct disorders. In J Hill, B Maughan (eds). *Conduct Disorders in Childhood and Adolescence*. Cambridge, UK: Cambridge University Press.
- Lynam DR, Moffitt T, Stouthamer-Loeber M (1993). Explaining the relationship between IQ and delinquency: class, race, test motivation, school failure or self-control? *Journal of Abnormal Psychology*, 102:187-196.
- Main M, Solomon J (1986) Discovery of a new, insecure-disorganised/disoriented attachment pattern. In TB Brazelton, MW Yogman (eds), *Affective Development in Infancy*. Norwood, NJ: Ablex, pp95-124.
- Malone RP, Delaney MA, Luebbert JF et al (2000). A double-blind placebo-controlled study of lithium in hospitalized aggressive children and adolescents with conduct disorder. *Archives of General Psychiatry*, 57:649-54.
- Maughan B (2001). Conduct disorder in context. In J Hill, B Maughan (eds) *Conduct Disorders in Childhood and Adolescence*, Cambridge, UK: Cambridge University Press, pp169-201.
- McGee TR, Farrington DP (2010). Are there any true adult onset offenders? *British Journal of Criminology*, 50:530-549.
- Moffitt TE (1993a). "Life-course-persistent" and "adolescence-limited" antisocial behavior: A developmental taxonomy. *Psychological Review*, 100:674-701.
- Moffitt TE (1993b). The neuropsychology of conduct disorder. *Development and Psychopathology*, 5:135-51.
- Moffitt TE (2005a). Genetic and environmental influences on antisocial behaviors: Evidence from behavioral-genetic research. *Advances in Genetics*, 55:41-104.
- Moffitt TE (2005b). The new look of behavioral genetics in developmental psychopathology: Gene – environment interplay in antisocial behavior. *Psychological Bulletin*, 131:533-554.
- Moffitt TE, Caspi A (1998). Annotation: implications of violence between intimate partners for child psychologists and psychiatrists. *Journal of Child Psychology and Psychiatry*, 39:137-144.
- Moffitt T E, Caspi A, Harrington H et al (2002) Males on the life-course persistent and adolescence-limited antisocial pathways: follow-up at age 26. *Development and Psychopathology*, 14:179-206.
- Moffitt TE, Caspi A, Rutter M et al (2001). *Conduct Disorder, Delinquency and Violence in the Dunedin Longitudinal Study*. Cambridge, UK: Cambridge University Press.
- Moffitt TE, Caspi A, Rutter M et al (2001) *Sex differences in Antisocial Behaviour*. Cambridge, UK: Cambridge University Press.
- Moffitt TE (2006). Life-course-persistent versus adolescence-limited antisocial behaviour. In D Cicchetti, DJ Cohen (eds), *Developmental Psychopathology, Vol 3: Risk, Disorder and Adaptation* (2nd ed), Hoboken, NJ: John Wiley, pp570-598.
- Murray J, Farrington DP (2010). Risk factors for conduct disorder and delinquency: Key findings from longitudinal studies. *Canadian Journal of Psychiatry*, 55:633-642.
- Nelson RJ (2006). *Biology of Aggression*. Oxford, UK: Oxford University Press.
- National Institute for Health and Clinical Excellence (NICE) (2006). *Conduct Disorder in Children - Parent-Training/Education Programmes (TA102)*
- Nigg JT (2006). Temperament and developmental psychopathology. *Journal of Child Psychology and Psychiatry*, 47:395-422.
- Nigg JT, Huang-Pollock C (2003). An earlyonset model of the role of executive functions and intelligence in conduct disorder/delinquency. In B Lahey, TE Moffitt, ACaspi (eds), *Causes of Conduct Disorder and Delinquency*. New York, NY: Guilford Press, pp227-253.
- Ogden T, Hagen KA (2006). Multisystemic treatment of serious behaviour problems in youth: sustainability of effectiveness two years after intake. *Child and Adolescent Mental Health*, 11:142-149
- Osofsky JD (1995). The effects of exposure to violence on young children. *American Psychologist*, 50:782-788.
- Pappadopulos E, Woolston S, Chait A et al (2006). Pharmacotherapy of aggression in children and adolescents: Efficacy and effect size. *Journal of the Canadian Academy of Child and Adolescent Psychiatry*, 15:27-39.
- Patterson GR, Yoerger K (1993). Developmental models for delinquent behaviour. In S Hodgins (ed), *Mental Disorder and Crime*, Newbury Park: Sage, pp140-172.

- Pennington BF, Ozonoff S (1996). Executive functions and developmental psychopathology. *Journal of Child Psychology and Psychiatry*, 37:51-88.
- Petrosino A, Turpin-Petrosino C, Buehler J (2003). Scared Straight and other juvenile awareness programs for preventing juvenile delinquency: A systematic review of the randomized experimental evidence. *Annals of the American Academy of Political and Social Science*, 589:41-62.
- Pine DS, Coplan JD, Wasserman GA et al (1997). Neuroendocrine response to fenfluramine challenge in boys. *Archives of General Psychiatry*, 54:839-846.
- Prinz RJ, Miller GE (1994). Family-Based treatment for childhood antisocial behavior: Experimental influences on dropout and engagement. *Journal of Consulting and Clinical Psychology*, 62:645-650.
- Raine A, Brennan P, Mednick SA (1997). Interaction between birth complications and early maternal rejection in predisposing individuals to adult violence: specificity to serious, early onset violence. *American Journal of Psychiatry*, 154:1265-1271.
- Reyes MB (2006). A randomized, double-blind, placebo-controlled study of risperidone maintenance treatment in children and adolescents with disruptive behavior disorders. *American Journal of Psychiatry*, 163:402-410.
- Rhee SH, Waldman ID (2002). Genetic and environmental influences on antisocial behavior: A meta-analysis of twin and adoption studies. *Psychological Bulletin*, 128:490-529.
- Rifkin A, Karajgi B, Dicker R et al (1997). Lithium treatment of conduct disorders in adolescents. *American Journal of Psychiatry*, 154:554-555.
- Robins LN (1966). *Deviant Children Grown-Up: A Sociological and Psychiatric Study of Sociopathic Personalities*, Baltimore: Williams and Wilkins.
- Rutter M (2000). Psychosocial influences: critiques, findings and research tools. *Development and Psychopathology*, 12:375-406.
- Rutter M, Giller H, Hagell A (1998). *Antisocial Behavior by Young People*. Cambridge University Press, Cambridge.
- Rutter M, Moffitt TE, Caspi A (2006). Gene-environment interplay and psychopathology: Multiple varieties but real effects. *Journal of Child Psychology and Psychiatry*, 47:226-261.
- Salekin RT, Lynam DR (eds) (2010). *Handbook of Child and Adolescent Psychiatry*. Guilford Press, New York.
- Sampson R J, Laub JH (1984). Urban poverty and the family context of delinquency: a new look at structure and process in a classic study. *Child Development*, 65:523-540.
- Sampson RJ, Raudenbush SW, Earls F (1997). Neighbourhoods and violent crime: A multi-level study of collective efficacy. *Science*, 277:918-924.
- Scott S (2008). Parent training programs. In M Rutter, D Bishop, D Pine et al (eds), *Child and Adolescent Psychiatry* (5th edition), Oxford, UK: Blackwell.
- Scott S, Briskman J, Woolgar M et al (2011) Attachment in adolescence: overlap with parenting and unique prediction of behavioural adjustment. *Journal of Child Psychology and Psychiatry* 52:1052-1062.
- Séguin JR, Boulerice B, Harden PW et al (1999). Executive functions and physical aggression after controlling for Attention Deficit Hyperactivity Disorder, general memory, and IQ. *Journal of Child Psychology and Psychiatry*, 40:1197-1208.
- Shirk SR, Karver M (2003). Prediction of treatment outcome from relationship variables in child and adolescent therapy: a meta-analytic review. *Journal of Consulting & Clinical Psychology*, 71:452-464.
- Snyder J, Patterson GR (1995). Individual differences in social aggression: a test of a reinforcement model of socialisation in the natural environment. *Behaviour Therapy*, 26:371-391.
- Snyder R, Turgay A, Aman M et al (2002). Effects of risperidone on conduct and disruptive behavior disorders in children with subaverage IQs. *Journal of the American Academy of Child & Adolescent Psychiatry*, 41:1026-1036.
- Speltz ML, McClellan J, DeKlyen M et al (1999). Pre-school boys with oppositional defiant disorder: Clinical presentation and diagnostic change. *Journal of the American Academy of Child & Adolescent Psychiatry*, 38:838-845.
- Speltz ML, DeKlyen M, Calderon R et al (1999). Neuropsychological characteristics and test behaviours of boys with early onset conduct problems. *Journal of Abnormal Psychology*, 108:315-325.
- Stallings MC, Corley RP, Denney B et al (2005). A genome-wide search for quantitative trait loci that influence antisocial drug dependence in adolescence. *Archives of General Psychiatry*, 62:1042-1051.
- Steiner H, Saxena K, Chang K (2003). Psychopharmacologic strategies for the treatment of aggression in juveniles. *CNS Spectrums*, 8:298-308.
- Stinchcomb JB (2005). From optimistic policies to pessimistic outcomes: why won't boot camps either succeed pragmatically or succumb politically? In BB Benda, N J Pallone (eds), *Rehabilitation Issues, Problems and Prospects in Boot Camp*, New York, NY: Haworth Press, pp27-52.
- Thomas A, Chess S, Birch HG (1968). *Temperament and Behaviour Disorders in Children*. New York: New York University Press.
- Thornberry TP, Krohn MD, Lizotte AJ et al (1993). The role of juvenile gangs in facilitating delinquent behavior. *Journal of Research in Crime and Delinquency*, 30:55-87.
- Tibbetts S, Piquero A (1999). The influence of gender, low birth weight and disadvantaged environment on predicting early onset of offending: A test of Moffitt's interactional hypothesis. *Criminology*, 37:843-878.
- Tremblay RE (2003). Why socialization fails. In B Lahey, TE Moffitt, ACaspi (eds), *Causes of Conduct Disorder and Delinquency*, New York, NY: Guilford Press, pp182-226.

- Turgay A (2004). Aggression and disruptive behavior disorders in children and adolescents. *Expert Review of Neurotherapeutics*, 4:623-632.
- Tyler J, Darville R, Stalnaker K (2001). Juvenile boot camps: A descriptive analysis of program diversity and effectiveness. *Social Science Journal*, 38:445-460.
- Van Ijzendoorn MH, Schuengel C, Bakerman-Kranenburg MJ (1999). Disorganised attachment in early childhood: Meta-analysis of precursors, concomitants, and sequelae. *Development & Psychopathology*, 11:225-249.
- Vitaro F, Tremblay RE, Bukowski WM (2001) Friends, friendships and conduct disorders. In J Hill, B Maughan (eds), *Conduct Disorders in Childhood and Adolescence*, Cambridge. UK: Cambridge University Press, pp346-376.
- Vitiello B (2001). Psychopharmacology for young children: clinical needs and research opportunities. *Pediatrics*, 108:983-9.
- Warr M (2002). *Companions in Crime*. Cambridge, UK: Cambridge University Press.
- Widom CS (1997). Child abuse, neglect and witnessing violence. In D Stoff, J Breiling, JD Maser (eds), *Handbook of Antisocial Behaviour*, New York, NY: John Wiley and Sons, pp159-170.
- Wilson SJ, Lipsey MW (2000). Wilderness challenge programs for delinquent youth: A metaanalysis of outcome evaluations. *Evaluation and Program Planning*, 23:1-12.
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