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Theories of conduct disorder: a causal modelling analysis

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Background: If a clinician has to make decisions on diagnosis and treatment, he or she is confronted with a variety of causal theories. In order to compare these theories a neutral terminology and notational system is needed. The Causal Modelling framework involving three levels of description – biological, cognitive and behavioural – has previously been used to compare causal accounts for dyslexia and autism. **Method:** In this article we present this framework and explore its application to four causal theories of conduct disorder. We discuss the problems we encountered in this application and evaluate both the framework and the theories of conduct disorder. **Conclusions:** It was possible to capture parts of the theories of conduct disorder in the Causal Modelling framework but a multi-model approach may be necessary for the alternative theories of conduct disorder we evaluate. The application of the framework helps to see the relationships among the various theories of aspects of conduct disorder and demonstrates the need for more explicitness in the causal theories. **Keywords:** Causal modelling, causal theories, conduct disorder.

Psychological assessment in the clinical field is not limited to classifying signs into disorders. In order to make an optimal treatment decision a clinician has to take a broad range of possible explanations into account (Frick, 1998; Haynes, 1992; Monsen, Graham, Frederickson, & Cameron, 1998). Research on psychodiagnostic decision-making (for an overview see Garb, 1998; Turk & Salovey, 1988) shows that clinicians tend to generate explanations based on their first impression and hardly consider alternative explanations (anchoring bias). Furthermore, they only look for data that confirms their first impression or initial hypotheses (confirmation bias). In a similar vein, Frederickson, Webster, and Wright (1991) expressed their concern that educational psychologists tend to consider too narrow a range of possible causal hypotheses.

In this article, one of our starting points is the observation that the clinician as a scientist-practitioner has to use theoretical knowledge in order to explain their client's dysfunctional behaviour and consequently to set up treatment. In deciding on diagnosis and thus on treatment, the clinician has, in effect, to choose among a variety of different causal theories both of the same and of different diagnostic categories. In principle, he or she ought to apply to the individual client the full body of scientific knowledge. This is not an easy job in view of the huge amount of literature published every month. Literature reviews on disorders such as conduct disorder (CD) focus on different types of causal factors, for example biological, cognitive or sociological, but rarely integrate them in a single developmental causal framework (AACAP; Dishion, French, & Patterson, 1995; Lahey, Waldman, & McBurnett, 1999; Loeber, Farrington, Stouthamer-Loeber, & Van Kammer, 1998). According to Haynes (1992), this may erroneously lead to the assumption that the causal models should only be at one level, and that causal accounts involving biological and cognitive accounts are incompatible. Other authors have also pointed to a need for multidisciplinary integration in order to understand developmental disorders (Cicchetti & Stroufe, 2000; Kiesler, 1999).

Comparing theories and evaluating them on their causal implications is a complex endeavour and most clinicians would not find it easy to do so as part of their clinical work. Our interest is in finding methods to support the clinician in this difficult task. In order to compare theories on their causal claims one needs a terminology and a related notational system that are neutral, i.e., not tied to any theory, and equally applicable to different causal theories. This theoretical neutrality is characteristic of what we call a *framework*, a term that contrasts with *theory* and *model*. A model is, in this way of thinking, the representation of a theory within a particular framework. However, it is only recently that psychologists have started developing notational frameworks to represent causal theories. Haynes (1998) has developed such a framework in the form of a vector-graphic diagram to represent the information the clinician has gathered from his or her client and reformulate it into a causal model. Such a Functional Analytic Clinical Case Model (FACCM) is specific to the client. In contrast with Haynes, we aim to represent the causal claims of theories which by their nature are generalisable. Moreover, the FACCM notational framework has been developed as a tool for functional analysis, and

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we are looking for a notational framework that is not associated with any particular theoretical approach. As a candidate for this goal, we propose the Causal Modelling framework developed by Morton and Frith (1995). This framework has been applied to a variety of developmental disorders (Blair, 1995; Frith, 1999; Frith, Morton, & Leslie, 1991; Morton & Frith, 1993a, b, 1995, 2002; Sims & Morton, 1998) and has been particularly useful for the comparison of different, often competing, causal theories of dyslexia (BPS, 1999; Frith, 1999). In line with the distinction made earlier, a causal model of a particular theory is a representation of that theory within the Causal Modelling framework. The framework itself is neutral. The framework has also been beneficial in the training of education psychologists (Monsen, Graham, Frederickson, & Cameron, 1998) and Frederickson and Cline (2002) have adapted the framework for understanding pathways of special educational needs.

In this paper we cannot spend too much time defining 'cause', 'causal factors' and 'causal relationship'. For this, we refer to Haynes (1992), Kiesler (1999) and Kraemer et al. (1997). However, following Morton and Frith (1995), we use the phrase 'causal model' partially to describe the *origins* of a disorder. Causal Modelling is concerned with the biological and cognitive origins of developmental disorders, and is a way of keeping track of the various levels of description – biological, cognitive and behavioural – we all use in discussing a disorder. This includes a specification of the cognitive conditions contributing to particular patterns of behaviour. We see this task as different from certain other tasks. In particular, the task of Causal Modelling contrasts with that of specifying the environmental conditions under which a particular behaviour pattern emerges, such as knowing the conditions under which an autistic child might have a violent tantrum. This does not reflect a judgement of relative importance. Knowing the conditions under which an autistic child might have a violent tantrum is vital for the design of treatment and management. We will see that Causal Modelling is partially an analytical tool. But there are limits to its applicability, and that is one of the themes we will be exploring.

The success of Causal Modelling to date has mainly been with disorders with a relatively clear biological origin and well-specified cognitive hypotheses. Our particular interest in this paper is to establish whether the Causal Modelling framework is capable of representing causal explanations for less cognitively defined disorders or whether modifications or additions to the framework are needed to do this. To explore this question, we investigate the application of the Causal Modelling framework for a causal account of CD. We chose CD because it is one of the most frequent diagnoses in outpatient and inpatient mental health facilities for children (APA, 1994) and because environmental factors are supposed to play a causal role. In doing so, the claim of Morton and Frith (1995) that 'any coherent theory about developmental psychopathology – even one considered to be wrong – should be expressible within in the framework' (p. 357) is challenged. In addition, we will use the Causal Modelling framework to critically examine the logical structure of some theories of CD.

In the rest of the paper we will first give a short description of the Causal Modelling framework and its general notation. Next, we explore different causal accounts of CD and its representation in the Causal Modelling framework. Note that we did not attempt to evaluate the empirical status of the different theories of CD, all of which have received a good deal of experimental support. Neither are we exhaustive in describing all the theories that have been postulated for CD, but have selected some that were prominent in textbooks and review articles and were well articulated. We work through these accounts of CD and discuss the problems we encountered. Finally, we evaluate this effort and discuss the implications for research and clinical practice.

Description of the Causal Modelling framework

Morton and Frith (1995) distinguish three levels of description, the biological level, the cognitive level and the behavioural level, and a separate space for environmental influences which can interact at the three levels. The biological level is used to describe genetic factors, brain conditions, and causal links between the two. The influence of environmental factors such as birth complications on brain conditions can be included at this level. At the cognitive level, affective as well as cognitive factors can be included. An affective factor will usually be placed at the intermediate cognitive level, although Morton and Frith acknowledge that affect can also be defined at the biological level as a physiological response or defined at the behavioural level as the manifestation of the affect, e.g., facial expression. However, because the internal process that interprets feelings is considered crucial in explaining how affect can have meaning, affect processing is placed at the cognitive level. Nothing substantive hinges on this decision, however, and if affect were implicated in a causal model in such a way that it was clear that the mechanism was physiological rather than cognitive, then such a factor should be included at the biological level, not the cognitive level.[1]

[1] We should make it clear that we are aware that all cognitive processes have a physiological underpinning. Morton and Frith's position is not a dualist one. An element in a causal model will be placed at the cognitive level if it exerts its influence by virtue of its function rather than by virtue of its biological specification. See Morton and Frith (2002) for a detailed discussion.

The behavioural descriptions of the disorders are placed at the behavioural level. These are the behaviours we can directly observe and where the causal flow of the model finally leads, e.g., poor reading. A description at the behavioural level alone does not explain why a person has a certain problem, e.g., why a child has problems in reading. In a causal model, explanation for a disorder is a function of the interaction of factors at the cognitive and the biological levels and from the environment. Some of these factors will be of developmental significance, others will describe current processing. The cognitive level cannot be observed directly, but is in fact assessed at the behavioural level.

Morton and Frith (1995) created the Causal Modelling framework in an attempt to understand more clearly the differences among various models of developmental disorders. Causal modelling consists of linking elements into the same or at different levels. As such, it can be an efficient tool to represent the multicausality of developmental disorders and to represent biosocial or biopsychosocial theories (Kiesler, 1999). Morton and Frith (2002) assert that different relations can exist between the levels of the framework but claim that the causal relation is a primary concern of causal modelling. They formulated a set of guiding rules for the creation of causal models, of which these are the first five:

1 The causal chain should start with the biological origins, or with a clear statement that there are no such factors.
2 The causal chain should be specified, or at least sketched, from the hypothesised developmental origin to current behaviour. The causal model could thus include factors which were crucial in the course of development (even though, as in the case of delayed development, that factor no longer applied), as well as an account of the current cognitive cause of particular behaviours.
3 All major signs and symptoms of the disorder must be accounted for (or at least mentioned).
4 A distinction between specific and general conditions must be made. Features that can be accounted for as part of a general condition need not be mentioned within the causal theory for the specific condition. For example, although the population of children with autism have impaired intelligence, the consequences of the impaired intelligence should not be part of a causal theory of autism unless they are a consequence of an interaction with an autism-specific deficit.
5 Do not confuse correlation with cause.

For more general information on the framework we refer to Morton and Frith (1995, 2002).

The effects of time

As a child develops, her behavioural repertoire will change. If one is characterising the behavioural phenotype of a developmental disorder, the consequences of cognitive development and of maturation may be that the anticipated behaviour changes dramatically. For example, there may be deviant behaviour at one age which is corrected as the child grows older. The full causal model of the disorder may involve representing the whole time range. To do this we may need to have multiple causal models, as in Figure 1. It will be the specification of the behaviour, $\mathbf{beh_{1-3}}$, which defines the time at which the particular causal model applies. We can also note that the defining biological and cognitive deficits of the particular disorder could remain constant over the three time periods. These are symbolised by the crossed-out **B** and **C** in the figure, which represent disturbed, deficient or missing biological and cognitive structures respectively. These problems remain unchanged over the three time periods.

Why then should the behaviour change? This could be because of the experience the child has had, which has led to changes in knowledge structures or in structures that control the operation of cognitive processes. These changes have enabled the individual to compensate for the abnormality or have enabled the individual to react in a different way to the environment. In the figure, the changing cognitive states are symbolised by $\mathbf{K_{1-3}}$.

Modelling theories of conduct disorder

Conduct disorder (CD) is a term used to describe a group of symptoms or problematic behaviours. The term as used in the DSM-IV (APA, 1994) includes four types of behaviour: (1) aggression to people or animals, (2) destruction of property, (3) deceitfulness or theft and (4) serious violations of rules. It is a very heterogeneous disorder both in its occurrence and in its aetiology. To emphasise this heterogeneity, Frick (1998) started using the plural term 'conduct disorders'. Two different types of CD are nowadays distinguished, i.e., 'the childhood onset type' (DSM-IV) or 'the life-course persistent type' (Caspi & Moffitt,

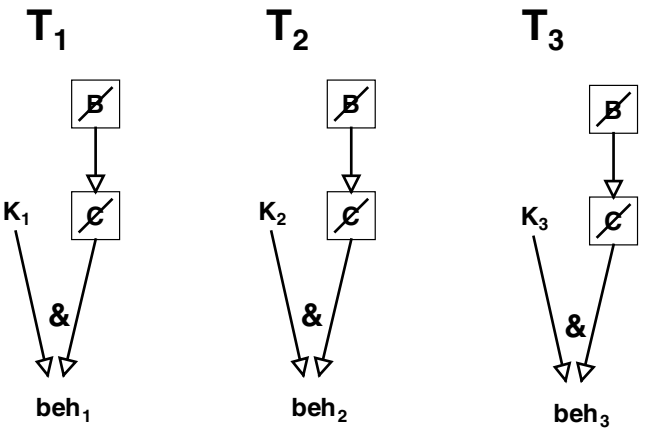

Figure 1 Causal model framework representing time range

1995) and 'the adolescent onset' (DSM-IV) or the 'adolescence limited type' (Caspi & Moffitt, 1995), each with a different aetiology. The spectrum of CD also includes more extreme forms such as psychopathic behaviour. It has been acknowledged that the distinction between these two types of conduct disorders is of importance for its causal accounts and treatment decisions (Frick, 1998; Caspi & Moffitt, 1995; Moffitt, Caspi, Harrington, & Milne, 2002). In this article we focus on the 'childhood onset' or 'life-course persistent type'.

Inspection of several textbooks and review articles (AACAP; Caspi & Moffitt, 1995; Dishion et al., 1995; Frick, 1998; Lahey et al., 1999) led us to select the following theories for modelling: the social information theory of Dodge (1991), the coercive parenting theory of Patterson (1992), the theory of life-course persistent antisocial behaviour by Moffitt (Moffitt, 1993; Caspi & Moffitt, 1995) and the violence inhibition theory of Blair (1995). One can question whether this latter theory can be used as a causal model for CD since the theory is very specific to psychopathy. Classifications of psychopathy are not synonymous with diagnoses of CD but represent an extension (Blair, 2001). An absence of moral emotions is reported in the clinical description of psychopathy. In the DSM-IV manual we find that 'little empathy', 'little concern for feelings, wishes, well-being of others', 'callous and lack of appropriate feelings of guilt or remorse' are described as associated descriptive features of conduct disorder. So this model can be seen as an explanation for a specific subtype of CD, a subtype that may develop to psychopathy. This subtype has as yet not been defined in the DSM manual, but parallels the childhood-onset type with severe symptoms, i.e., symptoms that cause considerable harm to others.

For all four theories, our principle will be to cite the theory precisely from the text and subsequently try to represent it in the Causal Modelling framework. That way, we hope to minimise any misrepresentation of the authors' ideas. We wish to make it clear that it was not the intention of any of the authors whom we discuss, apart from Blair, to produce a causal model for conduct disorder. They are interested in a variety of other aspects of the topic, such as pre-conditions, current circumstances, taxonomy and treatment. In these respects, all these authors have been enormously successful. We take all of these achievements for granted and concentrate on what we can find that is relevant to causal modelling. We start with the violence inhibition theory of Blair because this theory used the causal model framework for its representation.

Violence Inhibition Mechanism (VIM) model

The VIM model is the creation of James Blair. It is a developmental model. Mitchell and Blair (2000) summarise the approach as follows:

> … it is biological make-up that determines whether individuals show emotional difficulties. However, these emotional difficulties are only risk factors for the development of the disorder. It is the individual's adverse social environment that creates the conditions necessary for the development of psychopathy. (Mitchell & Blair, 2000, p. 357)

Blair's model is prompted by ethologists who proposed that most social animals possess mechanisms for control of aggression. These ethologists noted that a conspecific aggressor stops fighting if the opponent displays submission cues. For example, an aggressor dog ceases fighting if its opponent bares its throat. According to Blair, humans might have a functionally similar mechanism which he called a *Violence Inhibition Mechanism* or *VIM*. Blair considers VIM to be:

> a cognitive mechanism which, when activated by non-verbal communications of distress (i.e., sad facial expression, the sight and sound of tears), initiates a withdrawal response: a schema will be activated predisposing the individual to withdraw from the attack'. (Blair, 1995, p. 3)

The activation of VIM is

> considered to result in autonomic arousal and the inhibition of ongoing behaviour… VIM is thought to be activated whenever distress cues are displayed. … The display of distress cues has been found to result in the inhibition of not only aggression … but also non-violent disputes over property ownership … and sexual activity. (Blair et al., 2001, p. 800)

Blair et al. suggest that the 'representations that are current at the time of VIM activation will become triggers for the activation of VIM through a process of classical conditioning' (p. 800). Blair (1995) gives a developmental account for psychopathic behaviour as a causal model where VIM is conceptualised as a basic emotion mechanism that, when impaired, would act as a risk factor for the development of psychopathy. The model is given in Figure 2. Elements that are not affected by an absence of VIM are 'protected' within boxes. Blair (1995) reports that psychopaths are not impoverished on 'theory of mind' tasks, and that they showed arousal to fear stimuli.

In Figure 2 the absence of VIM is conceptualised as either a consequence of a physiological deficit, the absence of early socialisation experiences, or a combination of these factors. Such claims are hypotheses which are to be tested. In effect, this causal model can be seen as Blair's research programme. In the model, the lack of VIM will result in the absence of moral emotions and, as a result, no expressions of moral emotions such as guilt. According to Blair, the child without VIM would not be negatively reinforced by distress cues and would therefore be much more likely to show violent tendencies from an early age.

In Figure 2 we have a 'predisposing social environment' without explicit connections. In his paper

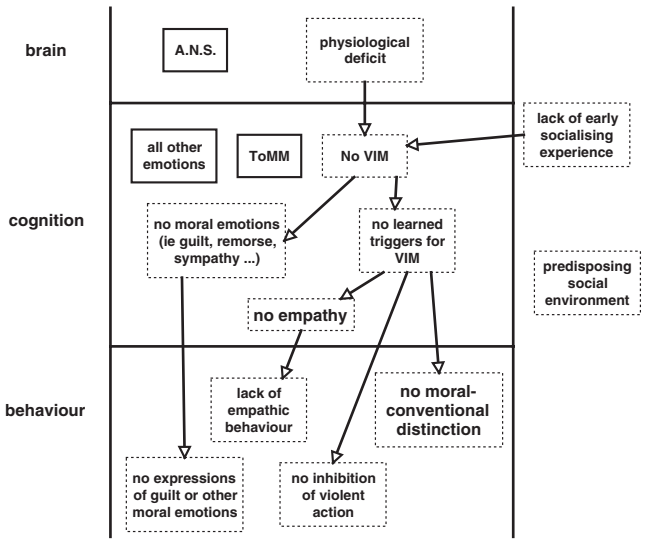

Figure 2 A causal model of the developmental consequences of an absence of VIM (Blair, 1995)

Blair (1995) postulates the idea that the environment fosters motive and motivation is necessary for the development of psychopathy. The environment must, therefore, influence some cognitive factor, although, in the paper, Blair doesn't specify such an interaction. This detail remains for Blair to elaborate.

Blair describes how the lack of VIM makes an individual fail to make the distinction between moral and conventional rules. VIM may be a prerequisite for the internal generation of moral meta-knowledge, i.e., explicit theories held by a person as to why moral transgressions are bad to do. According to Blair, people without VIM will judge an act as bad only because they have been told that it is bad and they will not make a reference to the victim's welfare. Empirical data (Blair, 1995, 1999; Blair et al., 2001) have been produced in support of the existence of the VIM mechanism.

Blair's model describes factors on all three levels, together with environmental factors. In later publications Blair specifies the biological level (Blair, 2001) and its relation to the cognitive level. Although they are not represented in the causal model (see Figure 2), Blair suggests that other cognitive factors, particularly the absence of particular executive skills, may also be associated with psychopathy.

In a later paper Blair (2001) also refers to the different types of behaviours that are distinguished by the Psychopathy Checklist: (1) the emotion dysfunction behaviours defined by emotional shallowness and lack of guilt and (2) the antisocial behaviours such as impulsive aggression and different types of offences. According to Blair, the emotion dysfunctional behaviours are to a certain extent determined by different influences than are the antisocial behaviours. The persistence of emotion dysfunction behaviours may more closely reflect neuro-cognitive impairments that are thought to result in the development of psychopathy. These developments in Blair's thinking would result in a slightly different causal model from that shown in Figure 2, but such additions and extensions are easy to do. Causal modelling readily allows the depiction of such alternative formulations and directly indicates where these formulations make divergent predictions.

The social information processing model for aggressive children

Dodge (1991) makes the distinction between reactive and proactive aggression and hypothesises that these types of aggression have different neural and cognitive mechanisms and different aetiologies and developmental courses. The distinction has received a measure of empirical support (Dodge, Pettit, Bates, & Valente, 1995; Dodge, Lochman, Harnish, Bates, & Pettit, 1997).

> Reactive aggression is displayed as anger or temper tantrums, with an appearance of being out of control. Proactive aggression occurs usually in the form of object acquisition, bullying, or dominance of a peer. (Dodge, 1991, p. 205)

Dodge has associated a range of social information processing biases with aggressive behaviour. In order to understand how these biases can lead to aggression, he gives a description of the steps an individual passes through in order to respond to an environmental stimulus. These include encoding the cues, representing them as threatening or benign, searching for possible responses and then evaluating these before selecting one.

Aggressive children demonstrate biased attention and encoding of hostile stimuli, intention-cue detection errors, hostile-attributional bias, inadequate response search and problem solving, and 'biased response evaluation in the form of expectations of favourable outcomes for aggression' (p. 211). Dodge discusses the mechanisms as follows:

> Problems at early stages of processing, such as hypervigilance to hostile cues, hostile attributions regarding minor provocations, and unwarranted fear responses, are hypothesized to lead to over reactive, defensive aggressive responses. On the other hand, a child who accurately perceives others' intentions but has a limited and biased response repertoire, and who evaluates the outcomes of behaving aggressively in positive ways may be likely to employ aggressive tactics proactively in instrumental ways. (Dodge, 1991, p. 211)

Dodge has found proof for the hypothesis that reactively aggressive boys both demonstrate inaccuracies in the interpretation of peers' cues and also demonstrate strong tendencies to hostile intentions to the peer in ambiguous circumstances. Also, the hypothesis that proactive aggression is associated with favourable evaluations of the outcomes of aggression has been supported.

According to Dodge:

> a history of trauma, abuse, deprivation, and insecure attachment relations will lead to hypervigilance and active aggressive behavior; on the other hand, a history of coercive training (in Patterson's terms) and observation of and experience with successful aggressive tactics will lead a child to access aggressive responses and to evaluate them favorably, resulting in proactive aggression. (pp. 213–214)

When we describe Dodge's theory in the Causal Modelling framework, as in Figure 3, it becomes clear that Dodge focuses on the cognitive level and that no causal factors are specified at the biological level. Dodge does refer to the importance of neural mechanisms but he doesn't explicitly relate them to the social information process or assign them any causal role, either developmentally or currently. He focuses, rather, on how the development of social information processing biases is causally influenced by environmental factors (Dodge, 1991). This was later tested by Dodge et al. (1995), who demonstrated that the influence of physical abuse on later conduct problems is mediated by the child's development of specific 'aggressogenic' styles of processing social cues. Discussing their results, they do refer to other co-occurring mechanisms such as neurochemical pathways or psychophysiological mediators that may play a role and hypothesise that early physical abuse may influence aggressive development by its influence on neurochemical factors. However, no attempt is made to develop this idea.

The factors used at the Cognitive level in Figure 3 are all related to social information processing, but this relationship has to do with (on-line) information processing and not with historical cause. In fact, underlying Dodge's causal claims is a theory of response production. This can be expressed in an information flow model. An information flow model is concerned with the successive steps in information processing independent of the underlying cognitive architecture. In Dodge's system there are three conceptually separable stages identified. In any new situation, cues will be extracted from the environment and interpreted. This is the function of the first stage. On the basis of this interpretation of the environment, the second stage involves possible responses being accessed and evaluated, leading to a response being selected. The third stage is the execution of the response. The cognitive factors that Dodge implicates in reactive aggressive behaviour can all be attributed to the first stage. Hypervigilance and attributions are factors concerned with the perception of the environment. On the other hand, the response repertoire and particular response evaluations belong to the second stage in the model. Effects at this stage lead to proactive aggressive behaviour. Note that there is no necessity in the account we have given that any of this information processing is conscious. When asked why he had acted aggressively, the child might have no idea.

These descriptions have been expanded into a more complex and elegant information processing model by Crick and Dodge (1994). In the reformulated model, more stages are distinguished, and there is virtually no mention whatever of either environmental or biological cause.

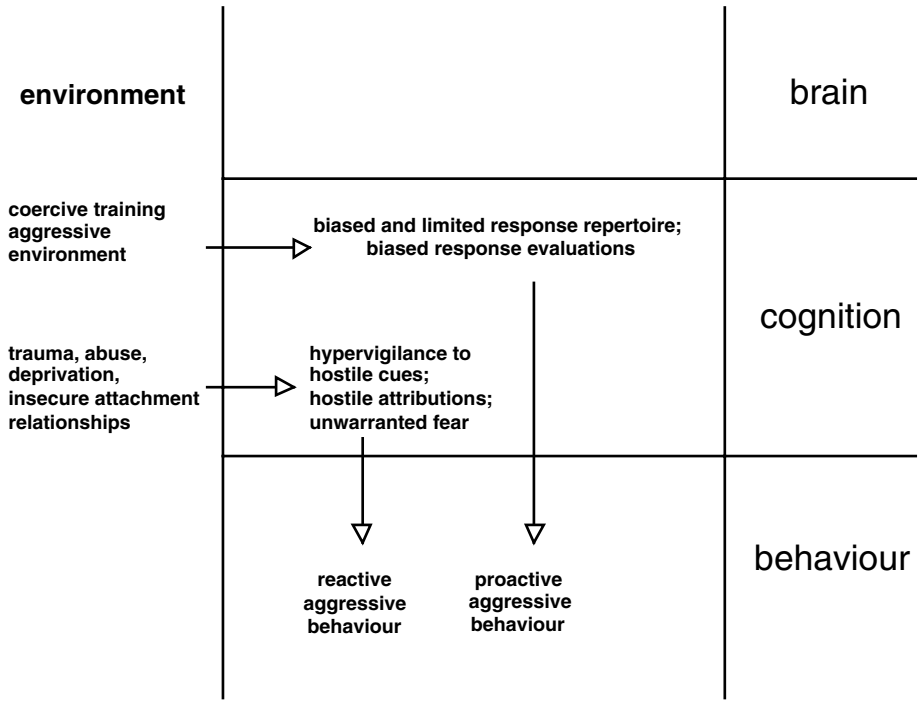

Figure 3 The origins for reactive and proactive aggression according to Dodge (1991)

Coercive parenting model of Patterson

The work of Patterson (e.g., Patterson, Reid, & Dishion, 1992; Dishion et al., 1995) has focused on the contributions of the parent–child interaction to antisocial behaviour. Their social interactional model, referred to as a coercion model, implies 'an emphasis on parent–child exchanges as the proximal cause of antisocial behaviour throughout the life span' (Dishion et al., 1995, p. 438).[2] The model focuses on the process by which the child learns antisocial behaviour within parent–child exchanges and describes how, through coercive exchanges, families train children to be antisocial.

> The child learns to avoid parent demands through a process of negative reinforcement. Repeated over thousands of trials, the child learns to use coercive behaviors to gain control over a disrupted, chaotic, or unpleasant family environment. These patterns become overlearned and automatic, and operate without conscious, cognitive control. In the absence of countervailing forces, the child may progress from displaying these trivial aversive behaviors in the family to exhibiting similar patterns with other people in other settings, to engaging in other social behaviors, including physical aggression, lying, or stealing. (Dishion et al., 1995, p. 439)

These coercion patterns consist of well-rehearsed action–reaction sequences that are performed without conscious awareness of the people involved. Patterson et al. (1992) refer to the coercion model theory as a micro-social reinforcement theory. They refer to the concept of 'overlearned behavior' and note that this concept highlights the difference between the social cognitive perspective (see Dodge) and their social interactional perspective. The events in the coercive patterns are 'performed too quickly to be mediated by cognitive processes' (Patterson et al., 1992, p. 56).

In their book on antisocial boys, Patterson et al. (1992) also propose other factors that relate to the development of antisocial behaviours, e.g., contextual variables such as stressors, socioeconomic status, parental personality traits. Such contextual factors 'might determine early start-up for the coercion process, particularly in conjunction with a temperamentally difficult toddler' (p. 113). Their findings 'suggest that family management variables serve as mediators for the effect of context on child adjustment' (p. 114).

The causal model representation of Patterson's ideas is rather limited. Indeed, the only possible causal claim seems to be that CD is caused by coercive parenting through the mediation of certain mental structures which are created through these parent–child interactions. This claim is shown in Figure 4. There are a number of factors specified which predispose the setting up of coercive parenting, but they do not seem to have the status of causal factors. The growth of the mental structures depends upon the micro-structure of reinforcement and punishment of behaviours under a wide variety of contexts. This would be inappropriate for causal modelling. The way in which the mental structures operate is specified as being 'automatic' and its operation would probably be most easily specified by a listing of the kinds of situation in which particular kinds of behaviour emerge. Again, causal modelling would not be an appropriate tool.

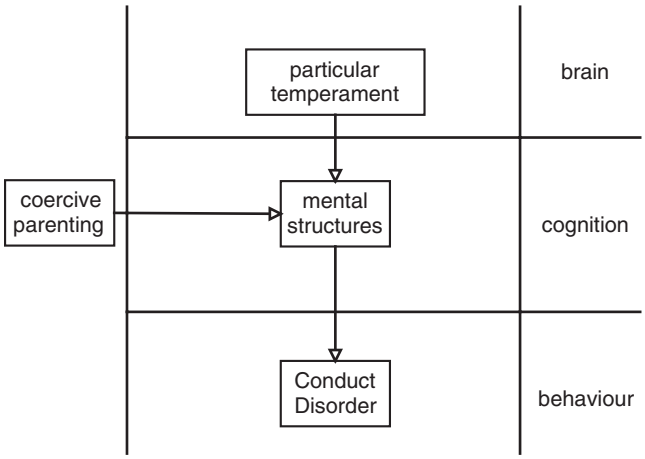

Figure 4 Patterson's causal theory

The theory of life-course persistent antisocial behaviour

Moffitt (1993) and Caspi and Moffitt (1995) develop their account of the roots of lifelong persistent antisocial behaviour along two lines. In the first, they expand on the cognitive consequences of various neurodevelopmental problems and in the second they discuss at length various kinds of maladaptive interaction between the growing child and their parents. This interaction presents some rather acute problems of representation in a causal model, as we found in the discussion of Patterson, and further discussion will be postponed. For the moment, we will focus on the former issue, what Caspi and Moffitt (1995) term 'neuropsychological' problems. We should firstly comment that the theoretical force of the term 'neuropsychological' is that the problems are supposed to arise as a result of long-term biological problems. This would be captured in a causal model by the causal link from the biological level to any specified psychological deficit.

Moffitt (1993) expands along these lines as follows:

> One possible source of neuropsychological variation that is linked with problem behaviour is disruption in the ontogenesis of the fetal brain. Minor physical

[2] In fact, in most of our causal models the proximal cause would be some factor at the cognitive level which led to particular behavioural consequences. Early parent–child interaction seems distal to us, though proximal compared with genetic influences on neurodevelopment.

anomalies … are thought to be observable markers for hidden anomalies in neural development. … Neural development may be disrupted by maternal drug abuse, poor prenatal nutrition, or pre- or postnatal exposure to toxic agents … some individual differences in neuropsychological health are heritable in origin. … After birth, neural development may be disrupted by neonatal deprivation of nutrition, stimulation and even affection. … Some studies have pointed to child abuse and neglect as possible sources of brain injury in the histories of delinquents with neuropsychological impairments. (p. 680)

These quotations make it clear that there is a substantial role for the biological level in the causal model underlying Moffitt's theory, both through the role of genetic factors and through environmental influence on neural development. Caspi and Moffitt (1995) state that the link between neuropsychological deficits and antisocial outcomes has been repeatedly documented in studies of children's aggression, adolescents' delinquency and adults' criminality and is one of the most robust effects in the study of antisocial behaviour. They report that two deficits are empirically associated with antisocial behaviour, verbal deficits and executive function deficits. In attempting to summarise their position in terms of a causal model, there is a problem as to how to express the deficit at the cognitive level. What we have done in Figure 5 is to regard the associations between neuropsychological deficits and antisocial outcomes as being causal, although Caspi and Moffitt do not make that step explicitly. We will point to some limitations of such a step, in the interests of illustrating the requirements of causal modelling.

Caspi and Moffitt spend some time exploring the relationship between the neuropsychological deficits and antisocial behaviour. In respect of the verbal deficit they refer to the fact that many studies found that performance IQ (PIQ) was greater than verbal IQ (VIQ) in delinquents and state that:

> The PIQ > VIQ effect suggests that delinquents may suffer from a specific deficit in language manipulation. This verbal deficit is pervasive, affecting receptive listening and reading, problem solving, expressive speech, writing and memory for verbal material. (Caspi & Moffitt, 1995, p. 478)

They also suggest that this verbal deficit may interact with an adverse home environment. Reference is given to a study of Moffitt (1990) in which she reported an interaction effect between family adversity and verbal ability for self-reported aggressive delinquent acts.

> [The children] characterized by *both* low verbal scores and adverse family environments earned a mean aggression score more than four times greater than that of children with either low verbal ability, or an adverse home environment. (Caspi & Moffitt, 1995, p. 478)

In this paper, we are not concerned to evaluate such empirical claims, only to chart the implications for a causal model for conduct disorder. As it stands, there is no satisfactory way to include such a raw finding in a causal model since we do not know the nature of the crucial verbal deficit, the particular aspect of the adverse home environment, or possible mechanisms of interaction.

Caspi and Moffitt (1995) do, however, refer to several processes by which verbal neurological deficits may contribute to children's antisocial behaviour (p. 478). These are discussed independently of the Moffitt (1990) study and are drawn from discussion from other authors. We will give only a brief

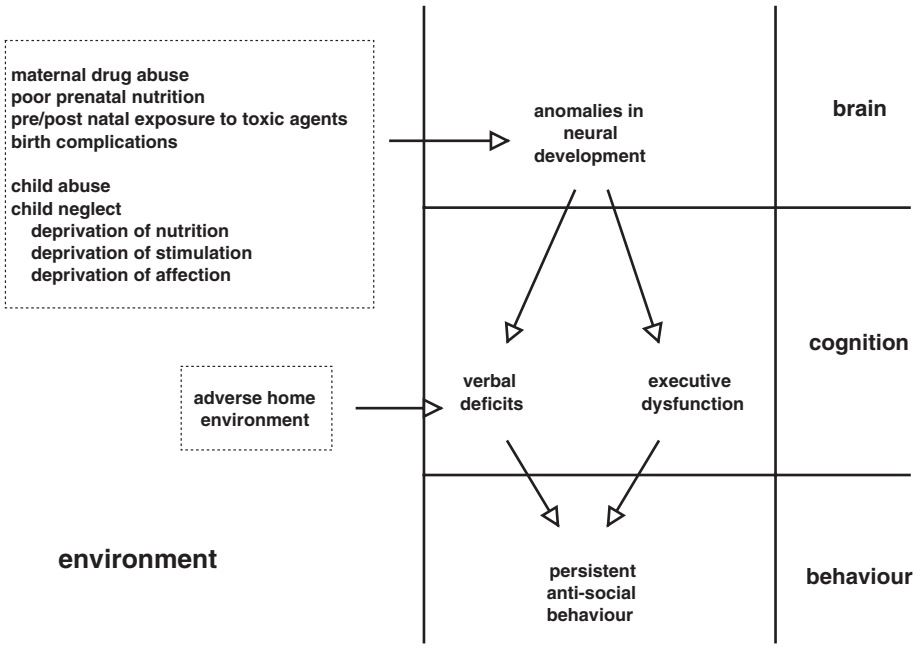

Figure 5 Causal model of Caspi & Moffit's theory of lifelong persistent antisocial behaviour

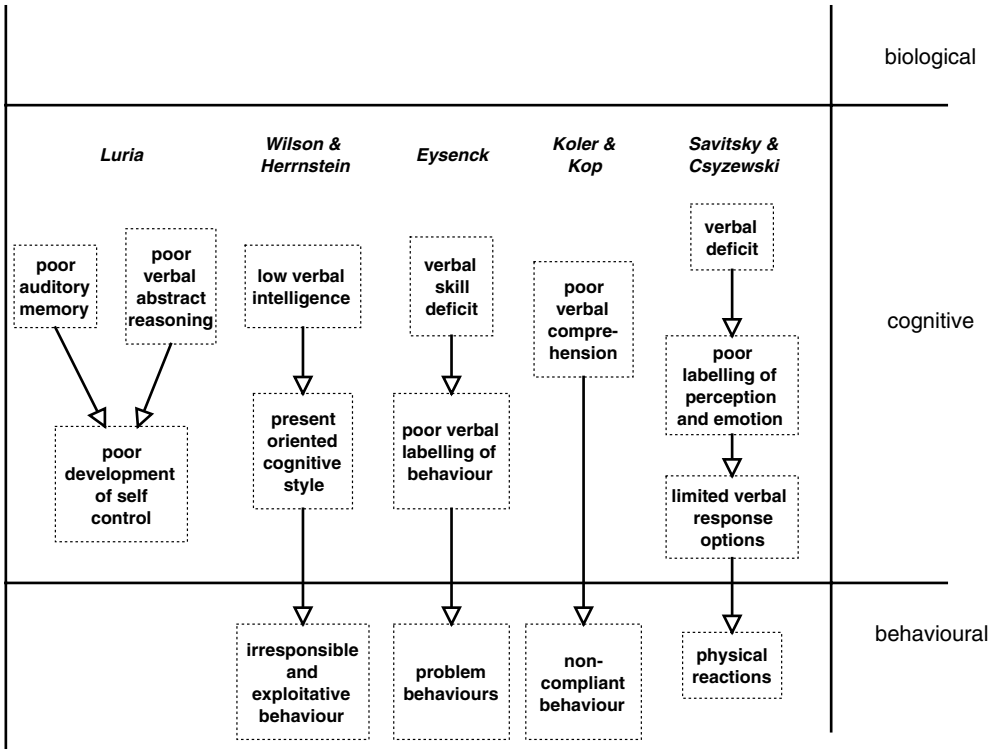

Figure 6 Different processes by which verbal neuropsychological deficits can contribute to children's antisocial behavior

mention of each of these theoretical proposals, and have listed them separately in Figure 6.

To start with, Caspi and Moffitt (1995) state that:

> according to Luria (1961), normal auditory verbal memory and verbal abstract reasoning are essential abilities in the development of self-control, and they influence the socialization process beginning with the earliest parent child–interaction. (p. 478)

They next refer to Wilson and Herrnstein (1985) who:

> suggest that low verbal intelligence contributes to a present-oriented cognitive style, which in turn, fosters irresponsible and exploitative behaviour. Humans use language as the medium for abstract reasoning; we keep things that are 'out of sight' from also becoming 'out of mind' by mentally representing them with words. Language is thus an essential ingredient in prosocial processes such as delaying gratification, anticipating consequences, and linking delayed punishments with earlier transgressions. (Caspi & Moffitt, 1995, p. 478)

An absence of these prosocial processes would define a 'present-oriented cognitive style'.

In accordance with Eysenck's autonomic conditioning theory, Caspi and Moffitt (1995) further suggest that:

> children with verbal-skill deficits might not profit from the labelling of a class of behaviours (e.g., 'naughty' or 'bad') as punishment-attracting; they may have to learn by trial and error … thus experience more frequent punishment events than verbally skilful children, but with proportionally less result in curbing their problem behaviors. (p. 478)

They also refer to a study by Kaler and Kopp (1990) that demonstrated that much of toddler non-compliance can be explained by poor verbal comprehension. They do not demonstrate, however, that the long-term effects of such non-compliance permeated into delinquency. Caspi and Moffitt (1995) also refer to Savitsky and Czyzewski's (1978) speculation that verbal deficits may limit delinquents' ability to label their perceptions of the emotions expressed by others. Such verbal deficits would limit their response options, predisposing them more to physical reactions rather than verbal ones.

This set of speculations, drawn from a number of authors, have in common some chain of events starting with some problem in the verbal domain and ending with unsocial behaviour. Caspi and Moffitt (1995) repeatedly call these verbal deficits 'neuropsychological', implying that they have a biological rather than an environmental origin. They make no attempt to specify what the nature of the verbal deficit might be – apart from noting that it appears to be 'pervasive' – nor is any link made to any of the literature on developmental language disorders. However, a feature of causal modelling is concerned with differential diagnosis. It becomes relevant to ask, then, about other groups of children with pervasive language problems. Let us be clear why this is so. Any element of a causal model stands on its own. If we make a bald claim that **<A causes B>** then A should cause B in all circumstances within the limits of normal variability. It becomes relevant to ask, then, about other groups of children with pervasive

language problems. If, as Wilson and Herrnstein (1985) claim, 'low verbal intelligence contributes to a present-oriented cognitive style, which in turn, fosters irresponsible and exploitative behaviour' then this relation should hold for any group with low verbal intelligence, otherwise the claim has very uncertain explanatory force. It then becomes relevant to ask whether 'irresponsible and exploitative behaviour' is typical of groups with specific language impairment (SLI). The same analysis can be applied to the other fragments described above and diagrammed in Figure 6. We suspect that such behaviour will not be found endemically in such groups, and conclude that the simple claim of a causal relation between verbal deficits and antisocial behaviour is incomplete.

How, then, are we to interpret the correlation between CD and verbal deficits? One possibility is that high verbal skills enable a child to overcome some other disadvantage. Within Moffitt's framework, this might be some to-be-specified executive skill that is missing. In more environmentally based framework (as in the Moffitt, 1990 paper already referred to), one might think of a high verbal child being able to talk their way out of trouble where a low verbal child digs themselves in deeper.

Caspi and Moffitt refer to two kinds of neuropsychological deficit, verbal and executive. The problems we have identified above, concerning the role of verbal deficits in the cause of maladaptive behaviour, do not apply to the same extent to executive deficits. In discussing the latter, Caspi and Moffitt begin by talking about:

> deficiencies in the brain's self-control functions … commonly referred to as 'executive' functions … According to neuropsychological theory, executive dysfunctions should interfere with children's ability to control their own behavior, producing inattentive, impulsive children who are handicapped in considering the future implications of their acts. Such children have difficulty in understanding the negative impact of their behavior on others, fail to hold in mind abstract ideas of ethical values and future rewards, and fail to inhibit inappropriate behaviour or adapt their behaviour to changing social circumstances. (p. 479)

We do not find such general language very helpful. There are two main reasons for this. The first is that many other diagnostic types fail on one or more 'executive' task. Examples include schizophrenia and Parkinson's disease in adults (e.g., Morris et al., 1988; Pantelis & Nelson, 1994), Korsakoff's amnesia, obsessive compulsive disorders, Fragile-X syndrome, ADHD (Chelune, Ferguson, Koon, & Dickey, 1986), autism (Hughes, Russell, & Robbins, 1994; Ozonoff, Pennington, & Rogers, 1991), treated phenylketonuria (Welsh, Pennington, Ozonoff, Rouse, & McCabe, 1990) and Tourette syndrome (Bornstein, 1990). This creates problems for differential diagnosis and for the specification of the causal model. As we have said before, one cannot have pairs of causal elements like:

<executive dysfunction causes conduct disorder>

and

<executive dysfunction causes autism>

because operation of the one should be accompanied by operation of the other. If this is not what is intended – and it surely isn't – then one has to say that 'executive dysfunction' is not the same as 'executive dysfunction', and it is clear that one is not in a theoretical mode of discourse, let alone a causal mode of discourse.

The second reason for dissatisfaction with the use of the term 'executive dysfunction' is the impression it gives of a single, well-defined functional problem. In fact, the term covers a variety of functions ranging from perception to response production, by way of every central cognitive function. Caspi and Moffitt are well aware of this, and give the list:

> sustaining attention and concentration, abstract reasoning and concept formation, goal formulation, anticipation and planning, programming and initiating purposive sequences of behaviour, self-monitoring and self-awareness, inhibition of unsuccessful, inappropriate or impulsive behavior. (Caspi & Moffitt, 1995, p. 479)

Given this diversity of function, it should come as no surprise that 'executive dysfunction' is not a unitary concept and that most patient groups fail only on some executive tests. Any causal model, then, will need to specify the nature of the executive deficit. Caspi and Moffitt do this, drawing attention to the connection between deficits in self-control of attention and long-term antisocial behaviour. They refer to studies by Moffitt of boys in New Zealand (e.g., Moffitt, 1990) where boys who exhibited comorbidity of CD and ADD scored poorly on neuropsychological tests of attentional function as well as having continuity of extreme antisocial behaviour from age 3 to age 15. Boys with CD without ADD had no neuropsychological deficits, nor were their behaviour problems stable. Caspi and Moffitt speculate about the possible genetic origins of the condition as well as pointing to possible 'perinatal sources of cognitive deficit to the development of antisocial behaviour' (p. 480), such as maternal drug abuse. Although these ideas are only briefly alluded to, in terms of work that remains to be done, we can see that Caspi and Moffitt are working towards a causal model such as that in Figure 7.

The question also arises as to whether the effect of the deficits in executive functions is independent of the environment. Do the environmental and neuropsychological factors have separate effects or are both factors necessary to generate the antisocial behaviours? In fact, Caspi and Moffitt (1995) lay great stress on the interaction between the individual and the environment, distinguishing among

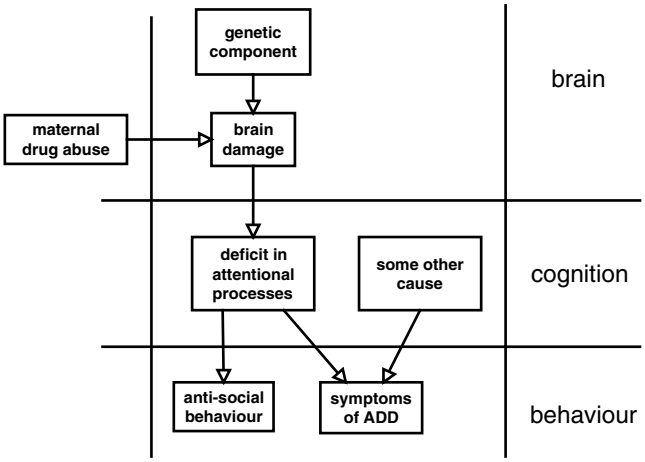

Figure 7 Causal model of relation between an executive dysfunction, i.e., self-control of attention, and antisocial behaviour

three kinds of person–environment interaction: reactive interaction, evocative interaction, and proactive interaction. These interactions result in the build-up of knowledge structures and schemas which will serve to interpret the environment and guide the choice of response. In effect, they are the mechanisms which build up the structures specified in Dodge's social processing model. Their proposals have recently received some empirical support (Moffitt et al., 2002).

Caspi and Moffitt (1995) are implying a causal model which has both biological and environmental input to the cognitive level. The complexity of the three kinds of interaction make it possible that, in the history of the creation of the adult conduct disorder, both verbal and attentional deficit are implicated.

Discussion

In this discussion we will evaluate our exercise to apply causal modelling to theories of conduct disorder, discuss the framework, the theories and the implications for clinical practice.

The applicability of the framework

We have seen that the causal model framework has been able to capture parts of the theories of CD described. It seems that the theories described focus on different descriptive levels of the framework. Only the theory of Blair could be expressed in terms of a full causal model, i.e., the biological, cognitive and behavioural elements as well as environmental elements are described. However, the environmental elements do need further specification. Although it seems almost self-evident that Dodge's social information processing (SIP) theory of CD relates to the cognitive level of the causal model, it seems to us that it is best described in terms of an information flow model of cognitive processing. There is a developmental component, in that particular control parameters have been learned by the child in interaction with the environment, but these are simply described as they exist at the time of the conduct disorder, rather than being derived as the consequence of particular developmental phenomena. In Patterson's work, the focus is on the microstructure of the process whereby environmental influences mould automatic behaviour. Moffitt's views can be clarified to a certain extent through a causal modelling treatment. There are both environmental and biological elements specified. However, the specification at the cognitive level is either too broad – as in the general appeal to verbal deficits – or not demonstrably differentiating, as with the mention of deficits in self-control of attention.

We have not been able to encompass everything in the framework and we are left with the belief that a multi-model approach is necessary. This modelling exercise reveals that at least two types of model may be necessary to really understand a developmental disorder: a causal model to illustrate the developmental prerequisites (e.g., the VIM model of Blair) and a cognitive/computational model to illustrate the dysfunction in the on-line functions that leads to the behavioural disturbance in any given situation (e.g., the SIP model of Dodge). In addition, some state transition models may be necessary to describe the detail of the child–parent interactions in Patterson's theory. We do wish to emphasise that causal modelling is only one way of expressing theories about disorders. Specifically it is about cause, and about historical cause. If specifying cause plays no role in a body of work, then causal modelling will not be able to add very much.

Finally, it seems to us that causal modelling is best suited for the kind of disorder for which it was created and from which it grew – autism and dyslexia. These two disorders have the characteristic that they have a well-established, genetically mediated biological basis, and are seen by most scientists and practitioners as having a well-defined core. We would have no confidence at all in making a similar claim about conduct disorder. CD is a term which refers to a socially defined set of behaviours whose acceptability depends upon the context. The term has then been used as if it had a precise, medical referent. Wakefield, Pottick, and Kirk (2002) also point to the inconsistent use of the term CD by clinicians and researchers, allowing a classification of CD of behaviours not caused by an internal dysfunction. It seems that Wakefield et al. are suggesting that a more cognitive descriptive level should be included in the DSM definition of CD to avoid false positive classifications. We agree that the term CD is diffuse in its application, and it is extremely unlikely that there is any such single thing that can be seen as central to its origin or to its maintenance. This is the case for many diagnostic categories, e.g., anxiety,

depression, and the answer is the same in all cases. Each category must be refined or subdivided until there is a single thing – biological or cognitive – that can be seen as central to its origin or to its maintenance. The contrast, here, is with the phonological deficit central to dyslexia and the mentalising problem central to autism. It does not seem to be established that CD is a proper diagnostic category. It would follow that, if we have two theories of CD which seem to be in conflict, we should not assume that the conflict can be resolved by deciding between them. Instead, they may be theories about different things. Our work in creating Figures 8 and 9 should thus be interpreted with due caution.

What does the application of the framework tells us about the theories?

The application of the framework has helped us see the relationships among the various theories of conduct disorder. To start with, it is clear that the approaches of Blair and Dodge are complementary. Blair is primarily concerned with the possible biological origins of the psychopathic personality and with the mechanisms of inhibition of aggressive responses in normal people. The causal model concerns the detail of the establishment of this inhibition for the normal child over the course of development and the way in which the development of this inhibitory process breaks down with the psychopathic individual. On the other hand, we have not been able to create a satisfactory causal model for Dodge, who is more concerned with what is happening at the time of the aggressive behaviour. In creating his causal model, Blair indicated that the environment had some role but did not specify how it would have an effect. The coercive parenting theory of Patterson does just that, describing the way in which the environment trains the child to become antisocial. In Figure 8, we indicate how Patterson's ideas would interface with Blair's in a causal model. We have also added a frontal/executive dysfunction to show how Caspi and Moffitt's conjectures might be fitted into the same framework. As we have already indicated, these would require some further specification before they could take their place in a developed causal model. The model in Figure 8 is to be interpreted in terms of the development of four kinds of cognitive dysfunction which combine to create the 'particular cognitive processing' of an idealised individual with conduct disorder. The detail of this processing is outside the scope of a causal model, and requires an information processing model such as Dodge's.

Dodge's SIP model requires various deficits in the cognitive parameters controlling information processing. In Figure 9 we have created an information flow model interpretation of this model. This is a slightly simplified version of the model in Crick and Dodge (1994). The model represents the passage of information through the cognitive system at the time of an aggressive act in logical sequence and is not meant to constrain the underlying cognitive architecture, which could be modular or distributed, parallel, cascade or sequential. We have specified a number of cognitive factors on the right-hand side of the figure. The verbal and executive limitations described by Moffitt and Caspi could be seen as examples of the kind of deficit required to make Dodge's model work in both the selection and evaluation of possible responses and in the response execution. In addition, it does not take much imagination to see how VIM-related influences could be seen to affect the perception of the environment as well as the selection of responses. In this way, we can see how the causal/developmental theories of Blair

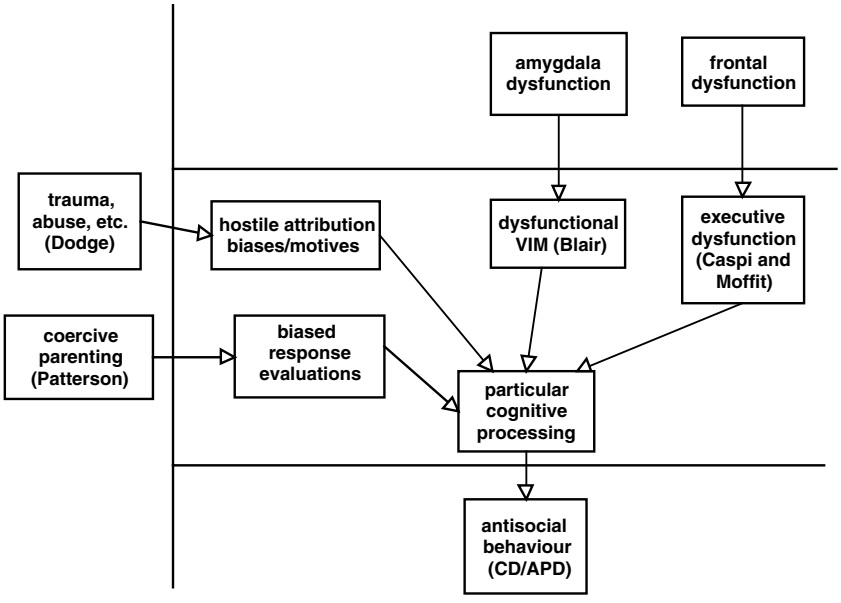

Figure 8 Integrative causal model on conduct disorder

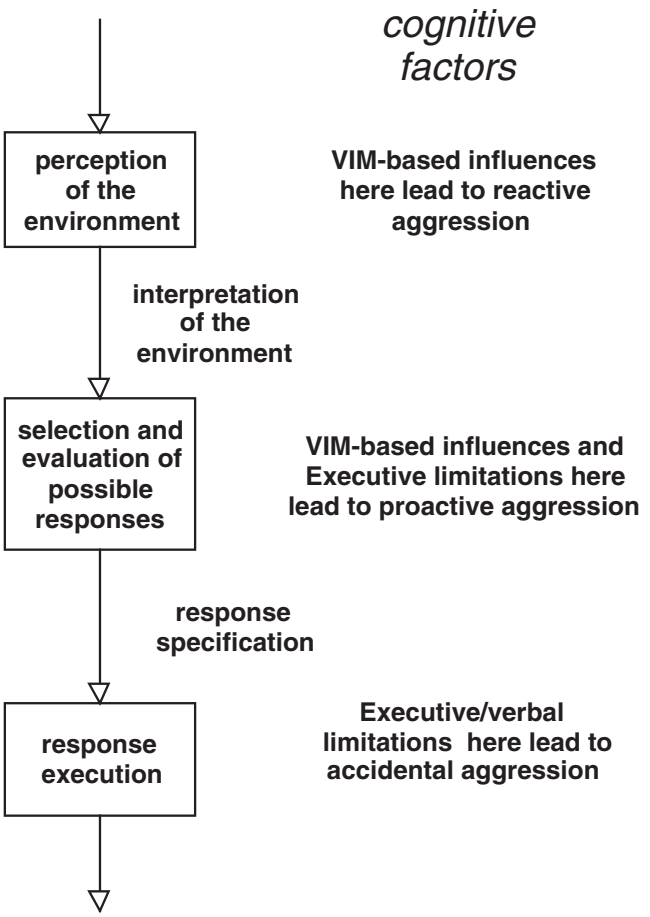

Figure 9 Integrative information flow model

on the one hand and Caspi and Moffitt on the other can be used to flesh out Dodge's theory. We thus end up with two models which link together the four theories, at least in principle. One of these is an information flow model and the other is a causal model.

We are aware that we were not exhaustive and that other theories of CD have been formulated (e.g., Raine, 1993). We invite the reader to use the Causal Modelling framework to represent this and other theories. Use of a common framework facilitates comparison of different theories and helps to see them both as competitive and cooperative, as we hope to have illustrated already.

The term 'cognition'

As we read the literature on CD it became apparent that the term 'cognition' has a different meaning for certain groups. Specifically, Patterson uses the term to refer to conscious processing. This is not the sense in which it is used by Morton and Frith. In common with the practice among cognitive psychologists, they use the term *cognition* to refer to all mental processes, whether conscious or unconscious – however etymologically unsound such a use might be. When Patterson makes the point that children of certain kinds of parents acquire habits which are not cognitively mediated, he means that the child's behaviour is not mediated by conscious decision making. He also claims that certain kinds of early child–parent interaction are not registered in memory. We suppose that what he means by this is that the individual parent–child transactions cannot be retrieved from episodic memory for conscious recall. From the context of cognitive psychology, this is probably equivalent to claiming that the child forms a schema of the repeated parent–child interaction. This schema would be accessed automatically under the appropriate environmental conditions and would control behaviour without conscious intervention. Such schema are seen as part of our cognitive apparatus (Mandler, 1985).

Moffitt uses the term 'behavioural development' in what we think is a similar way. She uses the term to refer to *speech* and *response inhibition*, both of which we would include inside the heading Cognition. For us, *behaviour* is what we see. It is the source of our data apart from biological observations. All test results are behaviour and are to be carefully contrasted with the cognitive abilities they may be intended to assess. Morton and Frith (1995) also stressed the importance of avoiding the confusion of a test result with a cognitive ability. The reason for this is that tests, as with all psychological tasks, are complex and involve the use of more than one cognitive process. Tests are designed to estimate particular abilities over a target population, usually the normal population. To give an example, the picture-word vocabulary test gives a very good estimate of general intelligence for the normal child population. However, for any child with a specific language deficit, this test will seriously underestimate intelligence.

A final point is to a difference between biological and cognitive factors on the one hand and behaviour on the other. It must be remembered that behaviour is ephemeral. What persist in the individual, and what change as a result of development or as a result of intervention, are the biological state and the cognitive state. Caspi and Moffitt (1995) make the same point when they comment:

> Although antisocial *behavior* is not inherited, a variety of biological traits may underlie the heritability coefficients for antisocial behaviour by predisposing persons to develop an antisocial phenotype. (p. 474)

As we pointed out in the introduction, it might be necessary to have a separate causal model for different ages, showing how different behaviour can arise from a common core deficit in interaction with time-sensitive factors (see Figure 1).

Implication for clinical practice

We started this modelling exercise partly from a clinical background and with a practical goal, i.e., to find a method that could support clinicians in using and comparing causal theories in the individual

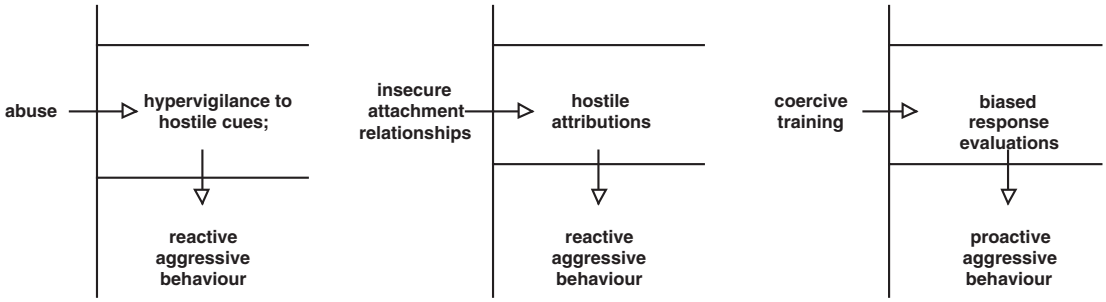

Figure 10 Representation of three causal models for three individual cases

case. Now, is the Causal Modelling framework a good candidate for this goal? The application of causal modelling to theories of CD makes explicit the intricacies of testing clinically relevant hypotheses in the individual case. But it does not just highlight these intricacies, it also offers the clinician a tool for doing his or her work as a scientist-practitioner. In clinical practice the clinician is confronted with the disturbed behaviour, e.g., aggressive behaviour in an individual child. The diagram we made from Dodge's theory in Figure 3 shows the different causal factors that could be involved in any individual case and could be used to guide the clinician in making testable predictions. This framework in Figure 3 shows that a possible explanation of aggressive behaviour is hypervigilance to hostile cues caused by an abusive parent. Once clearly formulated, this theory can be tested. An alternative account for that individual might be that he or she has hostile attributions due to insecure attachment relations or a biased response repertoire as a consequence of coercive parenting. Explanations in individual cases may be different, although they could all in principle account for the target behaviour. So for three children all behaving aggressively we may have three different individual causal models (see Figure 10). To test a hypothesis about the cognitive cause of a disorder we have to predict and observe behaviours other than the problem behaviour. To illustrate the point in the case of the VIM deficit in psychopathy, we cannot use the behavioural symptoms of the psychopath as proof for the VIM deficit. We should use some other behavioural aspect that could be impaired as a result of a VIM deficit. The aspect used by Blair (1995) in developing his theory was the absence of a moral versus conventional distinction. Dodge uses a variety of experimental procedures to carry out diagnosis in a similar way (Dodge et al., 1995). A complete causal model of the disorder would include all of these outcomes.

We know from experimental studies that clinicians are inclined to be very selective in considering causal hypotheses. They mostly consider a few explanations based on their first impression and prefer a confirmatory testing strategy (see Garb, 1998 for a critical review). One of the most frequently recommended debiasing strategies is to consider alternatives (e.g., alternative diagnosis, alternative explanations) when making judgements and, according to Garb, empirical data suggest that clinicians become more accurate when they consider alternative explanations (Garb, 1998). We think that the Causal Modelling framework can be an efficient tool in doing so. Frith (1999) makes a similar point when she suggests that practitioners 'may find the Causal Modelling framework useful because it carries a reminder that the behaviour in question always has multiple determinants' (p. 200). In addition, Fredrickson and Cline (2002) use the model to describe the different causal influences at work in individual cases. They demonstrate how the framework can represent what is known about the complex pattern of strengths and needs of an individual and how it can guide the selection of hypotheses about which particular influences are operating and how to set up the appropriate treatment. The systematic representation and comparative presentation of clinically relevant theories will not just help the clinician in choosing an accountable strategy, it will also allow them to critically evaluate the theories and to ask their colleague scientists to be more explicit whenever necessary. We hope that this will contribute to a more balanced discussion between theoreticians and practitioners to the benefit of both.

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