

## Causal processes in development and psychopathology<sup>†</sup>

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During the past two decades developmental psychopathology, which is the study of the origins and course of individual patterns of behavioural maladaptation, has emerged as a new science. It brings together and integrates a variety of disciplines, including epidemiology, genetics, psychiatry, psychology, the neurosciences and sociology. There are many features of developmental psychopathology that could make it important, but the defining features can be reduced to three key issues (Rutter & Sroufe, 2000). The first is the understanding of causal processes. It is now widely understood that most mental disorders are not due to single linear causes. Individual risk factors are seldom powerful. More often, psychopathology arises from the complex interplay of multiple risk and protective factors, some genetic and others environmental. A second central concept, which is at the core of much developmental research, is an emphasis on understanding the processes of development and investigating the emergence of patterns of adaptation and maladaptation over time. Developmental analyses therefore tend to be progressive, with one step leading to another. It is recognised that the mechanisms of causation may involve dynamic processes over time, with several routes to the same outcome. A third key concern has been a focus on the links between normality and pathology. Much causal research in psychiatry has been based on the idea that diagnostic categories represent some kind of reality or 'truth' distinct from normal behaviour. By contrast, many developmental psychopathological concepts are dimensional, with the need to take account of variations along dimensions.

We have been fortunate in receiving a large number of high-quality papers on developmental psychopathology and these will therefore be presented in two special

sections of the *Journal*: the first section includes papers on causal processes, whereas the second section will deal with developmental continuities and the links between normality and psychopathology.

This first section begins with risk research because some of the earliest developmental research involved the follow-up of high-risk samples. It has been known for a long time that profound privation in early childhood increases the risk of subsequent psychopathology. However, it is not clear whether all kinds of psychopathology are increased or whether there are certain patterns that are especially common. In a follow-up of institution-reared Romanian children, Rutter *et al* (2001, this issue) show that certain kinds of problem were more common in deprived children than in non-deprived UK-adopted children. These problems included attachment disorders, inattention/overactivity and quasi-autistic behaviour. There was no increase in the risk of other problems such as conduct problems. Clearly, there can be considerable specificity in some of the pathways linking early adversity to subsequent problems.

Less-specific risk pathways are the focus of the paper by Hill *et al* (2001, this issue). They use a study of women aged 25–36 years to draw out lessons about the different routes from childhood adversity to depression in adult life. The risk of depression associated with childhood sexual abuse was unaffected by quality of adult relationships, whereas the risk of depression associated with poor parental care was substantially altered. The implication is that subgroups of individuals manifesting similar problems can arrive at them via different routes. There could be important lessons for preventive strategies.

In both of these studies there was great variation in children's responses to adversity, with many of them escaping subsequent dysfunction. O'Connor *et al* (2001, this issue), in a study of children

from stepfamilies, single-parent families and non-stepfamilies, report similar findings. Differences in the level of child psychopathology within the same family were as large as differences between children in different families. Even children in the same family responded differently to parental separation or re-partnering.

The question therefore arises as to the nature of the factors that could influence individual differences in susceptibility to environmental risks. Over the past 10 years one of the major developments in the study of individual differences in behaviour has come from sophisticated quantitative analysis of data from genetically informative designs such as twin studies. The paper of Silberg *et al* (2001, this issue) is based on data from the Virginia Twin Study of Adolescent Behavioral Development, which is one of the largest and most systematic of the extant twin studies. They show that the effects of independent life events on adolescent depression depended to a large extent on the presence of parental emotional disorder. It seems that genetic factors play a role in individual reactions to environmental adversity.

The other major development in genetics has been the accelerating process of using molecular genetic techniques to analyse the human genome. Asherson & Curran (2001, this issue) describe how molecular genetic research has been applied in the field of development and psychopathology. Genetic studies have already resulted in the identification of susceptibility genes for autism, attention-deficit disorder and reading retardation. Nevertheless, for many of these disorders the amount of variance explained by the identified genetic loci has been quite small, suggesting either that there are multiple genes of small effect or that there are gene–gene or gene–environment interactions. Hyperphagic short stature (HSS), which is the subject of the paper by Gilmour *et al* (2001, this issue), could be an example of a syndrome that results from gene–environment interaction. The clinical features of HSS include stunted growth secondary to growth hormone deficiency and excessive appetite. Affected children often live in conditions of high psychosocial stress, and symptoms resolve when the child is removed from the stressful environment. Hyperphagic short stature is rare but important because it is one of only a very small number of behavioural phenotypes associated with a defined physiological mechanism.

<sup>†</sup>See pp. 97–163, this issue.

All of these genetic studies suggest, then, that it is necessary to go beyond questions about the proportion of variance explained by genetic or environmental factors, to find out how the causal processes actually operate. The study by Rubia *et al* (2001, this issue) illustrates how modern psychological methods can be combined with neuroimaging techniques to study these processes. They found that children with attention-deficit hyperactivity disorder (ADHD), but not psychiatric controls, were impaired on most tests of response inhibition. Moreover, a parallel neuroimaging study showed right pre-frontal impairments in hyperactive adolescents in higher-level tasks of inhibition. These findings are consistent with other research, suggesting that many of the basic processes of attention are intact in children with ADHD and that the disorder is associated with cognitive problems of a higher order than previously thought. Eventually, these studies could lead to new kinds of treatment for a common and disabling disorder.

Causal processes of a different kind are the focus of the paper by Boyce *et al* (2001, this issue). Their study aimed to establish measures of autonomic nervous system reactivity that could be used eventually to identify young children with early signs of developing psychopathology. Using state-of-the-art methods they found that measures of autonomic reactivity discriminated significantly between children with behavioural problems and those with emotional problems. Children with emotional problems showed high reactivity, particularly of the parasympathetic branch, whereas children with behavioural problems showed low reactivity in both sympathetic and parasympathetic branches.

Developmentalists have, of course, been much concerned also with the ways in

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which environmental factors can influence the risk of psychopathology in children. A variety of research designs have been used but one of the most common has been to study the children of parents with mental disorders. The paper by Essex *et al* (2001, this issue) describes a large and systematic longitudinal study of infants who had been exposed to maternal depression at different ages, or not at all. The impact of maternal depression was more severe with exposure in infancy than with exposure during the toddler/pre-school period. This highlights the importance of age-defined periods of especial vulnerability ('sensitive periods') to particular kinds of adversity.

But how exactly does exposure to parental mental disorder during a sensitive period lead to later psychopathology? Many processes are likely to be involved, one of which is probably through parent-child interaction. The study by Stein *et al* (2001, this issue) illustrates the kinds of intensive observations that are necessary in order to study this interaction. Their research shows how easily parental psychopathology (in this case, eating disorder) can extend to parenting.

## DECLARATION OF INTEREST

None.

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applications in child psychiatry and psychology. *British Journal of Psychiatry*, **179**, 122–128.

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