How the environment affects mental health*

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For many years there was an assumption that the extensive documentation of statistical associations between risky environments and mental disorders necessarily represented the operation of environmentally mediated causal mechanisms. Three considerations challenged that assumption. First, psychosocial researchers recognised the need to differentiate between risk indicators (features that indexed risks but did not themselves provide the risk) and risk mediators (features involved in the actual risk processes leading to mental disorders). Thus, in the 1970s it became apparent that the main risk for antisocial behaviour associated with 'broken homes' was a function of family discord and conflict, rather than family break-up as such. Similarly, in the 1980s it was shown that the risks of depressive disorders in adult life were a function of impaired parenting, rather than parental loss. As part of this same issue, it came to be appreciated that distal risks needed to be differentiated from proximal risks. Thus, poverty constituted a distal risk for child mental disorder because it made good parenting more difficult, but the proximal risk mediator involved family malfunction rather than lack of economic resources.

Second, Bell (1968) emphasised that children had effects on their parents, just as parents had effects on their children. The association between family features and child disorder could not simply be assumed to reflect adverse socialisation practices; instead it might derive from the effects of a difficult child on family functioning. Longitudinal data were essential to determine the direction of the causal arrow. Third, twin and adoptee studies showed that, even though risks were due to an environmental feature, the risks might nevertheless be genetically mediated in part (Plomin & Bergeman, 1991) – because, if the environmental feature concerned anything that was influenced by parental behaviour (as would be the case with variables such as family conflict, divorce or parent–child interaction), individual differences in such behaviour were likely to be genetically influenced to some extent. Study designs were needed that could differentiate between genetic and environmental mediation. Twin and adoptee strategies of various kinds provide just that possibility, and they have produced good evidence of the reality and importance of environmentally mediated risks for psychological and psychopathological outcomes (Rutter, 2004*a*). However, they are by no means the only relevant designs; psychosocial researchers have also pioneered the use of 'natural experiments' of diverse kinds, their common feature being that they involved a radical change of environment, and a pulling apart of variables that ordinarily go together, the effects of which could be studied by measuring within-individual change investigated through the use of longitudinal data. By these means, environmentally mediated risks have been demonstrated for various aspects of the family rearing environment, and also for peer group, school and community influences.

Four features of the research findings need to be particularly highlighted. First, despite some claims to the contrary, environmental influences have been found to operate within the normal range, and not just in relation to extreme environments (although, for obvious reasons, the effects of the latter are greater). Second, environmental effects have been shown not only for influences in infancy, but also for influences in middle childhood (Duyme *et al*, 1999) and even in adult life (Laub *et al*, 1998). Third, the environmentally mediated risks include prenatal influences (such as maternal drug and alcohol use and severe maternal stress) and postnatal physical influences (such as brain injury and adolescents' heavy early use of cannabis). The span of risk influences is substantially wider than has sometimes been assumed. Fourth, with all known environmental hazards (both physical and psychosocial) there is a huge individual variation in response (Rutter, 2004*b*): some individuals succumb; some appear remarkably resilient; and a few even seem strengthened as a result of having coped successfully with stress and adversity. It might be supposed that the individual differences merely reflect variations in the severity and number of risks involved, but experimental studies in both animals and humans have shown that this does not account for the phenomenon of resilience (despite the fact that some studies were flawed by a failure to assess the severity of risk satisfactorily, and/or by a failure to examine an adequate range of outcomes). The features underlying the individual differences include strengthening (or weakening) experiences prior to risk exposure, protective influences operating at the time of risk exposure, and recuperative positive turning-point experiences subsequent to the experience of risk. However, a key influence that has been highlighted by recent research (see Rutter, 2004*a*) is genetically influenced vulnerability to (or protection against) environmental risk.

SHARED AND NON-SHARED EFFECTS

Plomin & Daniels (1987) argued that environmental differences among families were of little consequence and that attention needed to be focused on child-specific environmental influences, because environments tended to make children in the same family different. The paper was helpful in its emphasis on the need to measure putative psychosocial influences as they actually impinge on individuals (reiterating a message from psychosocial researchers over 20 years earlier who developed the person-specific measure of negative expressed emotion). However, the distinction between shared and non-shared environmental effects, which was central to the paper, has held back progress because of the ways in which it was interpreted. Many reviewers (both geneticists and others) have supposed that the findings meant that family-wide influences had little effect on either psychological development or risk of psychopathological disorder. In fact, the research shows nothing of the kind. The shared *v.* non-shared distinction has

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nothing to do with whether the influences are or are not family-wide, and indeed has nothing to do with whether the influences are within or outside the family. The distinction is solely concerned with whether the environmental influences tend to make siblings similar or different. Child-specific experiences within the family (such as abuse or parental negativity) may nevertheless have a largely shared effect if the experiences of the siblings are sufficiently similar; see Pike *et al* (1996) for an example. The same would apply to peer group experiences if they were similar for different siblings. Conversely, family-wide influences (such as poverty, conflict or neglect) might have largely non-shared effects if the key features impinge on the children to differing degrees or in varying ways, or if the children vary in their vulnerability to risk environments. It is also pertinent that the relative importance of shared and non-shared effects varies according to type of psychopathology – so that shared effects are more important in relation to antisocial behaviour than to depression. The message to researchers is to measure environmental influences in individual-specific ways but not to assume that this means that overall family influences are unimportant. Similarly, the message to clinicians is to consider how risky environments actually impinge on, and affect, individual children (or adults), but not to assume that family-wide risks do not matter.

A somewhat related issue concerns the distinction between environmental effects on the level of a trait, or the frequency of a disorder, rather than on individual differences with respect to that trait or disorder. Thus, over the past half-century there has been a substantial rise in the rate of many types of mental disorder in young people (Collishaw *et al*, 2004). The causes of the rise remain ill-understood but the environmental factors involved urgently require investigation. The same applies to the higher rate of schizophrenia in individuals of Caribbean origin compared with ethnically similar individuals living in the West Indies or with White people living in the UK (Jones & Fung, 2005). Some sort of society-wide influence seems to be implicated, but it has yet to be identified.

RESEARCH CHALLENGES STILL TO BE MET

What are the main challenges ahead? Three stand out. First, there is a need for a better understanding of the kinds of environmental influences that have major risk effects. The evidence so far suggests that these include restrictions on the possibility of developing intense selective social relationships (as with institutional rearing), severe disruptions in the security of such relationships (as with neglect, rejection and scapegoating), life events that carry a long-term threat to such relationships (as with humiliating experiences, personal rebuffs or rejections), and social ethos or group influences of a maladaptive kind (as with antisocial peer groups or malfunctioning schools). Also, however, the overall quality of adult–child interaction and communication has been shown to matter. In addition, it is evident that both prenatal and postnatal influences that affect neuroendocrine or neurotransmitter functions are important.

The second challenge is to identify the origins of environmental risk factors, whether they lie in gene–environment correlations (so that genetic factors have their impact on behaviours that shape or select environments and, thereby, influence the likelihood of experiencing stress or adversity), societal elements (such as racial discrimination, poverty or housing policy) or personal experiences.

The third challenge is to determine the changes in the organism that provide the basis for the persistence of environmental effects on psychological functioning or psychopathology. In many respects, this constitutes the environmental equivalent of sequencing the human genome (i.e. the basic need). There is a major Canadian initiative on this topic (the Canadian Institute for Advanced Research consortium on 'Experienced-based brain and biological development'), but regrettably the UK is lagging behind. Several different types of mediation need to be considered. Exciting findings from Michael Meaney's research group have shown that environmental influences affect gene expression through influences on methylation (Weaver *et al*, 2004); in other words, environments affect genes – not through effects on gene sequence but through effects on gene expression (which is how genes act). Environments also affect the programming of brain development (Rutter, 2004*c*); this was shown first with respect to vision (leading to a Nobel prize for Hubel and Wiesel), but it is now clear that it applies more widely. Furthermore, environments affect neuroendocrine structure and functioning and, through such effects, may influence brain development. Experiences may affect patterns of interpersonal interaction that become influential through their role in the shaping of later environments; in addition, experiences have to undergo cognitive and affective processing, so that what happens to individuals influences their mental concepts and models of themselves and of their environments. The relative importance of these, and other, possibilities with respect to different outcomes has yet to be established. The questions are answerable, and require the bringing together of genetic, social and developmental perspectives in an integrated fashion. If this is to happen, funding agencies will need to take on the challenge of supporting research that can tackle these questions. Meanwhile, the message to clinicians is to consider the important interplay that shapes environmental effects.

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