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Michael Rutter Professor
[a] Institute of Psychiatry, King's College, London, UK

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Multiple meanings of a developmental perspective on psychopathology*

Michael Rutter
Institute of Psychiatry, King's College, London, UK

This review starts with a discussion of what is meant by developmental change. It is concluded that development is an inherently "fuzzy" concept but that it constitutes a useful framework for research into psychopathology. Some of the major changes over the last four decades in the approach taken for the study of development are noted. Ten key developmental issues are discussed: (1) prenatal influences; (2) sensitive period effects; (3) mechanisms mediating long-term effects of experiences; (4) age of onset differences; (5) sex differences; (6) normality and disorder; (7) connections among different psychological domains; (8) psychopathological progressions; (9) resilience; and (10) gene–environment interplay. It is concluded that developmental research is a rich field of high potential but it needs to be process-oriented rather than norm-oriented, it needs to focus on interconnections between brain and mind, and it needs to have a major interest in individual differences.

INTRODUCTION

Over the last 40 years there have been huge changes in the style of research approaches to the study of development. First, in the 1960s the main focus was on supposed normative changes, whereas now there is a strong interest in both the nature of individual differences and their origins. Second, an implicit acceptance that age constitutes a sufficient explanation for development has given way to a systematic search for a range of causal processes that play a part in the continuities and discontinuities in development. Age indexes, among other things, are: physical maturation; hormonal state; cognitive level; social circumstances; and life experiences (Rutter, 1989a). Accordingly, it is necessary to ask which of these features may be responsible for any age differences found with respect to a particular psychological or psychopathological function. The third major change is

Correspondence should be addressed to Professor Michael Rutter, PO 80, SGDP Centre, Institute of Psychiatry, Denmark Hill, London SE5 8AF, UK. Email: j.wickham@iop.kcl.ac.uk

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that, 40 years ago, almost all developmental research was undertaken with the expectation that it needed to deal only with the mind. Currently, as developmentalists became aware of the importance of biology, attention increasingly shifted to a search for the mechanisms involved in brain–mind relationships.

The magnitude of the change over time is well reflected in a comparison of the empirical papers published in *Child Development* in 1960–1961, and those published in 2000–2001. In 1960–1961 less than one in twenty empirical papers used longitudinal data, whereas in 2000–2001 over a third did so. There was more than a doubling of the proportion of papers concerned with one or other aspect of psychopathology and in 2000–2001 there was just the very beginning of an interest in genetics. This applied to only a handful of empirical papers but it had applied to none 40 years earlier. The other trend was the huge increase in international authorship. In the earlier period, scarcely any papers involved an international authorship, whereas in 2000–2001 nearly one in five did so. This was partly because child development in the United States had become much less inward looking, and in part it reflects a growing strength in European developmental psychology but, perhaps most of all, it reflects an increasingly positive attitude to the value of collaborations across disciplines and across different research centres. The key objective of this paper is to consider why and how developmental perspectives on psychopathology are important. The starting point, however, has to be the concept of development itself. Most exhortations to adopt a developmental perspective fail to specify what that means and it is obvious that many of the concepts of development are misleadingly simplistic.

WHAT IS MEANT BY DEVELOPMENTAL CHANGE?

Thus, the first question is what defines developmental change. In what way does development differ from non-developmental change? At first sight, it would seem obvious that the definition should involve reference to gains in skills or gains in capacity, but that does not work. That is because losses, as well as gains, are a normal part of development. That is evident, for example, in the neuronal pruning that constitutes an integral part of normal brain development (Curtis & Nelson, 2003; Huttenlocher, 2002) and it is also apparent in the loss of phonological discriminatory skills that are present in infancy but disappear if the sound differentiations are not part of the language of rearing (Rutter, 2002a). In addition, we need to appreciate that development is not just concerned with skills and capacity. For example, the development of the immune system is concerned with changes in the ways that the body deals with infections and this cannot sensibly be reduced to concepts of skills and capacity. In the psychological arena the

same would apply to emotional development. Of course, as they grow older, young children become better able to "read" other people's emotions and to understand what they might be feeling but that definitely does not constitute the whole of emotional development. It might also be thought that development needs to be considered in terms of more of the same, but obviously that is not right either. For example, the transition from the caterpillar to the butterfly involves radical change that is far from more of the same. In the human case, the onset of fertility at the time of puberty would be a similar example where there is the emergence of a new function that is not simply a growth in a pre-existing one.

It is tempting to try to define psychological development in terms of biological maturation because it is obvious that psychological development is crucially tied up with biological maturation. However, that does not easily translate into any kind of readily applied criteria because experiences affect some aspects of brain development. This was shown years ago with respect to the role of visual input in the development of the visual cortex but it is now known that the effects of experiences on brain development extend well beyond vision (Knudsen, 2004; Rutter, in press a). It is relevant that all forms of psychological functioning are affected by nurture as well as by nature; and psychological development cannot take place without genetically influenced biological maturation, but the biological changes are far from independent of environmental input.

A further temptation would be to seek to define development in terms that confined it to processes that are exclusively normal. Such an attempt would clearly run counter to all that is involved in the concept of developmental psychopathology (Rutter & Sroufe, 2000; Sroufe & Rutter, 1984) but, more importantly, there is no clear-cut categorical differentiation between normality and disorder. Thus, first, it is well established that there are continuities, as well as discontinuities, with respect to both depression and aggression or antisocial behaviour. At one extreme, depressive disorder is a serious life-threatening condition that involves numerous biological changes. At the other extreme, feelings of misery, and even despair, are a normal part of the human condition that most people experience at some time in their life. The point here is that there is no straightforward point at which one can say normality ends and pathology begins. Subclinical manifestations of depression constitute the precursors of overt major depressive disorders. The categorical distinction that is implicit in the diagnosis of a condition that is pathological is a meaningful one because a point comes when suffering, functional impairment, or suicidal risk means that treatment is essential. However, from the perspective of the development of that condition, continuities with normality are evident. Exactly the same applies to aggression and antisocial disorder (Rutter, Giller, & Hagell, 1998).

Second, plasticity and adaptation to injury or stress is a key feature of normal development. That is to say, as children grow up, they must expect to encounter degrees of trauma and adversity that require adaptation and coping. But, even beyond that, it is clear that the response to more major injuries involves the plasticity that is inherent in normal brain functioning. The example of the changes in response to unilateral brain lesions, with respect to the acquisition of aphasia, constitutes a dramatic case in point (Rutter, 1993). Equally, psychopathological progressions in serious mental disorders may be crucially dependent on aspects of brain development. Thus, for example, although the psychosis of schizophrenia does not usually begin until late adolescence or early adult life, the neurodevelopmental features that predispose to schizophrenia begin much earlier, in childhood (Keshavan, Kennedy, & Murray, 2004).

The very term ''development'' seems to imply that it ought to have a fixed end point that reflects the time when maturity is reached. That sounds reasonable, and there is something valid in the concept, but it is not clear how to define or measure psychological maturity. Thus, for example, how might that criterion be applied to the development of social relationships? Many adults remain rather inept in their social relationships. Does that mean that they are in a real sense ''immature'', or does it mean that they have some disorder, or, rather, does it imply that there is an artificiality to the concept of a maturity-fixed end point for social development? In addition, some aspects of psychological development may be dependent on late-occurring experiences. Thus, for example, this might well apply to sexuality and to childbirth. Do individuals who remain celibate necessarily lack maturity? It is evident from many studies that the experience of childbirth brings psychological changes but does that mean that the women who do not bear children are in some meaningful sense thereby immature? The concept of maturity with respect to psychological development is not wholly wrong but it does not seem to provide a criterion that is at all easily operationalized.

Finally, it might be thought that development could be defined in terms of the permanency of change. Thus, unless there is some manifest disease, once individuals have reached their adult height, they do not subsequently lose it (except in old age). By contrast, the changes that come with disease are usually lost (at least to a considerable degree) once there is recovery from the illness. However, yet again this does not provide a satisfactory way forward. To begin with, many changes due to disease are permanent. For example, that is obviously the case with Alzheimer's disease. Second, many developmental changes are not permanent. Mention has already been made of the neuronal pruning that follows the initial overgrowth of neurones in early life. Similarly, the loss of fertility at the menopause is part of a normal developmental process and is not a consequence of disease. An additional

problem concerns the uncertainty over how to conceptualize permanent changes stemming from experiences in adult life. For example, the advent of brain imaging has enabled studies to show substantial structural changes in the brain that derive from experiences in adult life. For example, this was evident in the changes in the hippocampus that were consequent upon the extraordinarily detailed knowledge of routes through London that is part of what is required of London taxi drivers (Maguire et al., 2000). Comparable changes have been found with skilled violin players (Elbert, Pantev, Wienbruch, Rockstroh, & Taub, 1995).

It would be satisfying to have been able to come up with a definitive definition of what is meant by development, but, in my view, it is an inherently "fuzzy" concept and although there are important distinctions between development and non-developmental change, the two overlap to some extent. The key needs are for research into the causal processes of both, together with an investigation of the continuities and discontinuities between normality and pathology. A working definition of development put forward a dozen or so years ago provides the guide to what is involved in concepts of development: "systematic, organized, intra-individual change that is clearly associated with age-related progressions and which is carried forward in some way that has implications for a person's pattern of functioning at some later time" (Rutter & Rutter, 1993). The emphasis on systematic organization does not, of course, imply either fixity or inevitability. Equally, it incorporates recognition of the role of chance events and the role of thought processes in bringing about action (Dennett, 2003). The key point, as expressed by Sroufe and Rutter (1984) is that the process has a unifying coherence. As Morange (2001) put it more recently, biology provides a regular pattern and an organized system of mechanisms. It is dynamic and probabilistic, not deterministic, in its effects, but it is systematic and orderly (see also Rutter in press b).

The ten key developmental issues that constitute some of the multiple facets of a developmental perspective on psychopathology to be discussed are: (1) prenatal influences; (2) sensitive period effects; (3) mechanisms mediating long-term effects of experiences; (4) age of onset differences; (5) sex differences; (6) normality and disorder; (7) connections among different psychological domains; (8) psychopathological progressions; (9) resilience; and (10) gene–environment interplay. They have been selected on the basis of their importance in reflecting the broad span of developmental perspectives, their implications for psychopathology, and the fact that each of them involves major recent advances in conceptualization and in empirical findings. The purpose of the paper is to consider these advances with the aim of identifying some research priorities. It is evident that the findings have implications for prevention and treatment but these are outside the scope of this paper.

PRENATAL INFLUENCES

Some half a century ago, paediatricians were very concerned over the supposed ill effects that derived from pregnancy complications, and there was much talk of the phenomenon of birth injury. Initially, the focus was primarily on cerebral palsy and mental retardation, but, particularly through the pioneering work of Pasamanick and Knobloch (1966), people came to accept the notion of a "continuum of reproductive casualty". The argument was that if severe reproductive complications led to devastating outcomes, it was likely that lesser complications would result in lesser degrees of brain injury with consequent effects on behavioural functioning and psychological development. This concept failed to stand the test of time for two main reasons. First, evidence accumulated that many of the supposed consequences of birth injury actually derived from the problems arising at a much earlier stage in gestation (Nelson & Ellenberg, 1986). Second, however, insofar as much of the evidence stemmed from statistical associations with premature gestation and low birthweight, the inference that these had led to brain injury was circumstantial at best. The uncertainty of this inference was underlined by the quite strong associations between social disadvantage, and the variety of circumstances that went along with it, and the rate of premature gestation and low birthweight. The query, therefore, was whether the sequelae derived from the social circumstances or from the effects on brain functioning. Because of these important doubts, attention rather turned away from the developmental consequences of prenatal and perinatal abnormalities.

The pendulum has now swung back to an important degree as a result of several research developments. First, the technological development of imaging techniques—especially ultrasound as used during the period of pregnancy and magnetic resonance imaging as used in childhood—has transformed the situation by providing the means of actually measuring brain effects rather than having to infer them indirectly. The evidence has accumulated to document the reality and frequency of anomalies in brain structure associated with very low birthweight and very premature gestation (Cooke & Abernethy, 1999; Fearon et al., 2004; Stewart et al., 1999). Follow-up studies have also shown that the deficits include behavioural problems and specific learning deficits as well as cerebral palsy and severe mental retardation (Marlow, 2004; Marlow, Wolke, Bracewell, & Samara, 2005). However, what has remained rather a puzzle is the somewhat inconsistent associations between the brain imaging findings and the measures of psychological function and dysfunction.

The evidence on prenatal influences is noteworthy for several different reasons. First, it is evident that, in considering the possible environmental influences on psychological development, influences during the baby's

period in the womb must be included (Coe & Lubach, 2005). Second, it seems that there may be effects from maternal stress during the pregnancy (O'Connor, Heron, Golding, & Glover, 2003) as well as the better-documented physical effects of toxins such as drugs and alcohol (Chasnoff et al., 1998; Koren et al., 1998; Mayes, 1999; Streissguth, Barr, Bookstein, Sampson, & Olson, 1999). Of course, the stress effects do not imply that the foetus appreciates or recognizes the mother's emotions during the intra-uterine period. It is more likely that the effects are mediated by stress-generated hormones that cross the placental barrier (Maccari et al., 2003). Third, animal studies provide a clear indication that prenatal male sex hormones have a lasting effect on brain development (Hines, 2004). The same seems to apply in humans to some extent, with implications for sex-differentiated behaviours. Fourth, the importance of individual differences in response to prenatal, as well as postnatal, experiences needs to be recognized, together with the appreciation that gene–environment interplay is likely to be implicated. For example, Kahn, Khoury, Nichols, and Lanphear (2003) showed that prenatal smoking exposure had a significant effect on hyperactivity/impulsivity and oppositional behaviour only for individuals who were homozygous for the DAT genotype. Also, though not as yet demonstrated, it is quite likely that sex-hormone effects may operate through influences on gene expression (Petronis, 2001). The key messages are that prenatal influences may well have a greater effect on psychopathology outcomes than has usually been assumed; that the mediating mechanisms need to be considered developmentally; and that follow-up studies need to include neuroendocrine immunological and brain-imaging measures, and use molecular genetic research strategies.

SENSITIVE PERIOD EFFECTS

Some half a century ago, many developmentalists operated on the assumption that critical periods in development were crucially important, and that the key critical period was the first two or three years of life. Imprinting was seen as the general model for effects that operated only during the early period of development and which were permanent in their consequences. Animal studies then cast doubt on the fixity of supposed critical period effects (Bateson, 1966) and human studies emphasized the role of experiences at all ages and not just in early life (Clarke & Clarke, 1976). It was not that the imprinting phenomenon was unreal and it was not that there were no measurable effects on the brain. To the contrary, careful experimental studies were instrumental in showing the neural basis of imprinting (Horn, 1990). Nevertheless, the generality of the effect was called into serious doubt and critical-period concepts went out of fashion.

During the last decade or so numerous studies in both humans and other animals have revived interest in age-specific experiential effects and have produced extensive evidence of their reality and importance. The terminology has changed from "critical periods" to "sensitive periods" in response to the evidence that the fixity of the earlier critical period concepts was mistaken, but the importance of developmental-phase specificity of some experiential effects (but not all) is valid.

It is useful to differentiate between what Greenough has called experience-expectant effects (Greenough, Black, & Wallace, 1987) and what others have called experience-adaptive effects (Rutter, in press a; Rutter, O'Connor et al., 2004a). The best known, and best demonstrated, example of experience-expectant effects concerns the role of visual input in the development of the visual cortex. The importance of this was first shown by Hubel and Wiesel (1965, 1970) in the work that led to their receiving the Nobel prize. What their experimental studies demonstrated was that patterned visual input was essential for the normal development of the visual cortex and that there needed to be co-ordinated visual input from both eyes in order for binocular vision to be established. The practical implication for human development is that if strabismus (meaning a visual squint) is not corrected in the first few years of life, normal binocular vision is unlikely to become possible later. The findings are crucially important in their demonstration that normal brain development is dependent on experiential input. Unfortunately, however, these findings have been generalized by many psychological commentators in highly misleading ways (see Bruer, 1999, for a critique).

To begin with, the environments that are adequate to provide for normal brain development cover an extremely broad range. The term "experience-expectant" was deliberately chosen to indicate that, under all normal circumstances, it may be expected that the environments needed will be available. The findings have no implications for the effects of variations within a wide normal range. Second, experience-expectant effects do not apply to all systems. It is known that they apply to vision and it is possible that they apply to aspects of social development and social experiences (Rutter, in press a), but it is dubious whether they extend to other aspects of psychological development. The conclusion must be that experience-expectant effects are crucially important for some systems but are probably the exception rather than the rule.

Experience-adaptive effects are different in several key respects. The general notion is that biological development is programmed to be adaptive to the particular environments experienced during the period when the relevant bodily systems are being established (Bateson et al., 2004; Bateson & Martin, 1999). The example that is best known concerns the effects of early subnutrition. Numerous studies have shown that babies who are

severely underweight at birth and during the first year of life have a much increased risk of later coronary artery disease, hypertension, and diabetes. Barker and others (Barker, 1997, 1999) have hypothesized that this risk effect comes about because the body is programmed, in these circumstances, to respond optimally to a subnutrition diet. If later diets are normal, or even worse if they are super-abundant, the organism is maladapted to respond in an appropriate fashion and pathological changes occur. The underlying physiology for all of this remains somewhat uncertain but the empirical findings are not in serious dispute. It is particularly striking that the risks derive from being underweight in the neonatal period, whereas the risks for the same outcomes derive from being overweight in mid-life.

The best known psychological example is provided by the findings on the relationship between language input and phonological discrimination (Kuhl, 1994; Kuhl et al., 1997; Maye, Werker, & Gerken, 2002; Werker & Tees, 1984). During the first six months of life, or thereabouts, babies all over the world show similar skills in phonological discrimination. In sharp contrast, during the second half of the first year and onwards, phonological discriminations become increasingly affected by the particular language environment experienced by infants. This is illustrated by the well-known difficulty that most Japanese people have in differentiating between the sounds of "r" and "l". This distinction has no place in the Japanese language, whereas it is a key distinction in English and in many other European languages. An abnormal example of the same phenomenon is provided by the effects of profound deafness. The sounds made by profoundly deaf infants are not particularly distinctive during the first six months or so of life but become obviously abnormal from then onwards. It seems that sound production is driven by sound input only after about six months of age (Lenneberg, 1967; Murphy, 1964).

It is quite likely that there are rather more in the way of experience-adaptive effects. For example, they apply in the arena of immunology (Bock & Whelan, 1991) and they probably apply to the effects of stress experiences on the development of the structure and function of the neuroendocrine system (Hennessey & Levine, 1979).

Most of the literature on sensitive period effects has focused on the role of biology but it is important also to appreciate that developmental-phase sensitivity may derive from influences that encompass social context as well as maturation. For example, the demonstrated psychopathological risks associated with multiple hospital admission or the separation of children from their families for other reasons tend to be maximal in the preschool period after infancy (Rutter, 1979). It is likely that the relative protection during the infancy period derives from the fact that young babies have not yet developed strong, selective attachments to other people. The relative protection during the school-age period onwards probably derives from the

fact that, by that age, children are able to maintain relationships during the course of the separation and are also better able to understand the meaning of separations. These cognitive changes are, of course, driven by biological maturation but the evidence suggests that they are also influenced by social context and social experiences. For example, children's understanding of hospital procedures and treatment issues is affected by the extent to which these have been part and parcel of their experiences. Also, there are suggestions that children's responses to multiple hospital admissions may be influenced by the extent to which they have experienced previous happy separations (Stacey, Dernden, Pill, & Robinson, 1970) and by whether or not they have background experiences of chronic family adversity (Quinton & Rutter, 1976). The evidence on such social contexts and social experience influences is decidedly limited, but they warrant more systematic study than they have received up to now. The implication is that, in considering the psychopathological risks associated with early adverse experiences, greater attention needs to be paid to the possibility of sensitive period effects—recognizing that these could reflect either biological programming or the influences of social context and social experiences.

PHARMACOLOGICAL EFFECTS

Clinical observations have suggested important age differences in the ways in which individuals respond to drugs. For example, amphetamine is a seriously addictive drug in adult life as a result of its euphoriant effects. By contrast, amphetamine does not seem to have this effect in childhood when, if anything, there is a dysphoric effect of the drug. Although amphetamine has been extensively used in the past for the treatment of attention-deficit hyperactivity disorder (ADHD) and was also used in the treatment of nocturnal enuresis, it seems that the drug is not used recreationally in childhood and has a quite different effect on mood. Similarly, although tricyclic medication has a well-documented beneficial effect in the treatment of depressive disorders in adult life, the studies in childhood and adolescence mainly document the lack of significant benefits. Surprisingly, there has been little systematic human research into these age differences in drug response (although there is confirmatory animal evidence for some age-related differences—see Spear, 2000). Accordingly, age differences in pharmacological effects seem to be real and they are certainly in need of more systematic investigation in order to determine the mechanisms involved. Clinical/epidemiological studies have shown that heavy early use of cannabis has a significant provoking effect on the onset of schizophrenic psychoses in genetically vulnerable individuals (Arseneault, Cannon, Witton, & Murray, 2004; Caspi et al., 2005; Henquet et al., 2005). The effect is seen only with heavy use and not occasional recreational use but, in addition, it seems to be

largely associated with heavy use in early life rather than adulthood. In this instance, there is animal evidence that pubertal, but not adult, cannabis usage impairs cognition (Schneider & Koch, 2003). Again, further research is needed into the biological basis for this age difference. It will be relevant for consideration of both risk effects of substance use and the planning of pharmacological treatments.

MECHANISMS MEDIATING THE LONG-TERM EFFECTS OF EXPERIENCES

Developmental research has shown the reality of the long-term effects on psychological functioning of psychosocial experiences in childhood (Rutter, 2000; Rutter, 2005). It is not that such experiences invariably have long-term effects and it is not that they have the same effects in all individuals. Accordingly, it is crucial to ask questions about the mechanisms that could mediate such long-term effects when they are evident. It is apparent that several quite different types of mediation need to be considered (Rutter, 1989b). Six major alternatives serve to illustrate the range of possibilities that have to be examined.

Effects on gene expression

First, recent research on rodents by Meaney's research group has clearly shown the importance of experiential effects on gene expression. Cross-fostering designs have served to differentiate between environmentally mediated and genetically mediated risks and have shown that maternal nurturant behaviour in early infancy affects gene expression (Cameron et al., 2005; Weaver et al., 2004). The experiences do not alter the gene sequence and therefore the effects are not genetic. Rather, they influence the expression of those genes in particular tissues, through effects on methylation, which seems to be one of the prime processes involved in what has come to be called epigenetic effects (Rutter, in press b; Rutter, Caspi, & Moffitt, in press). The effect is a functionally important one because, although DNA constitutes the particles of genetic inheritance, the effects of the DNA are crucially dependent on processes leading to gene expression. DNA is present in all cells but its expression tends to be both tissue-specific and developmental-phase-specific, although frequently long lasting.

Biological programming

As already discussed, biological programming of various kinds constitutes a mechanism by which long-term effects may be brought about. Whether or

not these involve effects on gene expression is not known, but they may well do so. The long-term follow-up of children who spent their early years in extremely depriving institutions in Romania, but who were later adopted into well-functioning adoptive homes in the UK, points to the need to invoke some mechanisms of this kind (Rutter, in press a; Rutter, O'Connor et al., 2004a). Of all the features examined, the duration of institutional deprivation provided much the strongest effect on psychological outcomes. What was very striking, and rather surprising, was that the effects of duration of institutional deprivation proved to be nearly as strong at age 11 years as they had been at age 6 years and, before that, at age 4 years. The children had sometimes improved over time in their functioning but there was little diminution in the predictive effects stemming from the early environment (Beckett et al., 2005). This was so for cognitive outcomes as well as for social outcomes.

These effects and others like them are important (see Knudsen, 2004; Rutter, in press a, for some of the animal evidence) but it is crucial to avoid false extrapolations from neuroscience. It is often argued that, because brain development is maximal in early life, only experiences in the first few years are likely to have enduring effects resulting in permanent changes in brain structure and function (see Bruer, 1999, for a critique). This extrapolation is known to be false for several rather different reasons. To begin with, human studies have shown the major impact of environments in the period after infancy. For example, in the follow-up study of Romanian adoptees, there was a remarkable catch-up of psychological functioning following the children's removal from the depriving institutions and adoption into UK families (Rutter et al., 1998; Rutter, in press a). It was not just a temporary improvement, because the benefits were still evident at age 11 years. The effects of variations in the family rearing environment during the years of middle childhood has been shown most clearly in the study undertaken by Duyme, Dumaret, and Tomkiewicz (1999). They studied children who had been removed from their biological parents because of parental abuse or neglect, who had been adopted between the ages of four and six years, who had IQ testing prior to adoption and whose IQ was reassessed at follow-up in adolescence. The group as a whole, not surprisingly, showed a rise in IQ following adoption but what was particularly new and important in the findings was that the degree of improvement in IQ was systematically associated with the social and educational qualities of the adoptive home.

These studies showed the importance of later environments on psychological functioning but they did not examine the effects on the brain as such. However, that has been investigated in both humans and animals (see Rutter, 2002a). Both have shown the structural effects of deprivation and of stimulation in the post-infancy years (Elbert et al., 1995; Greenough et al., 1987; Maguire et al., 2000). These are not biological programming

effects as ordinarily understood, but they definitely do represent the effects of experiences on brain structure and function.

Cognitive affective sets, models and self-concepts.

The effect of experiences on cognitive processing and cognitive models or self concepts constitutes a quite different possible mediator of long-term effects. It is well established that, from infancy onwards, individuals process, think about, and conceptualize their experiences. It has been argued that individual differences in how people process their experiences may make a major difference to whether or not there are long-term adverse effects (Main, Kaplan, & Cassidy, 1985). The notion is certainly a plausible one but it has been rigorously investigated only to an extremely limited degree. McCarthy and Maughan (2005) tackled the question by examining the internal working models of attachment and their association with adult love relationships and a sample of women who were known to have had poor parent–child relationships in childhood. The contrast was provided by the fact that the negative child relationships were associated with good adult love relationships in some women and poor adult love relationships in others. What the findings showed is that a secure adult attachment, as assessed on the adult attachment interview, was strongly associated with the difference in the qualities of adult love relationships. The study is limited, however, by the fact that the working models were assessed in adult life and not in adolescence. Accordingly, it is not possible to go beyond the statistical association to say which was causing which, or whether both derived from some other influence. Hughes and her colleagues (Hughes, Turton, Hopper, McGauley, & Fonagy, 2004; Turton, Hughes, Fonagy, & Fainman, 2004) used a different research strategy by focusing on women who had experienced a stillbirth and then following them up to examine outcomes in terms of emotional disturbances. The supposition from attachment theory would seem to be that unresolved attachments should be associated with worse emotional outcomes but, in the event, that is not what was found. Dodge and his colleagues (Dodge, Bates, & Pettit, 1990; Dodge, Pettit, Bates, & Valente, 1995) studied a sample of physically abused children in order to examine whether or not the consequences for later disruptive behaviour were mediated by cognitive processing differences. Their findings showed significant mediating effects but they accounted for quite a small proportion of the overall variance. Negative attributions have been shown to play a role in the course of depression but it is less clear whether they play a role in its initial onset (Teasdale & Barnard, 1993). Despite the prominence of theoretical notions on the important mediating effects of cognitive/affective sets, the

conclusion at the moment must be that they constitute an important potential form of mediation on the effects of experiences, but the phenomenon has yet to be tested systematically on a major scale.

Effects on patterns of interpersonal interaction

The research undertaken up to now on the mediating effects of patterns of interpersonal interaction have almost entirely taken childhood behaviour as the starting point, rather than childhood experiences. Nevertheless, given the importance of experiences on childhood behaviour, the findings are likely to have implications for the mediation of enduring effects on such experiences, although that has still to be subjected to systematic investigation. The strength of the influences was shown first in Robins' (1966) follow-up of antisocial boys and control boys into mid-adult life. The findings showed dramatic differences in adult relationships as reflected in divorce (especially multiple divorce) and in being practically without friends. The same study indicated, too, the major effects on the adult environment indexed in other ways. For example, antisocial boys were much more likely to be unemployed, to have at least ten job changes in ten years, and to be in an unskilled or semi-skilled job at follow-up. More recently Champion, Goodall, and Rutter (1995) showed the strong effects of emotional and behavioural disturbances at age ten and the likelihood of experiencing severely negative acute events and long-term difficulties 18 years later. The effect of conduct problems on later-life stressors was strongest but there were comparable, although weaker, effects for emotional disturbance.

Effects on environmental continuities

The findings just discussed on patterns of interpersonal interaction also reflect the effects on environmental continuities. However, these have been shown in other ways as well. For example, for a long time parental loss was viewed as a major risk factor for depressive disorders in adult life. Harris, Brown, and Bifulco (1986) confirmed this risk effect but went on to show that it was entirely dependent on whether or not parental loss led to a lack of adaptive parenting. The risk effects derived from poor maternal care, the loss being important only because it predisposed to such inadequacies in the rearing environment. It is noteworthy that the effects of poor maternal care were as great in the individuals who did not experience parental loss as in those who had. Loss was important because it predisposed to the proximal risk factor of lack of adequate care, but it was the latter that constituted the immediate risk process. Those findings were concerned with the immediate effects of loss on the rearing environment, but the same set of studies by

Brown and his colleagues also showed the important mediation that came through later experiences of a different kind. For example, teenage pregnancy constituted a further risk mediator nearer in time to the onset of depression (Harris, Brown, & Bifulco, 1990).

Effects on societal and other responses

Lastly, it is necessary to consider the possible mediating effects that derive from societal responses to behaviours that stem (at least in part) from adverse experiences. Thus, for example, beneficial effects were evident in the opening up of opportunities associated with army service for individuals from socially deprived backgrounds—as found by both Elder (1986) and by Sampson and Laub (1996). Of course, it was not that being in the army was in itself a good thing but, rather, that serving in the armed forces provided further educational and career opportunities and widened the peer group to individuals from non-deprived, as well as deprived, backgrounds. Adverse effects have been evident in the effects of imprisonment as a societal response to delinquent or criminal behaviour (Sampson & Laub, 1993). Of course, it may well be the case that the individuals "deserved" punishment for their antisocial behaviour, but the point of the findings is that imprisonment made it more likely that the individuals would have grave difficulty in getting a job and the experience of unemployment made it more likely that the people would return to crime. What has been crucially important in developmental studies of the long-term effects of experiences has been the demonstration of the wide range of possible mediating mechanisms. It is knowledge on such mediators that will be crucial in planning effective interventions, both preventive and therapeutic. A major clinical research need is the study of the mediating mechanisms for efficacy in intervention studies (Weersing & Weisz, 2002).

AGE OF ONSET EFFECTS

For the most part, both the psychological and the psychiatric literature has tended to deal with psychopathology without much reference to possible age of onset differences. Nevertheless, research findings point to the likelihood of important effects associated with age of onset. For example, some of the few genetic studies that have examined the matter have tended to show stronger genetic influences on depression that begins in adolescence or adult life than on depression that begins in childhood (Silberg et al., 1999; Thapar & McGuffin, 1996). The long-term follow-up of the Isle of Wight sample initially studied in childhood and adolescence and now reassessed in their mid-forties has also shown potentially important differences in the correlates of depression according to age of onset

(Maughan, Pickles, Collishaw, Messer, Shearer, & Rutter, 2005). Thus, the experience of maltreatment in childhood was possibly more strongly associated with an onset of depression in adolescence than in adult life. This could have arisen simply because an earlier onset made it more likely that there would be recurrence of depression and a comparison of the two variables suggested that recurrence and age of onset were influential. Even more striking differences applied to the association with conduct symptoms in childhood. This was much more frequently associated with adolescent-onset depression than with adult-onset depression. Because, up to now, there has been so little focus on age differences, we lack understanding of the mechanisms involved. Thus, it is necessary to ask whether the differences derive from a person's age when the depression was first evident or, rather, from the kinds of depression that begin early being different from those that begin later.

Comparable issues arise with respect to antisocial behaviour. Moffitt (1993) made the important distinction between what she called lifecourse-persistent antisocial behaviour and adolescence-limited antisocial behaviour. The two varieties have been shown to differ in important ways and there is no doubt that this differentiation within the overall field of antisocial behaviour has been helpful. Nevertheless, questions need to be asked about the mediating mechanisms. Thus, for example, do the differences that have been found with respect to associated risk factors derive from the degree of persistence of the antisocial behaviour, from the age when it first began, or from the association between early onset and hyperactivity? It has been shown that an early onset of delinquency is associated with a stronger familial loading in both first and second degree relatives (Taylor, Iacono, & McGue, 2000) but the same questions arise with respect to this contrast. Also, does the rate of familiality imply a stronger genetic effect or is it deriving from a more seriously adverse environment that leads to an early onset of delinquency? Once more, the findings clearly point to the need to have this issue on the research agenda but most of the needed investigations have yet to be undertaken. The main emphasis in the clinical research literature has been on the question of whether or not diagnostic criteria need to be modified for different age groups. That is an important question but the developmental evidence indicates that much greater attention needs to be paid to the meaning of differences in the age of onset. Is prepubertal depression the same syndrome as depression beginning in adult life? Is conduct disturbance with an onset in early or middle childhood different from that which begins in adolescence? Research to answer questions such as these in much needed but is only just beginning. General population long-term longitudinal studies are essential.

SEX DIFFERENCES

Up until recently, most researchers and most clinicians have tended to view sex differences as a "given", something that cannot be changed, and therefore not worth much attention. It is now clear that that is a quite mistaken view because, although biological sex is indeed a "given", the risk and protective mechanisms may involve a quite diverse range of mediators spanning hormonal influences, societal expectations, sex-related variations in risk experiences and gene–environment interplay (Rutter, Caspi, & Moffitt, 2003). This means that determination of the mechanism for the sex difference is likely to be relevant for the risk factors within each sex. Because multilevel causal mechanisms operating over time are likely to be important, the study of sex differences has to be part of developmental psychopathology. Most reviews of sex differences have emphasized the relatively small differences between males and females on a wide range of psychological functioning, the huge overlap between the two, and the large individual differences within each sex (Hines, 2004; Maccoby, 1988; Maccoby & Jacklin, 1974). The conclusion has been that the differences within each sex far outweigh the differences between the two sexes. However, while that is indeed the case with respect to many psychological features, the differences with respect to psychopathology are rather greater. Three examples illustrate the point. First, there is the finding of the marked rise in the rate of depression in females in adolescence, a rise that is much more marked than that seen in males. There is very little difference between the two sexes in the rate of depression in childhood but there is quite a marked difference in adult life, the difference arising during the teenage years (Hankin & Abramson, 2001; Hankin et al., 1998). There is some indication that the sex difference reflects the increasing role of genetic influences during this age period, an association with negative life events and experiences, with the interplay between genes and environments being possibly more influential in females than males (Silberg et al., 1999, 2001). Twin study findings emphasize the importance of gene–environment correlations and interactions in relation to depression in adolescent females (Eaves, Silberg, & Erkanli, 2003) but the necessary comparisons between males and females have yet to be undertaken.

Numerous studies have shown the greater frequency of antisocial behaviour in males than females but it is only recently that attention has been drawn to the evidence that this difference varies a good deal according to the type of antisocial behaviour being considered and also the age group being studied (Rutter et al., 1998). In particular, the most marked difference applies to the contrast between lifecourse-persistent antisocial behaviour, which is very much commoner in males, and adolescence-limited behaviour that shows a much small sex difference (Moffitt, Caspi, Rutter, & Silva,

2001). The contrast is particularly noteworthy because it has so often been assumed that the rise in crime in the years of adolescence, and the emergence of more seriously violent behaviour, reflects the consequences of the surge of testosterone that accompanies puberty in males. There is, of course, no doubt that testosterone has behavioural effects but the effects seem to be more on dominance than on violence (Rowe, Maughan, Worthman, Costello, & Angold, 2004) and the finding that the rise in antisocial behaviour in this age period is at least as marked in females as in males runs counter to the hypothesis of the major causal effect of male sex hormones on antisocial activities.

The third feature is the marked male preponderance for almost all neurodevelopmental disorders—such as dyslexia, language delay, autism, and attention-deficit disorder with hyperactivity (Rutter et al., 2003). For a while, some commentators argued that most of the sex difference in dyslexia was an artefact of referral (Shaywitz, Shaywitz, Fletcher, & Escobar, 1990) but it is now clear from multiple large-scale epidemiological studies that that is not the case. Reading disability is about twice as common in males as females (Rutter et al., 2004b). The sex differences in autism and ADHD are even greater. The use of the term neurodevelopmental disorders should not be thought to mean that the sex difference particularly applies to conditions involving brain pathology. Thus, there is not much difference between the two sexes with respect to the rate of either cerebral palsy or epilepsy. Rather, the difference applies to conditions involving neurocognitive impairments of one kind or another that are associated with psychopathology and which are first evident in the preschool years. What is very striking is that there has been almost no systematic research into the reasons for these sex differences. It is notable that the female preponderance is largely confined to emotional disorders arising in adolescence (depression and eating disorders) and the male preponderance to early onset neurodevelopmental disorders. That certainly implies that there may be common influences within these two groups but which differ between the two groups. If the matter is to be studied adequately, it will be essential to take on board an appreciation of the range of possible factors that may be involved in sex differences and the need to consider the differences at several different levels (Rutter et al., 2003).

NORMALITY AND DISORDER

The literature is full of rather fruitless disputes (mainly between psychologists and psychiatrists) on the pros and cons of dimensional versus categorical approaches to concepts of psychological functioning and of psychopathology (Rutter, 2003). All sorts of assumptions have been involved in these battles, many of which are unwarranted.

The evidence on the continuities between normality and psychopathology is of several different kinds and, taken together, the findings are compelling. Thus, as in the rest of medicine (Rutter, 2003), it is clear from longitudinal studies that variations in symptomatology within the normal range are predictive of later clinically significant mental disorders in the case of common multifactorial features such as depression and antisocial behaviour. What has been more surprising is that continuities seem to apply also to severe mental disorders such as schizophrenia and autism that used to be considered as qualitatively quite distinct from normality. The same appears to apply to dyslexia, severe specific language impairment and attention-deficit disorder with hyperactivity (ADHD). The evidence in these cases comes from twin and family research, with the findings from longitudinal studies of high-risk groups particularly important. Thus, family studies have shown that the genetic liability to schizophrenia includes schizotypal and paranoid disorders (Kendler, Neale, & Walsh, 1995), and longitudinal studies of individuals at high risk from a familial loading have shown the progressions from these prodromal features in adolescence to overt psychosis (Johnstone, Ebmeier, Miller, Owens, & Lawrie, 2005). Longitudinal studies of normal populations have similarly shown continuities between common psychotic-like features in childhood and the later development of schizophrenia-spectrum disorders (Cannon et al., 2002; Poulton et al., 2000). In the case of both these common childhood precursors and adolescent prodromata, it is important to note that, although these features are associated with a much increased risk of a schizophrenia spectrum disorder, only a minority (albeit a large minority) actually make that progression.

In the case of autism, the apparent continuity between normality and a handicapping disorder derives from twin studies showing that the genetic liability extends to social and communicative deficits in individuals of normal intelligence (Le Couteur et al., 1996) and to the family-study data showing that somewhat similar features occur in over a fifth of first-degree relatives (Bailey, Palferman, Heavey, & Le Couteur, 1998; Rutter, in press c). The same research strategies have given rise to similar conclusions in the case of specific language impairment (Bishop, North, & Donlan, 1995) and dyslexia (Snowling, Gallagher, & Frith, 2003). Also, twin-study findings with ADHD have shown that the genetic liability extends beyond the diagnostic category to a dimension of overactivity/inattention (Levy & Hay, 2001).

Two questions derive out of this reasonably well-established set of findings. First, should the evidence be interpreted as indicating simply a broadening of the diagnostic category or, rather, does it reflect a true continuity between the psychopathological condition and variations in psychological functioning within the normal range? At present, there is no

definitive answer to that important question and both possibilities need to be considered. Second, what are the mechanisms involved in the transitions between the psychopathological condition and either the broader phenotype or the normal dimension? Is it simply that the handicapping condition represents a more severe set of risk factors or is it necessary to invoke some kind of "two-hit" mechanism in which one set of influences apply to the broader phenotype or dimension and the second hit involves the transition into the handicapping disorder? No adequate answers are available as yet on these questions as applied to any of the three examples given. However, it is obvious that a developmental perspective is essential in order to address the issues.

CONNECTIONS AMONG DIFFERENT PSYCHOLOGICAL DOMAINS

On the whole, psychiatrists have traditionally considered mental disorders as if each constituted a cohesive whole. Thus, research has focused on the possible causal influences on the development of schizophrenia or autism or developmental language disorders, as if these are likely to operate on the disorder as such. Of course, that may be how the causes operate. Nevertheless, a key developmental question concerns the possible connections among the different psychological domains. For example, one of the major breakthroughs in relation to the understanding of autism came from the experimental studies of Hermelin and O'Connor (1970) that showed the importance of cognitive deficits, with the implication that these might underlie the social deficits that defined the disorder. In more recent times, the connections were taken much further through the demonstration of deficits in mentalizing that came to be categorized as an impairment in "theory of mind" (Baron-Cohen, Leslie, & Frith, 1985; Frith, 2003). Several different sorts of questions have arisen from these important findings. Thus, to begin with, the first manifestations of autism arise earlier than theory-of-mind skills become evident. Accordingly, there has been a need to question what are the precursors of theory-of-mind skills and, thereby, to ask questions about the key cognitive process that underlies the social abnormalities (Sigman & Ruskin, 1999). Second, although mentalizing deficits seem to have an understandable connection with social and communicative impairments, it was not so obvious how they could account for the repetitive and stereotyped behaviours that are a crucial part of autism. Attention then shifted to the concept of a lack of central coherence—in other words, the observation that individuals with autism tended to perceive features on the basis of details rather than the gestalt of a meaningful whole (Happé, 1994, 2003). Of course, that raises the further question as to what are the connections between central coherence and

theory of mind. Third, genetic researchers began to ask whether the genetic influences might be specific to these different features (Rutter, in press c). There are some indications that that might be the case but the findings so far are contradictory and inconclusive. Nevertheless, what is clear is that these are important developmental questions to be asking.

Similar issues arise with respect to developmental language disorders. Traditionally, these were viewed as disorders that were confined to language functions. That is indeed what defines them but a range of clinical, epidemiological, and follow-up studies have shown that these disorders (at least those involving receptive and pragmatic abnormalities) also involve a somewhat broader range of cognitive impairments (including theory of mind) and are associated with a surprisingly high frequency of social deficits in adult life (Clegg, Hollis, Mawhood, & Rutter, 2005). The evidence is not yet available to provide an understanding of how all this fits together but the concept of developmental language disorders has had to be modified and extended and new questions have arisen on how the interconnections among the different psychological domains operate over the course of development of these problems.

Somewhat similar questions arise in relation to the quasi-autistic features that have been found in both congenitally blind children (Brown, Hobson, & Lee, 1997; Hobson, Lee, & Brown, 1999) and in children who have been reared in profoundly depriving institutions (Rutter et al., 1999). Clinical pictures in these groups have remarkable similarities with autism as ordinarily diagnosed but, equally, there are some very striking differences—hence the use of the term "quasi-autistic" rather than autistic. Whether or not the findings have any implications for autism spectrum disorders as they arise in children without an institutional background or congenital blindness remains quite uncertain at the moment. However, what is clear is that we need to consider how these features develop and what are the mediating mechanisms.

PSYCHOPATHOLOGICAL PROGRESSIONS

One of the really important changes that has taken place over recent times has been the appreciation that a developmental approach is needed as much for adult psychiatric disorders as for those occurring in childhood. Nowhere is this more evident than in the change of view that has taken place with respect to schizophrenia (Keshavan et al., 2004). The range of studies using diverse research strategies showed the frequency with which schizophrenia was preceded in childhood by neurodevelopmental abnormalities, by attentional problems, and by behavioural abnormalities (Rutter & Garmezy, 1983). The connections over time became much more definitively delineated, however, by the same progressions being shown through large-

scale general population epidemiological cohort studies. A further new twist was added by the evidence from the Dunedin Longitudinal Study that psychotic-like abnormalities could be detected even in childhood (Cannon et al., 2002; Poulton et al., 2000). Accordingly, as noted above, the concept now is of childhood precursors that go on to a prodromal state, which, in turn, then leads to an overt schizophrenic psychosis. One of the key questions, of course, is what are the risk factors involved in this progression that extends over one or more decades? One clue has been provided by the evidence that the risk for schizophrenia is increased through the heavy early use of cannabis (Arseneault et al., 2004; Henquet et al., 2005). It is important to note, however, that the cannabis effect seems to operate largely or entirely on those who are genetically vulnerable (Caspi et al., 2005).

A rather different sort of psychopathological progression is involved in the transition from either ADHD as a disorder, or hyperactivity/inattention as a dimension, to later antisocial behaviour (Rutter et al., 1997). The progression is well documented across a range of different studies but the mechanisms remain somewhat obscure. To an important extent, they probably reflect a shared genetic liability between ADHD and antisocial behaviour (Nadder, Silberg, Rutter, Maes, & Eaves, 2002) but it is dubious whether that constitutes the whole story. Also, the role of hyperactivity as a risk factor needs to be considered alongside the extensive evidence that physical violence in early childhood is also a strong risk factor for later antisocial behaviour (Tremblay, Hartup, & Archer, 2005).

A third progression concerns the finding that prepubertal anxiety tends to lead on to postpubertal depression. This has been evident in both genetic studies and in longitudinal studies (Eaves et al., 2003; Silberg et al., 2001). To a large extent this reflects a shared genetic liability between anxiety and depression, but is that the total explanation?

A fourth example is provided by the route from early conduct disturbance to drug taking to depression (Rutter, 2002b). The same sort of questions arise. In other words, what is the role of shared genetic liability; what is the role of shared risk factors; and in what way might one behaviour constitute a risk factor for another? As with so many of the facets of a developmental perspective considered in this paper, the questions set the research agenda but the answers await further research.

RESILIENCE

The universal feature of all studies of environmental hazards, whether they be physical or psychosocial, and whether they concern humans or other animals, is the huge individual differences in response. That applies to all environmental hazards that have been studied. First, this led to concepts of vulnerability and invulnerability as if these were absolutes. That quickly

became a discredited notion but it was followed by the notion of resilience, with implications that although this was a graded feature (rather than a categorical one), nevertheless it, too, was viewed as a general quality. That, too, is clearly invalid (Rutter, in press d). Not only is it implausible that the same qualities will apply to resilience to infections, cardiovascular disease and psychopathological conditions, but also it has to be questioned as to whether the resilience resides in the individual characteristics or within the situation as more broadly conceptualized. What research with the developmental perspective has brought is evidence indicating that the influences on resilience include pre-stress qualities (in the individual and in the environment); factors operating during the adverse experience; and experiences that occur subsequent to the stress or adversity. In other words, the ''action'' does not reside simply in the chemistry of the moment at which the stress factor operates. Rather, it must be viewed in terms of a process that incorporates both risk and protective factors that influence the way people encounter the experience; features that concern the way they deal with the experience (including the coping mechanisms that they employ); and the experiences that occur later in life that may bring about recuperative turning point effects (Rutter, 1996). In other words, resilience needs to be reconceptualized as a process rather than an individual characteristic. One further point that emerges from the research findings is that individual differences apply even with the most severe adversities. It might be supposed that with gross pervasive deprivation, everyone would be affected to the same degree, but that clearly is not the case. The follow-up of the institutionalized children from Romania showed that the individual differences were as marked in those who had had prolonged deprivation as in those who had had quite short periods (Rutter, in press a). The implication is that the psychopathological consequences of adversity will not be wholly explicable on the basis of the aggregate of risk and protective influences; that research into mediating mechanisms will need also to be informed by evidence on the factors associated with individual differences in response to adversity; and that the investigation of resilience needs to move from a trait-focus to a process-focus (Rutter, in press d).

GENE–ENVIRONMENT INTERPLAY

The last topic to note concerns the huge importance of gene–environment interplay (Moffitt, Caspi, & Rutter, 2005, in press; Rutter, in press b; Rutter, Caspi, & Moffitt, in press; Rutter & Silberg, 2002). At one time, there was a general tendency to wish to subdivide disorders (and traits) into those that were predominantly socially determined and those that were predominantly genetically influenced. At least so far as multifactorial trait disorders are concerned, which is the vast majority of those of interest, that is an entirely

misleading, false dichotomy. Not only is it the case that all traits and all disorders involve both genetic and environmental influences but, much more importantly, these involve gene–environment co-action or interplay. Three main varieties need to be considered. First, there are genetic influences on exposure to risk environments (through gene–environment correlations). Second, there are genetic influences on susceptibility to risk environments (through gene–environment interactions). Third, there are environmental influences on gene expression (through effects on methylation as part of epigenetics). So far as interactions are concerned, these have been most convincingly demonstrated through the use of molecular genetic techniques. Thus, Caspi et al. (2002) showed that antisocial behaviour arose as a function of genetically influenced MAOA activity and a childhood history of maltreatment. There was no main effect of genes, there was a weak main effect of environment, but there was a strong interaction between the two. Similarly, Caspi et al. (2003) showed that the effects of maltreatment in childhood on liability to depression were moderated by the *5-HTT* gene. Most recently, Caspi et al. (2005) have shown that the risk effects for schizophrenia associated with heavy early use of cannabis are moderated by the valine allele of the *COMT* gene. This field of research is still at an early stage but it may be firmly anticipated that evidence on all three forms of gene–environment co-action and interplay will expand greatly over the years ahead (Moffitt, Caspi, & Rutter, 2005, in press; Rutter, Caspi, & Moffitt, in press).

OVERVIEW OF RESEARCH IMPLICATIONS

As illustrated by several of the examples given, there are five main research implications that derive from considerations of these ten facets of a developmental perspective on psychopathology. First, as noted in the introduction, developmental studies need to include both a focus on individual differences and on delineation of the continuities and discontinuities between normality and disorder. Second, there must be a hypothesis-testing approach to the study of causal mechanisms, and not just a documentation of age differences. Third, in order to study developmental mechanisms, long-term prospective longitudinal studies are essential. These need to include both general population epidemiological samples and groups at high risk on the basis of their behavioural characteristics, or adverse experiences, or genetic background. For the study of some kinds of risks, such prospective studies need to begin in the prenatal period, and to include assessment of immunological, neuroendocrine and toxic risk factors. Fourth, it is important that such research involves an integration of social, genetic and developmental concepts and strategies (Rutter & McGuffin, 2004). Fifth, the investigation of causal mechanisms must incorporate a

range of relevant and informative strategies including quantitative and molecular genetics, brain imaging and animal models—to mention but three particularly important examples.

CONCLUSIONS

In conclusion, as illustrated by several of the research examples given, key research strategies that are going to need to be employed in the years ahead span quite a broad range. They include quantitative molecular genetics; functional imaging; longitudinal research over the long term as well as the shorter term; natural experiments (see Rutter, Pickles, Murray, & Eaves, 2001) and internal working models and mental sets. In the past, it has to be accepted that developmental researchers employed too narrow a range of research strategies, failed to take adequate advantage of technological progress, and neglected the need to consider brain–mind relationships. Fortunately, those are largely problems of the past and the field is now moving ahead with increased vigour. Developmental research is a rich field of high potential, it is, and needs to be, process-oriented rather than norm-oriented, it needs to focus on the interconnections between brain and mind, and it needs to have a major interest in individual differences. The examples given all indicate that that is happening to an increasing degree but they also indicate the challenging research agenda that remains and the need to pay attention to the really important questions on mediating mechanisms that arise with respect to all the multiple facets of the developmental perspective on psychopathology that have been considered in this paper.

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