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Developmental psychopathology: Concepts and challenges

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Abstract
The defining features of developmental psychopathology concepts include attention to the understanding of causal processes, appreciation of the role of developmental mechanisms, and consideration of continuities and discontinuities between normality and psychopathology. Accomplishments with respect to these issues are reviewed in relation to attachment disorders, antisocial behavior, autism, depressive disorder, schizophrenia, and intellectual development. Major research challenges remain in relation to measurement issues, comorbidity, gender differences, cognitive processing, nature–nurture interplay, heterotypic continuity, continuities between normal variations and disorders, developmental programming, and therapeutic mechanisms in effective treatments.

Origins of a Developmental Psychopathology Perspective

Karl Popper (1972), the distinguished philosopher of science, made the astute point that definitions need to be read from right to left, and not left to right. In other words, it makes no sense to ask what a word means. Rather, we have to ask what is the concept or idea to which we wish to attach the descriptive term. Sixteen years ago we succinctly referred to developmental psychopathology as the study of the origins and course of individual patterns of behavioral maladaption (Sroufe & Rutter, 1984). We went on to emphasize that the main aim was to provide an understanding of the processes underlying both continuity and change in patterns. The focus was on the *how* of developmental processes, rather than on the charting of age-related progressions. Many, perhaps most, causal processes were viewed in terms of chain reactions operating over time. Development was seen as an active, dynamic process that involved individuals processing (cognitively and affectively) and adding meaning to their experiences, the biology of the individual influencing how individuals respond to their experiences but, in turn, the biology being shaped by those experiences. Because development was likely to be affected by experiences that often occurred for the first time only in adult life (such as marriage, rearing children, and establishing a work career), development was viewed as extending into adult life—a life-span approach. The end product of development was, therefore, conceptualized, not as the attainment of biological maturity or the highest level of competence or the stabilization of personality traits but rather as the establishment of a coherence of functioning as a thinking, feeling human being. In this essay, we look back on the origins of the domain of developmental psychopathology, consider its current stand-

We are greatly indebted to Dante Cicchetti for his invaluable suggestions on an earlier draft of this paper, as well as for his leadership of the field over many years. In addition, however, this account of developmental psychopathology has been possible only as the result of the creative research of many pioneers from both developmental and clinical fields; we wish to express our deep appreciation for their contributions, which have done so much to set an exciting and fruitful research agenda.

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ing, review some of its accomplishments, and look forward to the very considerable challenges that remain.

Positive influences on origins

Many ideas, and many people, played critical roles in the emergence of developmental psychopathology as a key perspective on both developmental processes and the causes and course of psychopathology. We make no attempt to cover these in exhaustive fashion but, rather, provide a few disparate examples to indicate the diversity of the roots of the perspective. In so doing, we have sought to provide a balanced overview of perspectives, rather than expound our own positions, although doubtless our own views will have colored our scientific vision to some extent.

We begin with risk research because it is paradigmatic of developmental psychopathology (Sroufe & Rutter, 1984). At first, particularly in the United States, both the origins and the outcomes of risk were viewed in diagnosis-specific terms (Garmezy, 1974). Thus, longitudinal studies were set up to examine the emergence of schizophrenia in the offspring of mothers with a schizophrenic disorder. It soon became clear, however, that many of the children who did not develop schizophrenia did show other forms of psychopathology, and that the risks for such psychopathology also derived from other forms of mental illness in parents (Rutter, 1989a). Epidemiological studies provided a further extension in their demonstration that many of the risks were found in families exhibiting discord, disruption and disharmony but where the parents had no diagnosable mental illness (Rutter & Quinton, 1984). Studies of all manner of risks, both mild and severe, were consistent in their demonstration of the extreme heterogeneity of outcome and in their indication that it was the number of serious risks, rather than the nature of any one, that was critical (Sameroff, 2000). Given the same risk experience, some children succumb with disorder whereas others seem to escape largely undamaged. Attention, increasingly, came to be drawn to the need to consider both risk and protective mechanisms (Rutter, 1990), to understand the developmental operation of the complex mix of influences that give rise to resilience in the face of adversity (Masten & Curtis, 2000; Rutter, in press-a; Sroufe, 1997), and to realize that individuals cannot be subdivided categorically into the vulnerable and invulnerable. There were many individuals who suffer in some ways but not in others (Luthar, Cicchetti, & Becker, in press). Furthermore, some of the adverse outcomes take the form of serious patterns of social malfunction that do not fit readily into any of the existing psychiatric diagnostic categories (Richters & Cicchetti, 1993; Rutter, in press-c). If risk processes are to be understood adequately, investigations need to include detailed longitudinal studies of risk populations, as well as large-scale epidemiological studies. Hetherington's (1989; Hetherington & Clingempeel, 1992; Hetherington & Stanley–Hagan, 1997) studies of divorce and stepparenthood well illustrate this trend.

Of all the specific features of development, the study of emergence of selective attachments, our second example, was possibly the most crucial in moving thinking in a developmental psychopathology direction. Early studies had made clear both the importance of selective attachments and the phases of early development that were involved in their establishment (Bowlby, 1969). Ainsworth, Blehar, Waters, and Wall's (1978) recognition that not only were universals of development involved but also patterns of attachment relationships differed in the degree of security provided and that these individual differences mattered for later development moved developmental thinking into an individual differences perspective. It was, however, an individual differences perspective that involved a key shift from trait views. The individual difference initially lay in a dyadic relationship, and only later did this become transformed into something more akin to an individual characteristic (Sroufe, 1989). Also, the individuality, as carried forward in time, was thought to derive from a particular way of thinking about relationships, or cognitive set, termed an internal working model (Bretherton, 1995; Bretherton & Waters, 1985). The functions of behavior were crucial. Thus, the

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approach behavior of attachment in the toddler age period was viewed as (and empirically found to be) a precursor of later independence (rather than dependence), if the attachment relationships were secure (Sroufe, 1983; Sroufe, Fox, & Pancake, 1983). A probabilistic "pathways" model of development was a key feature (Rutter, 1989b; Sroufe, Egeland, & Carlson, 1999).

A third area of research that was influential concerned the study of cognitive processing of experiences. Findings in the fields of both antisocial behavior (Dodge, Bates, & Pettit, 1990; Dodge, Pettit, Bates, & Valente, 1995) and depression (Garber, Quiggle, Panak, & Dodge, 1991; Teasdale & Barnard, 1993) showed the importance of individual differences in the attributions that people make to the experiences and personal interactions that they encounter. The importance of this body of research lay in its emphasis on the active thinking and feeling processes that were involved in the interplay between persons and their environments. The findings pointed to the need to move away from a sharp dichotomy between risk factors based on environmental hazards and risk factors involved with personal attributes. A more dynamic appreciation of how the two worked together was required.

The fourth positive stimulus derived from studies of children with handicapping disorders. The findings showed both the commonalities in the course of development shown by normal and handicapped children (Cicchetti & Sroufe, 1978) and also the marked differences associated with certain disorders, such as autism (Hermelin & O'Connor, 1970). Studies of individuals with gross sensory handicaps provided an interesting mixture of the two (O'Connor & Hermelin, 1978). What the research demonstrated was the very considerable extent to which comparisons across groups could be informative about the mechanisms involved in both normal and abnormal development. There was a need to avoid false decisions about whether there were continuities between normal and abnormal or whether the presence of disorder marked a qualitative discontinuity. The research was clear in its indication that both could be found, and it made no sense to assume one or the other. Instead, the research advantage lay in examining what was involved in the continuities and discontinuities between normal and abnormal behavior (Rutter, 1986a).

A further stimulus came from the growth of findings and theories in the domain of lifespan development (Baltes, Staudinger, & Lindenberger, 1999; Hetherington, Lerner, & Perlmutter, 1988; Rutter & Rutter, 1993). The importance of this field of inquiry derived from the important changes in functioning that take place in adult life; the powerful impact of key experiences in adult life; the finding that people varied in the life trajectories they took, and that it was misleading to see development as a single universal progression that varied only in its timing (see Emde & Spicer, 2000); the importance of social context; and the extent to which what had seemed to be universals were much influenced by life experiences. For example, it had been thought that a thinning of the bone structure was an inevitable part of aging but it became apparent that this was very considerably attenuated if the individuals continued to engage in strenuous physical exercise as they grew older (Lane, Bloch, Jones, Marshall, Wood, & Fries, 1986; Lane, Bloch, Wood, & Fries, 1987).

The principles that unite these disparate examples include the concept of development as an active dynamic process in which the meanings attributed to experiences alter their consequences, and the notion of individual pathways that diverge in both their origins and endings (Cicchetti & Rogosch, 1996).

Limitations in traditional theories and concepts

In part, too, developmental psychopathology perspectives arose because of limitations in the traditional developmental and psychiatric concepts as they applied during the 1970s. Progress in psychiatry had been greatly helped by the appreciation of the importance of diagnosis and classification and by the development of standardized approaches to both. On the other hand, there was concern about the unhelpful reification of diagnostic

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categories (Rutter & Shaffer, 1980). Instead of being treated as useful working hypotheses, diagnostic systems were becoming increasingly constraining, with their implication that categories represented some form of real truth. It was not so much that the categories lacked diagnostic validity (there was good evidence for at least partial validity of some of the main diagnoses; Cantwell & Rutter, 1994) but rather that there was too little appreciation of the limitations in what was known. All sorts of rules had been devised to differentiate between disorder and normality without an adequate understanding of the extent to which so many psychopathological concepts were dimensional (Achenbach, in press), with the need to account for variations along the dimensions and not just for the causes of extreme disorders. This was evident, for example, in the fields of disruptive behavior, anxiety, and depression. Much causal research seemed to be based on the expectation that one single main cause for any disorder would be found and that the cause would be diagnosis specific. Too little attention was paid to the possibility that the mechanisms involved in causation might entail dynamic processes operating over time, that indirect chain effects might often be present, and that there might be several different routes to the same outcome. Of course, there was a great deal of value in the psychiatric research perspectives that were being taken, and, in addition, there were many exceptions to these apparent rigidities. Nevertheless, a broadening of perspectives was needed.

Parallel, but different, concerns applied to developmental psychology. To a considerable extent, research seemed to be preoccupied with an undue concern to chart the universals of developmental progression. Three main limitations stood out. First, there was a tendency to regard age as a sufficient explanation for this progression (Wohlwill, 1970, 1973). It came to be appreciated that age constituted an ambiguous measure (Rutter, 1989c). Biological maturity was not a single thing; hormonal maturity and brain maturity, for example, did not proceed directly in parallel. Also, necessarily, age indexed the nature and extent of experiences as well as the years of biological growth.

Second, very little attention was being paid to the transition into adult life. This is a curious lack because, if development is seen as that which leads up to adulthood, its understanding necessarily required a study of adult outcomes (see Cicchetti & Toth, 1995). One might suppose that what would characterize developmental psychology most of all would be a focus on longitudinal studies that spanned the period from infancy to adult life. In fact, the opposite was more the case (De Ribaupierre, 1989). Most developmentalists specialized in a very narrow age period, and most research was cross-sectional rather than longitudinal.

Third, little attention was paid to the causal processes involved in individual differences in development. There were many exceptions to these limitations, and there was much that was good in developmental research. Nevertheless, the perspectives needed to be broadened. Achenbach's (1974) textbook, the first labeled "developmental psychopathology," was pioneering in that concern.

Fourth, it had become increasingly obvious that the prevailing developmental theories were inadequate in a whole variety of ways. To begin with, most focused on the universals of a progression through predetermined stages, whether defined in terms of psychosexual stages (Freud, 1953), cognitive phases (Gelman & Baillargeon, 1983; Piaget, 1970), or social tasks and crises (Erikson, 1950), with individual differences seen in terms of a stage fixation or regression or a phase adaptation.

There were three main types of problem. First, most of the theories seemed to portray an all-encompassing view of life, rather than an attempt to provide an explanation for particular empirical phenomena. On the face of it, there seemed to be an untenable assumption that a single theory could account for all the complexities that were involved in development. Second, theories failed to address a host of crucial developmental issues. Little attention was paid to biological processes or genetic influences, and gender differences were inadequately conceptualized. Third, all the

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theories had a poor empirical basis, and research findings showed the many ways in which the theories were seriously wrong on numerous essentials (Bowlby, 1988; Bryant, 1974; Piaget, 1970; Rutter, 1971a).

Of course, these big theories had been important historically in ways that have stood the test of time. Thus, psychoanalytic theory was very important in drawing attention to the role of mental mechanisms and of emotional influences, Piagetian theory to the role of active cognitive processes, and Eriksonian theory to the importance of social context and social challenges. Nevertheless, they were ceasing to serve a useful role, and it was necessary to move on. Although it was clear that good developmental research required a hypothesis-testing approach, it was equally apparent that it was completely implausible that any one theory could account for all developmental phenomena. Bowlby's (1969) judicious integration of perspectives in his formulation of attachment theory provided a useful alternative model.

The Current Standing of Developmental Psychopathology

Developmental psychopathology perspectives have become mainstream now, in both developmental psychology and psychiatry, although, quite properly, they do not constitute the whole of either. Thus, with respect to psychiatry, a major body of research is concerned with the evaluation of methods of treatment and of the delivery of services. Similarly, and equally appropriately, developmental psychology is concerned with studying what is involved in developmental processes as they apply universally. Both of these are of interest to developmental psychopathology, but they do not constitute a key feature.

It is also relevant that the perspective represents an equally mainstream, modern, medical model. Thus, for example, with respect to cardiovascular disease, it has long been recognized that there is multifactorial causation, involving influences as diverse as cholesterol levels, clotting factors in the blood, smoking, lack of exercise, obesity, and sex hormones. Many of the risk factors, such as cholesterol levels, operate on a dimensional basis with variations within the normal range being associated with differences in risk. The causal processes operate over a long period of time, beginning early in life. Thus, for example, it has been found that infants of low birth weight have a substantially increased risk of cardiovascular disease in later life (Barker, 1998; Nathanielsz, 1999). The finding is of interest because that is the opposite of the way in which weight operates in middle age (when the risks are associated with being overweight and not underweight). The notion of developmental programming (Bock & Whelan, 1991) has come to the fore, whereby it is thought that the body-regulating systems are adapted in early life to low levels of nutrition and that the risk comes from being, therefore, maladapted to high levels of nutrition in later life. The importance of social factors in the genesis of somatic disease (including cardiovascular conditions) has been well established for a long time. McKeown (1976), in the 1950s and 1960s, pointed out that the huge improvements in infantile mortality and the increase in life expectancy in the first half of this century were largely attributable to improved living conditions and improved nutrition, with medical advances being of limited importance. The huge social disparities in levels of illness are well documented (Acheson, 1998; Townsend & Davidson, 1992; Wilkinson, 1996), and psychological factors too have a well-demonstrated role (Marmot & Wilkinson, 1999). Medicine has also long appreciated that a single-risk factor can lead to quite diverse disorders (smoking is an obvious example of this kind) and that the same disease end point may come from rather varied causal pathways, as the study of comorbidity well illustrates (Rutter, 1997a). All of this closely parallels the perspective of developmental psychopathology. The perspective is a biological one that encompasses medicine and behavior equally. The importance of emphasizing this is that, in the past, there has been a tendency for behavioral scientists to wish to develop a special set of mechanisms and of concepts that apply specifically to psychological development, as if this were somehow separate or different from the rest of bodily

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functioning. It is not. It is true, of course, that the study of cognitive and affective processes is distinctive of psychology and psychiatry (and vital in developmental psychopathology perspectives), but this is part of biology and medicine and not separate from it.

Central Concepts of Developmental Psychopathology

Although there are many features of developmental psychopathology that could be considered important, we suggest that the defining features can be reduced to the approach taken to three key issues: (a) the understanding of causal processes, (b) the concept of development, and (c) continuities and discontinuities between normality and pathology.

Causal processes

The rising influence of behavior genetics in recent years, supported by empirical research findings (Rutter, Silberg, O'Connor, & Simonoff, 1999a, 1999b), has led many psychologists and psychiatrists to think increasingly that the study of causal processes can be reduced to the partitioning of population variance into additive and nonadditive genetic, and shared and nonshared environmental, effects. The findings have, undoubtedly, been important in showing that genetic factors play a major role in individual differences in all kinds of behavior. Among other things, the genetic findings have been crucially important in showing that genetic influences operate with respect to normal variations in behavior as well as to diseases and disorders.

Some developmentalists (Baumrind, 1993) and some neuroscientists (Rose, 1995) have worried that this implies a genetic determinism. We do not share that concern. One of the most important messages of genetic research has been that genetic influences are probabilistic and not deterministic, and that environmental factors and genetic factors are broadly speaking of roughly equal importance (Plomin & Rutter, 1998; Rutter & Plomin, 1997).

We do, however, have three rather different concerns that are central to a developmental psychopathology perspective. First, an understanding of causal processes requires knowledge about how risk and protective mechanisms operate. With rare exceptions, all behavior is influenced by both genetic and environmental factors, and knowing the proportion of the population variance accounted for by each is, on its own, uninformative about how the causal process operates. The need to find out how causal processes operate has important design implications for research. Reviews have considered those relevant to the study of environmental risks (Rutter, Pickles, Murray, & Eaves, 2000), and those that apply to genetic research (Rutter et al., 1999a, 1999b; Rutter, in press-a). As already noted, it will usually be necessary to study the operation of causal processes as they operate over time through direct and indirect chain effects, and a developmental perspective also requires questioning how effects are carried forward and how they influence the way the individual deals with later situations and circumstances.

Second, behavior–genetic partitioning of population variance is entirely concerned with the study of individual differences. That is only one of several types of causal questions. For example, both theoretical and public health considerations require attention to the causes of differences in level of a trait or a disorder. Thus, for example, why has there been such a massive rise in the rate of so many psychosocial disorders in young people over the last 50 years (Rutter & Smith, 1995)? Or, why has there been a rise in the overall IQ level over this century (Anderson, 1982; Flynn, 1987)? Or, why are most forms of antisocial behavior so much more prevalent in males than females (Moffitt, Caspi, Rutter, & Sylva, 2000), and why are depressive disorders (Bebbington, 1996) and eating disorders (Lucas, Beard, O'Fallon, & Kurland, 1991; Nielsen, 1990) more characteristic of women than men?

It is important to appreciate that the causal processes involved in differences in level may not be the same as those involved in relation to individual differences (see Tizard, 1975, with respect to the same point in relation to the huge secular increase in average height during the first half of this century). A differ-

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ent type of causal question concerns *course*. Many disorders involve several distinct phases. Thus, for example, in order for persons to develop drug dependency, they must first take the drug, they must progress to taking it regularly, and they must then develop a psychological or pharmacological dependency on the substance. Factors that influence each of these phases may be rather different (Robins, Davis, & Wish, 1977), as well illustrated by Robins's (1993) study of heroin dependency in Vietnam veterans. Also, the factors associated with the onset of the first episode of a depressive disorder may not be the same as those associated with its repeated recurrence (Post, 1992).

A further type of causal question concerns the factors involved in the translation of an individual liability into the commission of a particular behavioral act such as attempting suicide or undertaking a burglary. The predisposing influences are often rather different from those concerned with individual differences in liability, and they involve factors within the individual, such as alcohol intoxication, or situational factors, such as the availability of the means of the suicide or opportunities for crime (see Rutter, Maughan, Meyer, Pickles, Silberg, Simonoff, & Taylor, 1997, for a discussion of these issues with respect to antisocial behavior).

The third concern about the need to move beyond partitioning the population variance in the study of causal processes is that nature and nurture are not neatly separable in the way that was once envisaged (Bronfenbrenner & Ceci, 1994; Rutter, Dunn, Plomin, Simonoff, Pickles, Maughan, Ormel, Meyer, & Eaves, 1997; Rutter 1997b). Behavior geneticists have produced an increasing body of evidence on the importance of different types of gene–environment correlations and also gene–environment interactions. In the traditional way of tackling partitioning the variance, these will all be included under genetics, although, by definition, they have their effects through interplay with the environment.

In recent years, most of the discussion in the literature has concerned gene–environment correlations, but it is crucial to appreciate the importance of personal effects on the environment that are not genetically mediated. Bell (1968) first pointed out, in a seminal paper, the importance of recognizing the need to determine the direction of socialization effects. That there are important effects of children on their environments has been shown through a variety of research designs (Bell & Chapman, 1986); O'Connor, Deater–Deckard, Fulker, Rutter, and Plomin (1998) showed that, with respect to antisocial behavior, most of the children's effect on their parents was *not* a function of genetic risk. Longitudinal research designs are ordinarily necessary for differentiating between personal effects on the environment and environmental effects on the person; as already noted, they constitute a key feature of a developmental psychopathology research perspective (Sroufe, in press).

Person–environment interplay is characterized by complex chains, sometimes involving heterotypic continuity. For example, avoidant attachment in infancy is associated with later conduct problems, but avoidant attachment is also associated with low self-esteem (Sroufe, 1983), negative attributional biases (Suess, Grossman, & Sroufe, 1992), and rejection by peers and teachers (Sroufe & Fleeson, 1988), all of which may predispose to conduct problems. We are only beginning to understand how these chain effects operate in predisposition to psychopathology. Their understanding provides an unavoidable tension between the need to seek simplification (the hallmark of all good science) and the need to note complexity. Research has often suffered from unwarranted simplifying assumptions, but a true understanding of biological processes usually shows that the complexity is explicable in terms of a limited set of unifying mechanisms and principles.

The role of development

In 1984 (Sroufe & Rutter, 1984), we argued that it was possible to understand the complex links between early adaptation and later disorder only through detailed appreciation of the nature of the developmental process itself. This entails a progressive transformation and

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reorganization of behavior as the developing organism continually transacts with the environment. We stressed that development necessarily involved a change (because that is what development is all about) but, also, that it involved a degree of continuity and the establishment of some coherence. It could be argued that the introduction of the notion of development does not add anything to the study of behavior because development, like behavior, is subject to the same mix of genetic and environmental influences that have to be investigated and delineated. However, although that is the case, and although it is difficult to define the differences between development and other forms of behavioral change (Harris, 1957; Rutter, 1984), it misses the point for three main reasons (Gottlieb, Wahlsten, & Lickliter, 1998; Wahlsten & Gottlieb, 1997).

First, the overall, universally applicable pattern of biological development is determined by nonsegregating genes possessed by all humans. For example, over the course of evolution, all humans have come to have genes that provide the possibility of language (these genes not being possessed, e.g., by mice). Many of the universals of development are of this kind (see, e.g., Kagan, 1981). These universals (the occurrence of which is not influenced by either genes or experiences that differ among children) are important because they may have important implications for adaptation and maladaptation. Thus, there are well-documented age-dependent differences in vulnerability to particular kinds of experiences, and there are age-dependent variations in the incidence of disorders (Petersen, 1993; Rutter, 1986a; Sroufe, in press). Of course, it cannot be assumed that these variations are a consequence of biological maturation, but that does constitute one possibility requiring investigation. Moreover, with respect to certain well-established variations—for example, the response to brain injury (see Vharga–Khadem, Isaacs, van der Werf, Robb, & Wilson, 1992)—biological maturation almost certainly is responsible.

The second consideration with respect to development is that, even when considering individual differences, it is misleading to see the influences as restricted to the genetic and the environmental. The genetic programming of development is probabilistic rather than determinative (Michel & Moore, 1995). Thus, neuronal migration is a key feature of early brain development, but the genetic programming provides a general instruction as to where neurons are meant to go, rather than a control of what happens with each and every nerve cell (Goodman, 1994). It is because of this probabilistic feature that one important element in brain development concerns the selective pruning of neurons that have not ended up in the right place. That means that there is stochastic, or random, variation in development—what Molenaar, Boomsma, and Dolan (1993) have called "the third force," meaning beyond genetic and environmental influences. For example, each girl inherits two X chromosomes, one from each parent, but only one of these remains functionally active. One is inactivated, or "switched off," and it seems that which one it is is randomly determined, as is the case with various other biological processes (see Goodman, 1991).

The third consideration is that the study of causal processes has to be concerned with the carryforward of effects, whether they be genetic or environmental. In each case, it is important to ask about the nature of internal changes within the individual that are taking place (Rutter, 2000). Thus, with respect to psychosocial experiences, these may involve variations in brain structure (Cicchetti & Tucker, 1994; Liu, Diorio, Tannenbaum, Caldji, Francis, Freedman, Sharma, Pearson, Plotsky, & Meaney, 1997; O'Brien, 1997), or alterations in neuroendocrine functioning (Hennessey & Levine, 1979), or modifications of affect regulation (Sroufe, 1996), or changes in cognitive set or cognitive processing (Dodge et al., 1990, 1995), or altered patterns of interpersonal interaction (Pawlby, Mills, & Quinton, 1997; Pawlby, Mills, Taylor, & Quinton, 1997; Quinton & Rutter, 1988).

The continuities and discontinuities between normality and psychopathology

The central concern for developmental psychopathology is delineation of what is in-

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volved in the continuities and discontinuities between normality and pathology. Two points are crucial in this connection. First, the key issue concerns continuities and discontinuities in mechanisms, and not just in measures. Second, even with the same feature, it is often the case that both continuities and discontinuities are present. For example, IQ functions dimensionally as a predictor of educational attainment or social competence right across the range, extending from profound retardation to above-normal levels of functioning. On the other hand, for the most part the causes of severe and profound retardation are quite different from the causes of the variations in IQ within the normal range or, even, of mild mental retardation (Cantwell & Rutter, 1994; Rutter, Simonoff, & Plomin, 1996). Similar issues apply to many other forms of behavior. For example, the phenomenon of depression clearly spans normality and disorder in a dimensional way, but it may also be the case that bipolar affective psychoses are discontinuous with normal variations in mood (Perris, 1992).

A further point is that, regardless of whether or not the underlying liability to psychopathology is dimensional, with a continuum spanning normality and disorder, categorical decisions will often be required for practical purposes. Thus, clinicians need to decide whether or not to use antidepressant medication or to admit somebody to hospital. It is important to distinguish between these practical requirements for having diagnostic, or severity, categories and an understanding of the underlying patterning of behavior.

Past Value of Developmental Psychopathology

Because developmental psychopathology, as conceptualized by us, has been such a burgeoning field in the last 2 decades, it would require a textbook to discuss the many advances that have derived from adopting a developmental psychopathology perspective. In this section of the paper, we therefore provide only a limited set of examples to illustrate something of the range of accomplishments. Some of the examples that we give derive from research by scientists who would not identify themselves as using developmental psychopathology (that would perhaps particularly apply to research in the field of adult psychiatry). As noted in the introduction to this paper, one of the marks of the achievement of a developmental psychopathology perspective is that it has become mainstream and is no longer easily separated off as a different approach. Our aim, however, is to show something of the value of a developmental psychopathology perspective, rather than the achievements of individuals who would identify with this approach. We have chosen to structure this section of the paper according to different types of psychopathology and, in looking forward into the next millennium, we structure the challenges in terms of issues and mechanisms, rather than disorders. Nevertheless, in looking back into the past, we do draw attention to some of the outstanding research needs with respect to the disorders that we consider.

Attachment disorders

Attachment concepts have come to have an increasing prominence in developmental research in recent years. Six main trends may be identified. First, many studies have shown that early insecure attachment relationships are associated with a higher risk of later psychopathology (Belsky & Nezworski, 1988; Cassidy & Shaver, 1999; Sroufe, Egeland, & Carlson, 1999). Increasingly, clinical researchers have sought to apply attachment concepts to an understanding of the genesis of different forms of psychopathology in childhood and adolescence and to the development of methods of treating them. The findings emphasize the importance of social relationships in human functioning and the extent to which they are affected in a wide range of disorders (Hinde, 1997). Nevertheless, there is a danger that attachment concepts become too all explanatory. Attachment features do not constitute the whole of relationships (indeed, one of Bowlby's, 1969, key contributions, was the differentiation of attachment features from other aspects of relationships), and insecure attachments are not themselves indicative of

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psychopathology (Sroufe, 1988; Rutter, 1995). Research must meet the challenge of determining just how insecure attachment relationships play a role in psychopathology, putting competing explanations under research scrutiny and not just assuming the primacy of an attachment paradigm (see Rutter, 2000b, and Rutter, Pickles, et al., 2000, for a discussion of the broader issue of testing causal hypotheses).

Second, there has been a growing attention to the social development of children reared in grossly abnormal environments. This necessitated several developments. To begin with, it soon became apparent that the traditional ways of classifying security and insecurity were not always picking up the features that seemed most distinctive of these children reared in highly atypical circumstances (Vondra & Barnett, 1999). Much earlier, Main and Solomon (1986) had recognized the need for a category of "disorganized attachment" (see review by van IJzendoorn, Schuengel, & Bakermans–Kranenburg, 1999, on its qualities), and this seemed to apply particularly to children from grossly abnormal backgrounds. In addition, because many of the children being studied were substantially older than the age period below 2 years for which the "Strange Situation" had been developed, some modifications of procedure were required (Waters, Vaughan, Posada, & Kondo–Ikemura, 1995). They were also necessary because the scattered nature of unusual samples often meant that the children needed to be assessed in their own homes rather than in the research laboratory. In addition, studies of children reared in severely depriving Romanian orphanages, and later adopted into North American (Chisholm, 1998) or UK families (O'Connor, Bredenkamp, Rutter, & the English and Romanian Adoptees [ERA] Study Team, 1999; O'Connor, Rutter, & the ERA Study Team, in press), showed a pattern of somewhat indiscriminate friendliness to adults that was not well picked up by the presses of the "Strange Situation" procedure when it was used with postinfancy age groups (Goldberg, 1997; Marvin & O'Connor, 1999), although noted by Bowlby (1973) as indicative of attachment problems. Both interview and other forms of observational methods were needed.

Earlier research had shown that the old-style notion of fixed critical periods for development were misleading, but new findings of various kinds have highlighted the importance of age-defined periods of especial vulnerability ("sensitive periods") to particular kinds of stress or adversity. Thus, the follow-up study of children placed in residential nurseries in infancy, undertaken by Hodges and Tizard (1989a, 1989b), showed that antisocial behavior was more a function of current social circumstances and adolescence than the situation in very early childhood. By contrast, both the children who had been restored to the biological parents (and often were living in discordant, disadvantageous circumstances) and those who had been adopted (and who were mainly in above-average quality homes) differed from controls in showing a pattern of peer relationships that lacked intensity and selectivity. The features were not normal, but they did not constitute a disorder in the usual psychiatric sense. Like the findings of indiscriminate friendliness, the links with earlier attachment relationships remain uncertain, but it seems very likely that, in some way, the lack of opportunity to form selective attachments in early childhood predisposed to these later features in peer relationships and adult relationships. Findings of this kind highlight the need to understand how early selective attachments are related to other social relationships in later life.

A fourth important trend in the field of attachment concerns the extrapolation of attachment concepts into adult life (Hazan & Shaver, 1994). Various measures of different aspects of adult relationships have been developed. The adult attachment interview has focused especially on the cognitive set of relationships that is thought to have been derived from the individual's relationships in early life (Main, Kaplan, & Cassidy, 1985; van IJzendoorn, 1995). From all points of view, it makes sense to extend attachment concepts into adult life. After all, the loss of a love relationship constitutes a major risk factor at all age periods after infancy, and there is a universal wish to seek security in close intimate

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human relationships. On the other hand, much work has still to be done sorting out the continuities and discontinuities across the life span. The early childhood measures are dyadic, whereas the measures in adult life reflect individual characteristics. Also, in order to extend into older age, it will be necessary to alter concepts in fairly substantial ways. In the years ahead, it will be important to put to rigorous test the various assumptions that underlie the extrapolations across ages and across different types of relationships.

A fifth trend has been the study of intergenerational continuities and discontinuities (see Fonagy, Steele, Steele, Higgitt, and Target, 1994). Strikingly, several studies have found that the security of the mother's adult attachments, as assessed by the Adult Attachment Interview during her pregnancy, predicted the security of the infant's attachment to its mother. Similar, the mothers' prenatal representation of their infant has also predicted the infants' attachment security (Benoit, Parker & Zeanah, 1997). Questions remain on the mediation mechanisms involved and on the factors leading to differences in attachment security among children being reared in the same family.

Over recent years, too, there has been the application of attachment concepts to all sorts of abnormal groups, but all too often the measures used both depart considerably from standard attachment measures and also lack proven validity (Rutter & O'Connor, 1999). The potential is rich, but there is a great need to bring together the kind of approaches used to study normal development and those devised to study disorder. In that connection, uncertainty continues over the precise meaning and boundaries of so-called attachment disorders (Zeanah, 1996). The pattern of indiscriminate friendliness provides a close approximation to the prevailing concepts of one sort of reactive attachment disorder (disinhibited type), and it seems probable that that will stand the test of time, although questions are still being posed as to how this relates to early selective attachments (Zeanah, in press). Much less work has been done with the other form of attachment disorder (inhibited type), and that remains an important task to be undertaken. Traditionally, the measurements of security of attachment have been categorical, although it would seem conceptually that there are underlying dimensions. Well-validated dimensional measures are needed, and are being developed, in order to examine continuities and discontinuities across the span of behavioral variation (Waters et al., 1995).

Antisocial behavior

It has been appreciated for a long time that antisocial behavior is heterogeneous, but until recently the ways of subdividing it had proved rather unsatisfactory. A way forward was provided by the recognition by Patterson (1995; Patterson & Yoerger, 1997), Moffitt (1993), and others that the age of onset, together with course, might prove to be a key differentiating feature. A distinction was drawn between life-course-persistent antisocial behavior and adolescence-limited antisocial behavior. Empirical research has broadly confirmed the validity of this distinction (Lahey, Waldman, & McBurnett, 1999; Rutter, Giller, & Hagell, 1998). The life-course-persistent variety, which is much more common in males (Moffitt et al., 2000), is characterized by the first manifestations of markedly disruptive behavior in the preschool years, by an association with hyperactivity–inattention, by poor peer relations, by difficult behavioral features (often involving aggressivity, impulsiveness, and sensation seeking), and by mild cognitive impairment. Family discord and disruption stand out as part of the mix of risk features, and insecure attachment relationships are often present (Aguilar, Sroufe, Egeland, & Carlson, 2000). By contrast, the adolescence-onset variety is much less characterized by these risk features (although they may be present in mild degree), but poor parental supervision and being part of an antisocial peer group stand out as the main risk features. Although males predominate to a slight extent, the sex ratio is much near parity than is the case with the life-course-persistent variety. Research evidence, so far, is inconclusive on whether or not the two groups differ qualitatively or, rather, quantitatively; and it is likely that there are more than just two types (Nagin, Farring-

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ton, & Moffitt, 1995). Nevertheless, the distinction according to age of onset and developmental course has proved useful and is likely to hold up as a key differentiator.

Because delinquency reaches its peak in adolescence, there has often been an assumption that aggression and disruptive behavior start at a low level and then increase over time in vulnerable groups. The implication has been that the search needs to be for factors that foster antisocial behaviors. Longitudinal studies undertaken by Tremblay and his colleagues (Nagin & Tremblay, 1999; Tremblay, Japel, Pérusse, Boivin, Zoccolillo, Montplaisir, & McDuff, in press; see also Loeber & Hay, 1997) have called that assumption into question. Their evidence suggests that the peak occurs during the preschool years and falls off thereafter. If confirmed by other studies, the implication is that the search needs to be for factors that fail to inhibit aggressive–disruptive behavior, as well as for factors that foster it.

Traditionally, psychiatric classifications have differentiated between oppositional–defiant disorders and conduct disorders, the former being characterized by disruptive interpersonal interactions and the latter by more severe predatory or illegal acquisitive behavior. A range of longitudinal studies have suggested that this categorical distinction is probably misleading. Oppositional–defiant behavior is more characteristic of the earlier years of childhood, but if it continues it tends to lead into conduct disorder (Lahey et al., 1999). In other words, the slightly different manifestations probably reflect what is characteristic of different age periods rather than of different disorders.

Longitudinal studies have shown that hyperactive–inattentive behavior is a risk factor for later antisocial behavior, particularly for that which persists into adult life (Farrington, Loeber, & van Kammen, 1990). On the other hand, psychiatric diagnostic conventions have tended to place attention-deficit disorder with hyperactivity as a strongly constitutional, and in some respects "organic," condition that is rather different from the broad run of antisocial behavior. Findings have to some extent confirmed this concept (Taylor, 1999), insofar as there is good evidence of a much stronger genetic component in the liability for hyperactive behavior than is the case for other forms of antisocial behavior (Rutter et al., 1998; Thapar, Harold, & McGuffin, 1998), and in showing the importance of cognitive deficits, and of a pattern of correlates that differ from that evident for other forms of disruptive behavior (Taylor, Sandberg, Thorley, & Giles, 1991; Taylor, 1994). Longitudinal studies, particularly those using latent variable analyses, have also shown that the developmental paths of hyperactivity and of antisocial behavior are somewhat different (Ferguson & Horwood, 1993). It seems that hyperactivity is a marker for early disruptive behavior but that the *continuation* of antisocial behavior is dependent on these early associations, rather than on any risk for later onset antisocial behavior. Findings are much in line with those already considered with respect to life-course-persistent and adolescence-limited antisocial behavior. On the other hand, concerns have been expressed that the mere presence of hyperactivity–inattention is too readily used as an indicator of constitutional origin, and that the likelihood is that the phenomenon is more heterogeneous than the diagnostic category suggests (Carlson, Jacobvitz, & Sroufe, 1995; Sroufe, 1997). Recent research on children reared in institutions supports this concern. Studies of both children in well-run residential nurseries in the United Kingdom (Roy, Rutter, & Pickles, 2000a, 2000b), and of children from very poor quality Romanian orphanages (Kreppner, O'Connor, Rutter, & the ERA Study Team, 2000), have both shown strong associations with hyperactivity–inattention. Of all the forms of unusual behavior, this stands out as the one that is the more distinctive. Whether or not the particular characteristics of the hyperactivity–inattention are the same as those found with the diagnostic category in children reared in more ordinary circumstances remains quite uncertain. The genetic evidence is also important in indicating that the genetic liability applies to a much broader range of hyperactivity–inattention than that which is included in the extreme diagnostic category (Rutter, Maughan, et al., 1997; Thapar et al., 1998).

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Research in the field of antisocial behavior has also been important in throwing new light on the role of early experiences. When reviewed some years ago (Rutter, 1981), it was concluded that it was unusual for even markedly adverse early experiences to have enduring effects that were independent of later circumstances. That finding still holds true (although there are some important exceptions, as research into the adoptees from Romania has shown, for at least some outcomes; O'Connor et al., in press; Rutter, O'Connor, Beckett, Castle, Croft, Groothues, Kreppner, & the ERA Study Team, in press). However, the question had been posed the wrong way. What has been found to be important is the considerable extent to which early experiences shape and influence later experiences (Rutter, Champion, Quinton, Maughan, & Pickles, 1995). Indeed, it is rather unusual for later experiences to be independent of earlier ones. Although the direct effects of early experiences on functioning many years later are quite small, the indirect effects are very much greater (Sroufe et al., 1999). These continuities and experiences play a substantial role in enhancing behavioral characteristics, either in an adaptive or maladaptive direction (Caspi & Moffitt, 1991, 1993, 1995).

On the other hand, research has also shown the major importance of experiences in adolescence and adult life, provided that they bring about either a radical alteration in life opportunities or a substantial alteration in a person's self-concept or cognitive set. This has been shown, for example, with respect to army experiences when they bring about discontinuities from the past for disadvantaged youths (Elder, 1986; Sampson & Laub, 1996) and for a cohesive, harmonious marriage (Zoccolillo, Pickles, Quinton, & Rutter, 1992; Quinton, Pickles, Maughan, & Rutter, 1993; Laub, Nagin, & Sampson, 1998). Depending on whether or not the experiences in later life show continuities or discontinuities with early experiences, the consequence may be either an accentuation (Caspi & Moffitt, 1993) or a turning-point effect (Rutter, 1996).

Magnusson's Swedish longitudinal study (Stattin & Magnusson, 1990) showed that an unusually early menarche was associated with an increase in disruptive behavior and heavy drinking. The Dunedin longitudinal study in New Zealand showed much the same but added to the finding in an important way through its demonstration that, although the stimulus was clearly biological, the mediating mechanism was almost certainly social. The effect of early puberty in girls was found to be dependent on the girls' entering an older peer group (as also found by Stattin & Magnusson, 1990) and that the effect was confined to coeducational schools, where, presumably, the presence of males increased the likelihood of an antisocial ethos (Caspi, Lyman, Moffitt, & Silva, 1993; Caspi & Moffitt, 1991).

The importance of interplay between nature and nurture has also been brought out in genetic studies of antisocial behavior (Rutter, 1997b). Gene–environment correlations are frequent and gene-environment interactions are also evident (Bohman, 1996; Cadoret, Yates, Troughton, Woodworth, & Stewart, 1995; Cadoret, 1985; Rutter et al., 1998). Individuals showing antisocial behavior interact with other people in ways that frequently bring about further stressful or risky circumstances. This is strikingly evident, for example, in the strong tendency for antisocial individuals to marry or cohabit with other antisocial individuals (Krueger, Moffitt, Caspi, & Bleske, 1998). Indeed, this assortative mating tendency is actually quite a lot stronger than the tendency for antisocial parents to have antisocial children (Farrington, Barnes, & Lambert, 1996).

Autism

At first sight, it might seem that autism is the diagnostic category least likely to require a developmental psychopathology perspective. As traditionally diagnosed, it stands out as a severely handicapping disorder that appears qualitatively quite distinct from normality. Nevertheless, there are two ways in which this view has proved, at least partially, mistaken. Psychiatric writings in the 1960s tended to consider autism as a variety of infantile psychosis, with parallels being drawn with schizophrenia. Much research indicated that the differences between autism and schizophrenia

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far outweighed the similarities and that also it was probably highly misleading to consider autism as a psychosis that involved some break with a previous normality (Rutter, 1972). Research by Hermelin and O'Connor (1970) and their colleagues was the first to show the value of an experimental psychological approach that sought to examine the cognitive features in greater detail and to link them with the social deficits. Nevertheless, there were considerable difficulties in knowing just how the cognitive deficits might lead to the particular social features that are diagnostic of autism (Rutter, 1983).

A major step forward was provided by the application of "theory of mind" concepts, originally derived from studies of normal children (Baron–Cohen, Leslie, & Frith, 1985). Subsequent research has confirmed that most autistic individuals lack the mentalizing skills to know what another person is likely to be thinking, given knowledge of the social context or past happenings (Frith, 1989; Baron–Cohen, 1995; Happé, 1994). The detailed findings provide good grounds for the inference that this cognitive deficit plays a major role in autistic individuals' severe limitations in the development of reciprocal relationships. One difficulty, however, has been the fact that autism is manifest before age 2 years in most instances, well before normal children show theory of mind skills when tested in the usual way. In part, this is probably a consequence of the fact that the particular experimental situation used does not pick up theory of mind skills that are demonstrable in more naturalistic circumstances (Dunn, 1988, 1996). In addition, the focus shifted to earlier emerging cognitive skills, such as joint attention, that might constitute precursors of theory of mind or an alternative cognitive route to the social dysfunction (Sigman & Ruskin, 1999). Although theory of mind deficits constitute a plausible explanation for social dysfunction, it is not so obvious that they could account for the repetitive stereotyped behaviors that are also characteristic of autism. For that and other reasons, research has sought to determine whether a lack of central coherence (Happé, 1996) or deficits in particular aspects of executive planning (Pennington & Ozonoff, 1996) might also provide part of the cognitive basis for autism. Doubts have been raised about both (Griffith, Pennington, Wehner, & Rogers, 1999; Plaisted, Swettenham, & Rees, 1999) but they warrant further study.

A second area of autism research where a developmental psychopathology perspective has been useful concerns the delineation of the diagnostic boundaries. Genetic research has been important in indicating that the phenotype, as defined by genetic liability evident from twin and family studies, extends far beyond the traditional boundaries of the diagnosis of autism as a major handicapping condition (Bailey, Palferman, Heavey, & Le Couteur, 1998; Rutter, 2000a; Rutter, Maughan, et al., 1997). This broader phenotype, or lesser variant, of autism may occur in as many as one in five of the first-degree relatives of individuals with autism, and the possibility that autistic features might extend even further into the general population, although not considered plausible a few years ago, cannot now be ruled out entirely.

One further feature to mention, with respect to recent autism research, concerns the finding that atypical varieties of autism may derive from congenital blindness (Brown, Hobson, & Lee, 1997) and profound early social and nutritional deprivation (Rutter, Andersen–Wood, Beckett, Bredenkamp, Castle, Groothues, Kreppner, Keaveney, Lord, O'Connor, & the ERA Study Team, 1999). The clinical pictures associated with these unusual risk factors seem somewhat different from "ordinary" autism, and it is doubtful whether they have much direct relevance for the causation of autism arising in more ordinary circumstances. Nevertheless, the findings do raise important questions about the heterogeneity of autism and the possibly different routes by which it may arise.

Depressive disorders

Up until about a generation ago, depressive disorders were rarely diagnosed in childhood because psychoanalytic theory claimed that they could not occur in the young (see Rutter, 1986b). The situation changed as people be-

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came more sceptical about the unvalidated claims of psychoanalytical theory, and as empirical observations, and especially the use of standardized interview methods, made it clear that depressive phenomena could and did occur in children. The pendulum has now rather swung to the opposite extreme, depressive disorders being diagnosed with a high frequency (Lewinsohn, Rohde, & Seeley, 1998). Indeed, there have even been claims that bipolar disorders are extremely common in high-risk groups (see Biederman, Faraone, Milberger, Guite, Meck, Chen, Mennin, Marrs, Ouellette, Moore, Spencer, Norman, Wilens, Kraus, & Perrin, 1996). A key problem is that, at one extreme, feelings of depression are simply a normal part of the human condition but that, at the other extreme, they constitute a serious psychosis. A key issue, from a developmental perspective, is whether or not the depressive disorders arising in early life are the same as those occurring in adulthood.

Several findings are crucial in drawing conclusions. First, longitudinal studies have made it clear that children who suffer from a depressive disorder have a greatly increased risk of a recurrence of depressive disorders in adult life (Harrington, Fudge, Rutter, Pickles, & Hill, 1990; Weissman, Wolk, Goldstein, Moreau, Adams, Greenwald, Klier, Ryan, Dahl, & Wickramaratne, 1999). However, it is also relevant that very few of the recurrences in adult life take the form of a bipolar disorder. That has been true even when samples have consisted of severe depressive disorders in childhood that have led to hospital admission (Weissman et al., 1999). Second, there is some evidence from questionnaire studies that depressive phenomena (not necessarily disorders) show a lower genetic component in childhood than they do in adolescence or adult life (Thapar & McGuffin, 1994). Early-onset depressive disorders may also be more likely to be associated with antisocial problems in the individual and in the family (Harrington, Rutter, Weissman, Fudge, Groothues, Bredenkamp, Pickles, Rende, & Wickramaratne, 1997), although this finding requires replication. The data are too sparse for firm conclusions, but it seems that there may be important differences between depressive disorders arising for the first time in childhood and those arising in adolescence or later (Duggal, Carlson, Sroufe, & Egeland, in press).

One of the striking features of depressive disorders is that they become much more frequent at some point during the adolescent age period and that this increase in frequency is much stronger in females than males (Silberg, Pickles, Rutter, Hewitt, Simonoff, Maes, Carbonneau, Murrelle, Foley, & Eaves, 1999). Clearly, a developmental perspective is likely to be useful in investigating why depression becomes so much more common during the teenage years and why the sex ratio alters from, roughly, parity in childhood to a marked female preponderance in adulthood (Peterson, 1993). Again, the evidence is thin on the ground, but findings from the Virginia twin study suggest that genetic influences related to both life events and depression play a significant role (Silberg et al., 1999).

Research from many research groups but especially that from Brown and Harris (1978) has shown the high frequency with which the onset of depressive disorder in adult life has been preceded by a stressful life event that carries long-term threat. On the other hand, it is obvious that many people have life events without developing depression, and it has been necessary to consider why there are such marked individual differences in response to apparently similar circumstances. Three features have seemed influential. First, many studies have shown that adverse experiences in childhood are associated with an increased liability to depressive disorder in adult life (Harris, Brown, & Bifulco, 1990). Second, the personal meaning of the event seems to influence its impact (Brown & Harris, 1978, 1989). Third, there is evidence that genetic factors may play a role in susceptibility to life stressors (Kendler, Kessler, Walters, MacLean, Neale, Heath, Corey, & Eaves, 1995; Silberg et al., 2000).

For a long time, it has been implicitly assumed that all depressive disorders can be treated as equivalent (apart from, perhaps, bipolar and severe melancholic varieties). However, recent findings have raised queries as to whether the factors involved in the onset of

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the first episode of the disorder may not be the same as those involved in later recurrences (Brown, Harris, & Hepworth, 1994). The issue is an important one, if only because most depressive disorders are recurrent (Judd, 1997). The evidence suggests that genetic factors may be more important in the lifetime liability to depression than in the onset of the first episode (Kendler et al., 1995), and that depressive-style attributions may also play a role in the persistence or recurrence of depression (Teasdale & Barnard, 1993). Psychiatric classifications have tended to draw sharp distinctions between depressive disorders on the one hand and anxiety disorders on the other, despite the fact that, at an individual level, there is very considerable overlap. Twin-study findings indicate that both anxiety and depressive disorders probably reflect the same genetic liability and that this may well reside, to a considerable extent, in the temperamental feature of neuroticism (Eley & Stevenson, 1999; Kendler, 1996).

Although there is good reason for concluding that adverse life experiences play a substantial role in the genesis of depressive disorders (see Rutter, in press-c), a caveat is necessary. Very little of the research is longitudinal, and because, in adults, the life experiences and the depressive symptomatology are reported by the same informant, some of the association may reflect reporting bias. Also, few of the studies have used genetically sensitive designs, and it is relevant that genetic factors play a role in the individual differences in the likelihood of experiencing and of reacting adversely to stress events, and that the genetic factors may also overlap with those that underlie depressive disorders (Kendler & Karkowski–Shuman, 1997; Thapar et al., 1998).

An immense amount has been learned from the study of depressive disorders in adults. There is a growing body of research findings on comparable disorders occurring in young people. Nevertheless, the adoption of a developmental perspective has raised a number of important queries that have yet to be fully resolved. There is a need for more longitudinal research that is explicitly focused on understanding the details of risk and protective mechanisms and that focuses on the less investigated features of gender differences and of age changes in frequency of depressive disorder, including features that maintain on or deflect individuals from a depression pathway.

Schizophrenia

Like autism, schizophrenia was long considered as a severe disorder that provided a sharp, qualitative departure from normality. It was not obvious that a developmental perspective would be useful because it was so uncommon for schizophrenic psychoses to begin in childhood.

Nevertheless, it has become increasingly appreciated that a developmental perspective is needed. (See Cicchetti & Cannon, 1999, for an overview of the issues involved in relating neurodevelopment to psychopathology.) Offord and Cross (1969) pointed to the evidence that about half of schizophrenic psychoses with an onset in adult life had been preceded by substantial nonpsychotic abnormalities in childhood. In the 40-odd years since then, the evidence that it is so has steadily mounted, with the impact from prospective longitudinal studies of the general population being crucial (Done, Johnstone, Frith, Golding, Shepherd, & Crow, 1991; Jones, Rodgers, Murray, & Marmot, 1994). It is not just that social and behavioral abnormalities may be evident in childhood (although that is the case) but also that there are developmental delays in a substantial proportion of individuals who later develop schizophrenia. A neurodevelopmental concept of schizophrenia has come to the fore (Cannon & Murray, 1998; Harrison, 1997; Murray & Woodruff, 1995). At first, it was thought that the neurodevelopmental risks were specific to schizophrenia. Recent findings have begun to cast doubt on whether that is, strictly speaking, so. It seems that rather similar risks may apply in the case of affective psychoses, although the findings are stronger in the case of schizophrenia (Jones, 1997). Genetic findings, like those in autism, have shown that the genetic liability extends beyond schizophrenic psychoses to include schizotypal and paranoid disorders (Kendler et al., 1995). The evidence is still in favor of

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a substantial degree of diagnostic specificity, but it is necessary to extend the concept of schizophrenia beyond psychoses, to include some forms of personality disorder. Precisely how and where to draw the boundaries is, however, still unclear.

There is good evidence that genetic factors play a substantial role in schizophrenia (Moldin & Gottesman, 1997), and the notion that it is a psychogenic disorder has largely been abandoned (with good reason). Nevertheless, one of the most striking findings in recent years has been the evidence that schizophrenia has a much increased rate among people of West Indian origin living in the United Kingdom. The rate is higher than that in the West Indies and is higher than in other ethnic groups within the United Kingdom (McKenzie & Murray, 1999). Careful analysis of putative causal influences indicates that some form of psychosocial stress or adversity that impinges differentially on people of West Indian background in the United Kingdom is likely to be responsible. Whether or not this form of schizophreniform disorder, in which psychosocial factors appear relatively strong, is different from other forms of schizophrenia remains to be established.

Intellectual development

It used to be said that if you were going to have a head injury it would be better to have it early because the effects were so much less—the so-called Kennard principle (1942). Research evidence has shown that this is a somewhat misleading oversimplification (Rutter, 1982, 1993). The age of the individual at the time of brain injury clearly is a most important factor, but its effects are somewhat more complicated than the original principle suggested. Contrasted with the situation in adults, lateralization effects following brain injuries in infancy are negligible. That is to say, unlike the situation in adults, left-brain injuries do not result in specific verbal deficits, and right-brain injuries do not lead to circumscribed visuospatial deficits. It seems that either there is a transfer of functions across the hemisphere when one side is damaged, or there is greater plasticity in brain development at that early age. On the other hand, early brain injuries are perhaps more likely than later ones to result in global intellectual impairment (Vharga–Khadem et al., 1992). Also, adverse intellectual sequelae are more likely if the injury is associated with epilepsy—suggesting that active brain dysfunction, rather than loss of function, may be critical. The results of research are clear cut in indicating marked age-dependent variations in response to brain injury, but the differences lie more in the pattern of sequelae than in whether or not cognitive impairment occurs. Until recently, the available evidence has given no clear indication of gene–environment interactions in relation to intellectual development, but new findings have suggested that genetic influences are less when children are reared in less advantageous environments (Rowe, Jacobson, & van den Oord, 1999). The possible importance of nature–nurture interplay needs to be reconsidered.

In most psychological theorizing about intellectual development, dietary factors have played a negligible role. It has, therefore, been quite surprising that well-conducted research, both naturalistic and experimental, comparing the effects of natural breast milk and formula milk on the later intellectual development of prematurely born children has shown substantial benefits of breast milk (Lucas, Morley, & Cole, 1998). It has been argued that some form of developmental programming may underlie the finding. The research is sound and well designed, but, as with all forms of research, independent replication with other research groups is required before too much is built into this finding, however compelling the evidence from one set of investigations. Moreover, the effect may not apply to full-term babies (Richards, Wadsworth, Rahimi–Foroushani, Hardy, Kuh, & Paul, 1998).

Numerous studies over the years have shown that IQ scores in the first few years of life are of negligible value for intellectual functioning in adolescence and adulthood. The findings had seemed to suggest a sharp discontinuity between earlier and later intellectual functioning. There were many reasons why there should be a degree of discontinuity, particularly because language skills become

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increasingly important in cognitive performance during childhood. Also, animal studies had shown that with maturation, different parts of the brain may come to be responsible for the same cognitive functions (Goldman–Rakic, Issernoff, Schwartz, & Bugbee, 1983). Nevertheless, it had seemed surprising that almost no continuity was evident. About 20 years ago, researchers began to adopt different strategies for studying cognitive functioning in infancy, recognizing that different skills might be needed at this early age. The result was the development of methods for attention and habituation. These showed substantial individual differences that, contrary to findings with developmental milestones of a traditional kind, did show modest continuities with intellectual performance as assessed in the usual ways in middle childhood (Bornstein & Sigman, 1986; Colombo, 1993; McCall & Carriger, 1993). Many questions remain, and it has not proved easy to define tests that are applicable across more than quite narrow age periods. Even so, the findings have been important in showing that there are continuities as well as discontinuities in intellectual achievement and that the continuities are heterotypic. That is to say, although it seems likely that the underlying function remains broadly the same over development, its surface manifestations differ markedly.

Looking Forward Into the 21st Century

As illustrated by this selective review of some key findings, considerable progress has been made in understanding many of the features presented by the domain of development psychopathology. Even so, it is equally apparent that major research challenges remain. In this final section of the paper, we outline a few of these key challenges. We make no attempt at being exhaustive in our coverage, but we do seek to highlight issues that have broad implications beyond the specifics of a particular trait or disorder.

Measurement issues

The last few decades have seen major improvements in the methods of measurement that are available for the assessment of psychopathology in childhood and adolescence, as well as in adult life (Shaffer, Lucas, & Richters, 1999). A rich assortment of questionnaire, interview, observational, and psychometric measures have been developed and for many of them there is good evidence of their reliability and validity. It might well be thought that we have got as far as we can in the field of measurement, but there are substantial unresolved problems. In many respects, the most pervasive, and perhaps the most surprising, finding has been the poor agreement between parents, teachers, and children on almost all aspects of psychopathology, and also of family experiences (Achenbach, McConaughy, & Howell, 1987; Elander & Rutter, 1996; Simonoff, Pickles, Meyer, Silberg, Maes, Loeber, Rutter, Hewitt, & Eaves, 1997).

There are many reasons why this might be so. Thus, for example, children in rating their own behavior may focus particularly on the contrast between how they are feeling now (or what they are doing now) as compared with how things were some time ago. Parents, on the other hand, are more likely to compare children with their siblings or with other children whom they know. Teachers, too, will make comparisons with other children but will have a much larger sample of comparison available to them. Inevitably, too, parents will focus more on what they have perceived in the context of the home, whereas teachers will do so in the context of the school, the situational demands of the two settings being rather different.

Evidence has accumulated that, with some behaviors such as hyperactivity, parents may exaggerate the contrasts between siblings, leading to an artifactually low correlation between ratings of the behavior of the twins or siblings (Simonoff, Pickles, Hervas, Silberg, Rutter, & Eaves, 1998). But the problem goes more broadly than that. It is not just that the agreement between informants is modest but also that the correlates tend to be rather different. Thus, for example, genetic influences on behavior as rated by children tend to be lower than as rated by parents (Hewitt, Silberg, Rutter, Simonoff, Meyer, Maes, Pickles, Neale,

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Loeber, Erickson, Kendler, Heath, Truett, Reynolds, & Eaves, 1997; Eaves, Silberg, Meyer, Maes, Simonoff, Pickles, Rutter, Neale, Reynolds, Erickson, Heath, Loeber, Truett, & Hewitt, 1997; Eley & Stephenson, 1999). Interest has also grown with respect to the ways in which ratings may be biased by the current mental state of the person making the rating (such as the effects of parental depression on the parental ratings Boyle & Pickles, 1997; Brewin, Andrews, & Gotlib, 1993). The findings of such comparisons have been informative, but it has to be said that the mechanisms involved in the similarities and differences among informants remain very poorly understood. Their elucidation requires systematic epidemiological–longitudinal studies focused on testing specific hypotheses about possible explanations.

Comorbidity

After years of neglect, the issue of comorbidity has become the "flavor of the moment," with the literature full of papers on its occurrence and patterning. Unfortunately, many of the studies are not as informative as they might be because the research has not been planned in a way that provides an adequate test of competing mechanisms. The fact that supposedly separate forms of psychopathology frequently co-occur in the same individual has been well demonstrated in numerous epidemiological studies (Angold, Costello, & Erkanli, 1999; Caron & Rutter, 1991). The key question is what the co-occurrence means; it is likely that some, perhaps much, is artifactual, deriving from mistaken assumptions in diagnostic concepts and boundaries. Alternatively, both may derive from the same set of intercorrelated risk factors. Or, yet again, the presence of one form of psychopathology may, through its effects, constitute a risk mechanism for another form of psychopathology. There are numerous examples in internal medicine of all of these (Rutter, 1997a), and they provide useful models for research strategies. It is crucially important to determine the meanings of comorbidity, both because its presence is likely to confuse and distort the meaning of findings (because what are being found as risk correlates of one disorder may, in fact, be the risk factors for some other form of psychopathology with which it happens to be associated), and because, if properly handled, the investigation of the mechanisms underlying comorbidity may be highly informative on risk and protective factors more generally.

Gender differences

The existence of major gender differences in rates of particular forms of psychopathology has been well recognized for many years (Earls, 1987; Eme, 1979, 1992), although their meaning has been ill understood (Rutter, 1970; Gualteri & Hicks, 1985; Taylor & Rutter, 1986). Thus, autism, attention-deficit hyperkinetic disorder, developmental language disorders, and delinquency all show a marked male preponderance, whereas adolescence-onset eating disorders (such as anorexia and bulimia nervosa) and depressive disorders in the years after childhood are both substantially more frequent in females. In all cases, it is necessary to check that the gender differences are not artifactual. For example, Zoccolillo (1993) has suggested that conduct disorders in girls are much more frequent than usually appreciated but that the diagnostic threshold for a number of symptoms needs to be different for girls than for boys. Likewise, Crick (1996) has argued that much aggression in girls has been overlooked because it is of a different form from that in boys. As both Nolen–Hoeksema (1987) and Moffitt et al. (2000) have sought to demonstrate, it is possible to postulate a range of different explanations and to undertake appropriate research tests to choose between competing hypotheses. Clearly, there is a need to understand the processes underlying these quite marked gender differences. We do not even know whether the causes of gender differences are the same across different disorders or whether quite different risk processes are operating. On the face of it, it is not obvious that the explanation for the male excess in delinquency and in autism would be the same. However, it would be premature to entirely reject the possibility of a unifying explana-

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tion. The point is that this major phenomenon, gender differences in rates of specific psychopathology, requires systematic study, and this is likely to involve somewhat different research strategies to those ordinarily employed. That is because the topic being investigated concerns differences between groups in level, rather than individual differences within a single population.

Cognitive processing

The study of cognitive processing has constituted one of the major growth areas in psychology over the last few decades. Two somewhat different phenomena seem to be important in relation to psychopathology (Rutter, 1987). On the one hand, there are disorders, such as autism, where the underlying problem seems to lie in the presence of cognitive deficits for particular kinds of mental processes. Similar arguments have been put in relation to schizophrenia (Frith, 1992) and antisocial behavior (Moffitt, 1993). On the other hand, some disorders seem to be characterized by unusual styles of cognitive processing, rather than deficits as such. That seems to apply, for example, to affective disorders and their associated depressogenic attributions (Teasdale & Barnard, 1993) and antisocial behavior with its associated tendency to make hostile attributions with respect to other people's actions (Dodge & Schwartz, 1997; Coie & Dodge, 1997). In some cases, too, it remains uncertain which type of cognitive feature applies. For example, attention deficits have long been considered as fundamental in hyperactivity disorders. However, despite earlier views to the contrary, the evidence now suggests that the basis does not lie in any defect in selective attention (Schachar, 1991). There are associated cognitive impairments, although it is uncertain how far they show specificity, but also there seem to be more stylistic problems in terms of impulsivity and a difficulty in delaying gratification (Sonuga–Barke, Taylor, Sembi, & Smith, 1992; Schachar, Tannock, Marriott, & Logan, 1995). In each of these cases, there is still a need to determine the degree of diagnostic specificity of the cognitive features, and there is a need to obtain a better understanding of how they are involved in the risk processes. For example, the evidence suggests that a depressogenic attributional style may be more important in the persistence or recurrence of depression than in its original onset (Teasdale & Barnard, 1993). In addition, there is an equally pressing need to understand the connections between brain and mind. The development of functional brain-imaging techniques, as represented at first by positron emission tomography (PET scans) (Bremner, Innis, Ng, Staib, Salomon, Bronen, Duncan, Southwick, Krystal, Rich, Zubal, Dey, Soufer, & Charney, 1997), and more recently by magnetic resonance imagery (MRI scans) provide one possible way forward (Rugg, 1997; Fletcher, Happé, Frith, Baker, Dolan, Frackowiak, & Frith, 1995; Schultz, Gauthier, Klin, Fulbright, Anderson, Volkmar, Skudlarski, Lacadie, Cohen, & Gore, 2000). In addition, however, studies of experiential effects on cognitive styles, and on their changing manifestations with age, are needed.

Sometimes functional imaging is "sold" as a means of seeing the brain in action, and of identifying the parts of the brain that are malfunctioning in the case of particular forms of psychopathology. That is, however, a somewhat misleading way of putting things. What the scans show is the part of the brain where the main metabolic activity, as reflected in blood flow, is taking part during particular cognitive tasks. That provides a most valuable means of differentiating among supposedly separate cognitive functions, and it should also be possible to determine whether the same brain areas are used to solve specific cognitive tasks by normal individuals and by individuals with psychopathological disorders. There is no doubt that this linking of brain function with the workings of the mind is going to increase our understanding of cognitive and affective processing, but on its own it will not identify brain pathology. It is important to appreciate that changes in behavior brought about by psychological treatments may also alter brain functioning as shown on scans, as illustrated by the case of obsessive–compulsive disorder (Baxter, Schwartz, Bergman, Szuba, Guze, Mazziotla, Alazraki, Selin,

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Ferug, Munford, & Phelps, 1992). Nevertheless, this is going to constitute one of the highly important technologies that will help us understand brain–mind interrelationships. At present, there are few such studies in developmental journals, but we predict that the situation is likely to change radically during the next decade.

Nature–nurture interplay

As already noted, with respect to the past, the study of the details of nature–nature interplay are likely to constitute another major growth area during the years ahead. The importance of personal effects on the environment, and of gene–environment correlations and interactions, has been well demonstrated, but we still lack adequate understanding of the role of these mechanisms in the emergence of psychopathology. Thus, almost all of the research into person effects on the environment have concerned very short-term effects (the study by Maccoby & Jacklin, 1983, constituting one of the few exceptions). Findings in the field of antisocial behavior suggest that these may be particularly important during the preschool years, and that the effects may be quite long lasting (O'Connor et al., 1998), but very few relevant data are available. The topic is a most important one because it necessarily changes the way in which both genetic and environmental risk mechanisms have to be thought about. If genetically influenced susceptibilities operate indirectly, rather than directly, it may be that the search needs to be not for the susceptibility genes for depressive disorder but rather the susceptibility genes for reactivity to environmental stressors. Similarly, if environmental effects are mainly evident in genetically vulnerable individuals, we need to understand how individual characteristics play a role in people's responses to the environment.

It is also important that we determine what it is that environmental effects do to the organism that enables the sequelae to be carried forward to a later age. As already noted, mechanisms to be considered are heterogeneous, spanning changes in brain structure, changes in neuroendocrine function, alterations in affect regulation, and changes in cognitive set or styles of cognitive processing.

As discussed more fully elsewhere (Rutter et al., 2000), there is also a major need to provide a much better understanding of environmental risk mechanisms. We know a good deal about what are key risk *indicators*, but we know much less about the environmental risk processes that they reflect. Research in the past has made clear that it is easy to mistake risk indicators and risk mechanisms, and that the theoretical policy implications are quite different according to which is which. For example, in relation to both childhood (Rutter, 1971b; Ferguson, Horwood, & Lynskey, 1992) and adult life (Harris, Brown, & Bifulco, 1986), it has become evident that parental loss is not usually a major risk mechanism. It is, however, a risk indicator because it tends to be associated with deficits in parenting that do constitute something closer to a risk mechanism. Research in the field of divorce and remarriage has also brought out the important point that it should not be considered as a time-limited event but rather as a process in which the family interactions before and after the marriage breakup are as important as the breakup itself (Block, Block, & Gjerde, 1986; Cherlin, Furstenberg, Chase–Lansdale, Kiernan, Robins, Morrison, & Teitler, 1991). Studies have begun to address the possible mediating factors that are involved in these risk processes operating over time (O'Connor, Thorpe, Dunn, Golding, & the ALSPAC Study Team, 1999b), and more research of this kind is clearly indicated (Gottlieb, 1997).

Heterotypic continuity

Several examples of heterotypic continuity have already been given. The social deficits in adult life associated with severe developmental disorders of receptive language constitute another (Howlin, Mawhood, & Rutter, in press). It is important to appreciate, however, that to establish heterotypic continuity, it is not enough to show that one behavior follows another during development. Such consecutive occurrence could occur for a whole variety of reasons. Rather, it is crucial to show

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that the different behaviors thought to represent heterotypic continuity have comparable correlates. Very little research of that kind has been undertaken. Genetic research strategies would be informative. Thus, for example, is there the same genetic liability for the measures of attention and habituation in infancy and for the measures of general intelligence in later childhood and adolescence? Similar questions arise with respect to the neurodevelopmental precursors of schizophrenia. Do they constitute a diagnostically nonspecific risk factor or are they indexing a specific genetic liability to schizophrenia? Again, genetically sensitive designs would provide an answer. Of course, the correlates to be examined should include psychosocial risk factors as well as genetic ones. Thus, with respect to the links between a lack of early selective attachments and a pattern of indiscriminate friendliness later, as shown by adoptees from Romania, it is necessary to ask whether the correlates are similar. That is, is there evidence not only of within-individual continuity between the two, but are the correlates similar? The fact that both show the same association with duration of privation begins to provide evidence of heterotypic continuity, but much more needs to be done. The negative findings with respect to disruptive behavior show that disproof is possible. Thus, although hyperactivity–inattention in Romanian adoptees was associated with the duration of privation, disruptive behavior was not (Kreppner et al., 2000) indicating that it probably had somewhat different origins.

Continuities and discontinuities between normal variations and disorder

Similar issues apply to the study of continuities and discontinuities between normal variations in behavior and disorder that might reflect an extreme on the same dimension. The need, again, is to determine whether the correlates are the same or different. The example of severe mental retardation has already been given with respect to discontinuity. Recent findings suggest that the same might apply with respect to normal variations in language and severe language delay (Dale, Simonoff, Bishop, Eley, Oliver, Price, Purcell, Stevenson, & Plomin, 1998). So far, the findings, from a twin study, apply only to 2 years of age (which is rather too early to determine language disorders), and the sample was biased in certain key respects. Nevertheless, the findings are provocative in showing that genetic influences for the extremes were much stronger than for variations within the normal range. The comparison of so-called individual heritability and group heritability (the latter referring to the difference between the extremes and the normal variation) provides a means of determining whether the differences are statistically significant (DeFries & Fulker, 1985). So far, there has been very little systematic research testing for continuities and discontinuities across the range of behavioral variation, and there are substantial practical problems in view of the very large numbers that are required to do this. Nevertheless, it does remain an important research priority for the future.

Developmental programming

Animal studies have provided strong evidence for the existence of certain forms of developmental programming by which environmental input influences brain structure and development. This is best established with respect to the role of visual input in the development of the occipital brain area underlying later visual function (Blakemore, 1991). The practical importance of the phenomenon is shown in human studies by the evidence that an uncorrected squint in early childhood leads to a later loss of binocular vision. Hormonal influences in animals have also been shown to affect the sexual differentiation of the brain (Gorski, 1996). In the fields of biology and internal medicine, there are also parallels with respect to the development of immunity and responses to diet (Bock & Whelan, 1991). In the field of psychopathology, there is a possible parallel in the case of the effects of profound early social privation on the later development of close confiding friendships and love relationships. We have already noted the extent to which such social deficits seem to persist in spite of a radical change for the bet-

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ter in the environment. The concept of developmental programming, at least as extended more broadly, remains a controversial one, and much has still to be learned about the biological processes that it reflects. The issues, however, are of obvious importance, and further research into the phenomenon is much needed.

Therapeutic mechanisms in treatment

Studies of treatment, as such, are not a central feature of developmental psychopathology, but an understanding of the mechanisms involved in psychotherapeutic efficacy are, because of the light they may throw on developmental processing generally (Rutter et al., submitted). The work of Clark and his colleagues in the field of panic disorders provides a good example of what can be done in this connection (Clark, 1996; Clark & Fairburn, 1997), and the studies of Patterson and his colleagues have begun to do the same in the field of antisocial disorders (Forgatch & DeGarmo, 1999). The effects of attachment-based interventions are similarly informative (van IJzendoorn, 1995). The field is, however, very underexplored up to now. The comparison of experimental and control groups will not, on its own, give this information. What is needed is the demonstration that, within the treated group, there is a systematic dose–response relationship between changes in the postulated mediated mechanism and changes in the target feature of psychopathology. Much greater use of this research stratagem should be made.

Conclusions

Developmental psychopathology perspectives have already provided many useful leads and lessons for the understanding of both normal development and disorder. For example, they have shown the importance of a pathways approach to causal processes that recognizes both direct and indirect effects, and which accepts that a single risk factor may have diverse consequences and that a single disorder outcome may arise by a variety of routes. It is likely that, with full understanding of the mechanisms involved, these complexities will reduce to a limited set of unifying processes, but the striving for parsimony needs to be tempered by a frank appreciation of the complexities that have to be explained. Similarly, developmental psychopathology approaches have underlined the importance of considering both continuities and discontinuities in development and of examining age-indexed variations in vulnerability to specific influences and of variations in onset and life course as indices of psychopathological heterogeneity. Concepts of risk and protective mechanisms, of resilience, of nature–nurture and person–environment interplay, of the cognitive and affective processing of experiences, of dimensional risk and protective processes and psychopathological outcomes, and of the interplay between different domains of development have all proved informative. But much work lies ahead. Developmental psychopathology arose out of a recognition of the value of combining developmental and clinical perspectives but also out of an appreciation of the limitations of the grand theories of the day. There is a continuing need to remain sceptical about the new evangelisms that have come to take their place, but equally the imperative must be to replace doubt with programmatic research that truly tests competing hypotheses and which has the potential of providing a real understanding of the range of causal processes as they apply across the span of behavioral and developmental variation.

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