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Developmental psychopathology: A paradigm shift or just a relabeling?

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Abstract

Developmental psychopathology is described as a conceptual approach that involves a set of research methods that capitalize on developmental and psychopathological variations to ask questions about mechanisms and processes. Achievements are described in relation to attachment and attachment disorders, autism, schizophrenia, childhood antecedents of adult psychopathology, testing for environmental mediation of risk effects, gene–environment interplay, intellectual and language functioning, effects of mentally ill parents on the children, stress and vulnerability to depression, ethnicity and schizophrenia, and drug response. Continuities and discontinuities over the course of development are discussed in relation to attention-deficit/hyperactivity disorder, antisocial behavior, eating disorders, substance abuse and dependency, pharmacological and behavioral addictions, and a range of other disorders. Research challenges are considered in relation to spectrum concepts, the adolescent development of a female preponderance for depression, the mechanisms involved in age differences in response to drugs and to lateralized brain injury, the processing of experiences, the biological embedding of experiences, individual differences in response to environmental hazards, nature–nurture integration, and brain plasticity.

Developmental psychopathology (DP) has been described as a conceptual approach that involves a set of research methods that capitalize on developmental and psychopathological variations to ask questions about mechanisms and processes (Rutter, 2008; Sroufe & Rutter, 1984). There are several distinctive features that are crucial: there is an expectation of *both* continuities and discontinuities; a recognition that it is not a theory or a discipline; there is a focus on individual differences and not universals; a focus on mediating processes and not just risks; a recognition that age is an ambiguous variable that reflects both biological maturation and the accumulation of experiences (Rutter, 1989); a focus on indirect and direct causal changes; and a recognition that indirect change may involve a complex mixture of both transformation and coherence (Rutter, 2008; Sroufe & Rutter, 1984). The reasons why a concept of DP was needed is that the "life" theories of systematizers (such as Freud, Piaget, and Erikson) had proved seriously inadequate, and it was clearly most unlikely that any one type of mechanism could explain everything.

Child psychiatry, like DP, was concerned with individual differences and mediating mechanisms, but it had become much too preoccupied with narrowly defined and conceptualized diagnostic issues. Research findings had made it all too clear that the concepts underlying the official diagnostic classification, such as DSM-IV and ICD-10 (World Health Organization, 1992), needed radical revision (Rutter, 2011; Rutter & Uher, 2012). There was much more overlap among diagnostic categories than had previously been appreciated and, in particular, there was no "clear water" between categories. Moreover, not only the risks for mental disorders were largely dimensional but also the disorders themselves were mainly dimensional (Rutter, 2003, 2011; Rutter & Uher, 2012; Uher & Rutter, 2012a). Developmental psychology had become focused too exclusively on universals in development rather than on individual differences in development, and it had also been concerned with quantification of trait stabilities over time, without any explicit recognition of both continuities and discontinuities (Rutter & Rutter, 1993).

For all of these reasons, it is clear that DP did involve a paradigm shift and not just a relabeling. Almost all people working in the field would agree on that. In contrast, there has been some disagreement on the breadth of the DP approach. Thus, for example, Cicchetti and Toth (2009), two outstanding leaders in this field, have emphasized interdisciplinarity and the need for multiple levels of analysis; the need to integrate DP with biology, neuroscience, and genetics; the need to apply knowledge to prevention and to intervention and to undertake translational research; and the need to consider culture and social context. All of those features are highly desirable, but they should apply to the whole of science and not just to DP. In addition, the notion of this degree of generalization needs to apply across studies and not necessarily within any one single study.

Achievements of DP

In considering the achievements of DP approaches, it is important to recognize that many of these are derived from research-

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ers who would not have identified themselves as DP researchers. For example, many of the advances came from adult psychiatrists and from genetics researchers (Rutter, 2010).

Attachment and attachment disorders

The first area of achievement lies in the field of attachment and attachment disorders (Rutter, Kreppner, & Sonuga-Barke, 2009; Rutter & Sroufe, 2000). Attachment research undoubtedly had its beginning in a theoretical approach about the meaning of attachment (Ainsworth, Blehar, Waters, & Wall, 1978; Bowlby, 1969). The DP qualities were shown in the recognition of the need to extend measurement to "disorganization" as well as insecurity (Main & Solomon, 1986) and to use a mixture of qualitative and quantitative approaches. Much more recently there was the demonstration that the pattern of social disinhibition more crucially involved social dysregulation than insecure attachment (Bruce, Tarullo, & Gunnar, 2010; Rutter et al., 2009). Moreover, it is evident that the pattern is mainly a consequence of an institutional upbringing (Zeanah & Gleason, 2011). Finally, there was the finding that problems of social relationships in early childhood were highly predictive of adult mental health outcomes, but there was very weak prediction from strange situation measurements in infancy (Grossman, Grossman, & Waters, 2005).

Autism

A second area of achievement concerned the very different psychopathological domain of autism. The DP feature here that was distinctive was the recognition of the need to move from a diagnostic approach to a study of the possible underlying mentalizing deficits (Hermelin & O'Connor, 1970). The term *theory of mind* came to the forefront, which was first studied in chimpanzees (Premack & Woodruff, 1978) and then in humans with normal children (Wimmer & Perner, 1983) and extended to children with autism (Baron-Cohen, Leslie, & Frith, 1985). In short, there was a focus on continuities and discontinuities between normality and disorder and a focus on possible mediating mechanisms. Both genetic and epidemiological evidence later showed that the genetic liability for autism extended far beyond the traditional diagnosis (LeCouteur et al., 1996; Rutter, 2000). Twin and family studies showed that there was a broader autism phenotype (BAP) that had a raised rate among the family members of those families with an autistic individual as compared with controls (Bailey, Palferman, Heavey, & Le Couteur, 1998). Imaging and other neural studies showed that physiological brain differences were detectable by 12 months of age before the clinical manifestations had become evident (Bosl, Tierney, Tager-Flusberg, & Nelson, 2011; Luyster, Wagner, Vogel-Farley, Tager-Flusberg, & Nelson, 2011). A rather different sort of finding was that social and language regression, usually evident about the age of 2 years, seemed to be much more common in autism than in other neurodevelopmental disorders (Pickles et al., 2009). Having demonstrated the continuities between autism and normality, this was an indication that there might be important discontinuities as well. This was apparent, too, in the finding that the rate of epilepsy was not only raised in autism but was distinctive in having a later age of onset than was usually the case (Bolton et al., 2011).

Schizophrenia

Schizophrenia provides the third domain where there have been major achievements of DP approaches. Probably, this began in the 1970s when Johnstone, Frith, Crow, Husband, and Kreel (1976) showed that schizophrenia was associated with enlarged ventricles, a finding carrying the inference that it was not just a functional psychosis. This provided evidence of discontinuity from normality. The second finding came from long-term, general-population, longitudinal studies showing that schizophrenia (but not bipolar disorder or depression/anxiety) was associated with impairments in language and/or motor function in the preschool years and with impairments in intelligence over the whole period from early childhood onward (Cannon et al., 2002). The implication was that schizophrenia had its beginnings in early onset neurodevelopmental impairment. At that time it was not clear whether this was an impairment that was part of the liability for schizophrenia or something independent from it that nevertheless carried a risk for the onset of a schizophreniform psychosis. The next finding was that schizophrenia was associated with minor psychotic-like features in late childhood/early adolescence (Poulton et al., 2000). Although these were surprisingly common in the general population (Laurens, Hobbs, Sunderland, Green, & Mould, 2011), they were associated with a substantially increased risk for the later development of schizophrenia. The fourth finding derived from a genetic, high family risk study in which abnormalities akin to schizotypy, as manifest in late adolescence, were associated with the development of schizophrenia in about half of the cases (Johnstone, Ebmeier, Miller, Owens, & Lawrie, 2005). In addition to all of these findings, Weinberger (1987) and Murray and Lewis (1987) argued that, despite overt psychosis usually not being manifest until early adult life, research findings indicated that schizophrenia should be considered to have arisen on the basis of an early neurodevelopmental impairment. Later findings suggested that, although this was the case, in some cases there were further neurodevelopmental changes that arose after the onset of the psychosis (Andreasen, 2010; Rapoport & Gogtay, 2011). However, there are still uncertainties regarding the extent to which the postonset changes reflect drug effects (Thompson et al., 2009).

Childhood antecedents of adult psychopathology

The fourth DP area of achievement lay in the evidence of the childhood antecedents of serious adult mental disorder. Findings from the Dunedin longitudinal study showed that over

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half of mental disorders that were diagnosed when the individuals were aged in their 20s and which had resulted in the receipt of treatment had been manifest in childhood/early adolescence (Kim-Cohen et al., 2003). Although there was substantial (but minority) continuity in the type of disorder, oppositional defiant and conduct disorders were the most frequent antecedents of adult disorders (Kim-Cohen et al., 2003). That is to say, as would be expected from any developmental perspective, homotypic continuity was frequent, but what was new was the evidence of heterotypic continuity. That is, the problems in childhood were often disruptive behavior, but the adult outcome lay in rather different phenomena, such as eating disorders.

Testing for Environmental Mediation of Risk Effects

The fifth area of DP achievements lay in the multiple different approaches to the testing of hypotheses of environmental mediation of risks for psychopathology. All researchers and clinicians will have been taught that statistical associations, however strong, do not necessarily mean a causal effect. Nevertheless, neither developmental psychologists nor child psychiatrists had paid much attention to the possible ways in which the causal inference of environmental mediation might be tested. The situation changed with the recognition of the ways in which "natural experiments" that pulled apart variables that ordinarily went together could do much to strengthen or weaken the causal inference (Rutter, 2007, 2012b).

Alongside this recognition, but also part of it, was the appreciation that genetically sensitive designs could be enormously informative. For example, Kendler and Prescott (2006) used the discordant twin design, in which one twin had experienced sexual abuse and the other had not, to test whether such abuse was associated with the later development of psychopathology. The findings provided strong confirmation of a likely environmental mediation of such effects. Jaffee and colleagues (2004) used a multivariate twin analysis to compare the effects of physical abuse and of corporal punishment on psychopathology. The findings were striking in showing that most of the effects of physical abuse were environmentally mediated, but most of the effects of corporal punishment were not. The implication was that the association between corporal punishment and mental disorders arose from the evocative effects of disruptive behavior in eliciting parental punishment. However, the findings also indicated that, although corporal punishment and physical abuse functioned rather differently, nevertheless, the extensive use of physical punishment carried an increased risk that this would later escalate into abuse.

One of the issues that needs tackling in testing for environmental mediation is the possibility that, although the risk factor is manifestly and rightly labeled an environment (e.g., as would be the case with parental discord or family poverty), risks may nevertheless be at least partially genetically mediated (Plomin & Bergeman, 1991). Thapar and Rice (Rice et al., 2009) had the creative idea that assisted conception provided just the sort of natural experiment that would be valuable for this purpose (Rice et al., 2009; Thapar & Rutter, 2009). Thus, some forms (such as sperm donation) maintain the genetic link between mother and child, whereas others (such as egg donation) disrupt that link. They used this approach to test the hypothesis that maternal smoking in pregnancy had a prenatal effect that predisposed to later attention-deficit/hyperactivity disorder (ADHD) and antisocial behavior. The assisted-conception design confirmed that there was a prenatal effect on low birth weight (as shown also by both observational studies and animal models), but there was not an effect on either ADHD or antisocial behavior. It was clear that statistical controls for confounding failed to provide what was needed in the absence of the natural experiment. The same finding that fetal exposure to maternal smoking led to an increased likelihood of a low birth weight but no causal effect on either ADHD or antisocial behavior has also come from other types of natural experiment (D'Onofrio, Rathouz, & Lahey, 2011; D'Onofrio, Van Hulle, Goodnight, Rathouz, & Lahey, 2011; Obel et al., 2011) such as sibling comparisons (Obel et al., 2011).

A different design is to focus on situations in which there is a radical, sudden and easily dated change of environment. The longitudinal study of children from profoundly depriving Romanian institutions who were adopted into UK families constitutes just such an example (Rutter, Kumsta, Schlotz, & Sonuga-Barke, 2012; Rutter & Sonuga-Barke, 2010). There were many features that made this a clear-cut natural experiment. The first was the massive improvement in functioning that followed the adoption and departure from the institution. However, what was of greater interest was not the immediate situation but what happened with respect to later development. The findings showed that a substantial minority of the young people did show deficits and that these took an unusual and distinctive form (such as quasiautism and disinhibited attachment). The environmental mediation inference was strongly supported by the association with the duration of institutional deprivation applied to those who did *not* have measurable subnutrition (Rutter et al., 2012) and the deficits persisted right up to the time of the most recent follow-up at age 15 years.

Another confounder in most studies is the possibility of social selection, namely, that the association derived from the opportunity of people to shape and select their own experiences. A natural experiment in this case is a situation in which all people are exposed to the same experience without the opportunity for any kind of selection or shaping. One such example is provided by the Dutch famine in World War II (Susser et al., 1996) and a similar famine in China (St. Clair et al., 2005). The findings in both cases showed that a prenatal exposure to famine conditions was associated with an increased risk of the development of schizophrenia many years later.

A rather different example is provided by the study of the effects of a casino set up on a Native American reservation. This was a natural experiment because, by Federal regulations in the United States, the setting up of a casino on a reservation

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had to be associated with the distribution of a proportion of the funds to all members living on the reservation without their needing to either ask for it or to "deserve" it. Costello, Compton, Keeler, and Angold (2003) had the ingenious idea to use their own Smoky Mountains longitudinal study to examine the effect of the setting up of the casino in the middle of a period of data collection. The findings showed that the relief of poverty was associated with a significant improvement in the children's behavioral functioning. It is striking that, despite the obvious need, very few developmental studies of social development have tested for environmental mediation (Rutter, 2012c). The need for such testing is the key feature of resilience research, but it is also a key feature of DP.

Gene–Environment Interplay

The sixth achievement of DP concerns gene–environment interplay (Rutter, 2012a). This involves at least three different types of interplay that are important. First, there are the effects of epigenetic mechanisms in the biological embedding of experiences (Meaney, 2010). Many questions remain about epigenetic effects, but what has been really exciting and important is the recognition that genes can only have effects if the genes are "expressed," and the expression of genes involves several DNA elements, stochastic (random) effects, and experiences. What this means is that although the environment cannot alter gene sequences, it can alter the effects of genes through affecting gene expression. That is directly relevant for DP because it concerns a key mediating mechanism, a central role of DP research. Second, another type of interplay concerns gene–environment correlations (*r*GE; Kendler & Baker, 2007). These are important because they constitute the mechanism by which environments can have genetically mediated effects (Plomin & Bergeman, 1991). However, they are also important because they point to the various ways in which people's behavior shapes and selects environments. Once more, the DP significance lies in the highlighting of mediating mechanisms.

The third type of interplay concerns gene–environment interaction (G × E). The initial reports of epidemiological evidence showing G × E in humans emphasized the environmental role of life events. As it has turned out, however, the evidence for G × E is much stronger in relation to maltreatment than it is for acute life events (Karg, Burmeister, Shedden, & Sen, 2011). This means that the causal path begins with maltreatment in early childhood with the effect being evident in psychopathology only manifest in late adolescence or early adult life. In other words, the biological pathway involves very substantial continuity over the developmental course. The second G × E finding that is crucial for DP is that human experimental studies utilizing structural and functional brain imaging have shown that the G × E neural effects are evident in individuals who have already been screened for an absence of psychopathology (Hyde, Bogden, & Hariri, 2011; Meyer-Lindenberg & Weinberger, 2006). This shows a continuity between normality and disorder.

Intellectual and Language Functioning

The seventh DP achievement concerns the effects in relation to intellectual and language functioning. Two findings require particular emphasis. The first is that unilateral brain damage in the dominant hemisphere leads to aphasia in adults but generalized intellectual impairment in infancy (Rutter, 1993; Vargha-Khadem, Issacs, Van Der Werf, Robb, & Wilson, 1992). That is to say, unilateral brain damage has a marked effect in all age groups, but the pattern is quite different in infancy compared with later childhood, adolescence, and adult life. The second finding is that severe intellectual disability is frequently associated with major pathogenic genes, but mild disability is not to the same extent (Einfeld & Emerson, 2008). Thus, Down syndrome is a major cause of severe intellectual disability, but it is much less often associated with mild intellectual disability. Pathogenic genetic mutations do account for a small proportion of cases of mild intellectual disability, but for the most part, mild disability operates at the extreme end of a normal distribution. That is not the case with severe intellectual disability. The implication is that there is almost complete discontinuity between severe intellectual disability and normality, whereas continuities are relatively strong in the case of mild disability.

Effects of Mentally Ill Parents on the Children

The eighth DP achievement concerns the effects of mentally ill parents on the children. Using an extended children of twins design to study the genetic environmental mediation of transgenerational transmission, Silberg, Maes and Eaves (2012) found that parental antisocial disorder had an environmentally mediated effect on child depression, a genetically mediated effect on ADHD, and an effect on child conduct disorder that involved both genetic and environmental mediation (Silberg et al., 2012). A somewhat similar pattern applied to parental depression (Silberg, Maes, & Eaves, 2010). Parental depression had a direct, environmentally mediated effect in increasing the risk that the children developed depression. There was also an increased risk of conduct disorder in the children, but this involved a mixture of genetic and environmental effects. This research made it abundantly clear that a general answer on whether the effects were environmentally or genetically mediated was going to be completely wrong. The mode of mediation varied according to the child outcome.

A rather different approach concerned looking at the effects of intervention on the treatment of mentally ill parents with respect to the benefits for the children. A meta-analysis showed that preventive interventions (with cognitive, behavioral, and psychoeducational components) were effective for preventing both behavioral and emotional problems in the children (Siegenthaler, Munder, & Egger, 2012). Using longitudinal analyses, other research showed that changes in the level of maternal depression were accompanied by parallel trajectories with respect to child symptoms, with the main ef-

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fect appearing to be from parent to child but also including some bidirectional effects (Garber, Ciesla, McCauley, Diamond, & Schloredt, 2011; Garber & Cole, 2010; Kouros & Garber, 2010).

Stress and Vulnerability to Depression

The ninth DP achievement concerns research seeking to examine mediation effects in relation to the associations between the experience of stress and vulnerability to depression. There is a good deal of evidence from many studies that stress is associated with depression, but the suggested mechanisms are very variable. Thus, three main models have been proposed. First, there is stress sensitization, meaning that a sensitivity to stressors increases with the number of stress experiences. Second, there is the stress inoculation model, that is, stress experiences have a diminishing effect over the course of repeated stress experiences. Third, there is diathesis–stress model, that is, a vulnerability to stress associated with continuing biological features. Using longitudinal data, Garber and colleagues found little support for the stress inoculation model but evidence in favor of both the stress sensitization and diathesis–stress models (Morris, Ciesla, & Garber, 2010). This is an important topic, but it is one in which definitive answers are hard to come by. Thus, animal models have shown that, provided the stress is mild and manageable, there are stress inoculation effects in the sense that they lead to an increased resistance to later stressors (Rutter, 2012c, 2013).

Ethnicity and Schizophrenia

The tenth DP achievement concerns the association between ethnicity and schizophrenia. There is good evidence that the incidence of schizophrenia (and other psychoses) is substantially elevated in individuals of Black Caribbean or Black African origin living in the United Kingdom as compared with that of individuals of similar ethnicity living in the Caribbean and that of indigenous, White individuals living in the United Kingdom (Fearon et al., 2006; Jones & Fung, 2005). The findings showed that the effects were particularly mediated by social disadvantage in adult life and separation from parents (Morgan et al., 2009). Other research (Schäfer & Fisher, 2011) has shown the role of childhood trauma in psychosis and the DP relevance lies in the focus on both continuities across diverse social risks and the importance of these in an illness that involves strong genetic influences.

Drug Response

The eleventh achievement concerns drug responses. Rapoport showed more than 30 years ago that the response to stimulants was very similar in the general population and in patients with ADHD (Rapoport et al., 1980). It was previously considered a paradoxical response, but stimulants improve attention in everyone to some degree. In contrast, there is a difference according to age in that stimulants are recreational drugs in adults but are not so in children. Children with ADHD benefit from taking stimulants, but they do not actually like the sensation that the drug provides. Another age effect is that the adverse effects of psychotropic drugs, such as risperidone, are much more common in children than in adults (Fedorowicz & Fombonne, 2005; Stigler, Potenza, Posey, & McDougle, 2004). Finally, depression in childhood does not respond to tricyclics, whereas depression in adult life usually does. This is not a question of resistance to antidepressant medication, because depression in young people does respond to selective serotonin reuptake inhibitors; it is just that it does not seem to respond to tricyclics (Hazell, O'Connell, Heathcote, Robertson, & Henry, 1995).

Continuities and Discontinuities Over the Course of Development

The 12th achievement concerns a better understanding of continuities and discontinuities over the course of development.

ADHD

ADHD is ordinarily thought of as a clinical disorder, but it is evident that the genetic liability operates across a dimensional range and not just on an extreme representing disorder (Thapar, Harrington, Ross, & McGuffin, 2000). Classification has traditionally dealt with subcategories in terms of whether it is predominantly inattention or predominantly hyperactivity/impulsivity or some combined pattern. At any one point in time these appear rather different, but it is clear that the differences among them are not stable over time and there are systematic changes in pattern with increasing age. Attention deficits tend to increase and hyperactivity/impulsivity tends to decrease (Larsson, Dilshad, Lichtenstein, & Barker, 2011).

It has also been found that inattention/overactivity is a consequence of an institutional upbringing (Rutter & Sonuga-Barke, 2010; Vorria & Ntouma, 2012). This need not be a surprise, because ADHD is a multifactorial disorder and, despite high heritability, clearly environmental factors will play a part. However, this cannot be the usual way in which environmental factors operate because an institutional upbringing has an infrequent association with ADHD. Moreover, the inattention/overactivity found following institutional deprivation is somewhat unusual in its association with quasiautism and/or disinhibited attachment.

Antisocial behavior

It has long been realized that antisocial behavior is heterogeneous. At one extreme, the majority of people commit antisocial acts at some time, although they are generally well functioning (Rutter, Giller, & Hagell, 1998). At the other extreme, some antisocial behavior persists into adult life in association with substantial social dysfunction. Moffitt's (1993) paper brought clarity through its differentiation of life-course-persistent (LCP) and adolescence-limited (AL) antisocial behav-

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ior. The former was said to be characterized by a childhood onset, individual neurodevelopmental deficits, and chronic family adversity. In contrast, the latter was viewed as largely developmentally normative and associated with an antisocial peer group. Subsequent research has largely supported the LCP and AL distinction, but it has indicated the need for three main modifications. First, as shown earlier by Robins (1966, 1978) and confirmed by Odgers and colleagues (2008), about half of those with an early onset do *not* continue their antisocial behavior into adult life, pointing to the need to recognize a substantial childhood-limited (CL) group. Second, adolescent-onset antisocial behavior often constitutes a precursor of more severe, long-term social malfunction (Odgers et al., 2007). Third, the adolescent-onset group differed from nonantisocial youth in showing both individual and family adversity, albeit less than that associated with LCP (Roisman, Monahan, Campbell, Steinberg, & Cauffman, 2010). In summary, in line with a DP perspective, antisocial behavior should be conceptualized as showing both continuities and discontinuities across the twin spans of behavioral variation and life span development.

Eating disorders

The strictness of the criteria for anorexia and bulimia nervosa in DSM-IV and ICD-10 meant that the great majority of eating disorders could not be included and had to be put in a residual category of eating disorders not otherwise specified. The evidence clearly indicated that these were part of the same group of disorders and that the boundaries needed to be altered to respond to that recognition (Uher & Rutter, 2012b). In contrast, there is much more uncertainty over the continuity between the dieting and weight preoccupations of a high proportion of adolescent girls and anorexia nervosa. It is also clear that there are important differences, as well as strong similarities, between anorexia nervosa and bulimia nervosa. The former has a substantially earlier age of onset and the latter usually does not begin until late adolescence or early adult life. However, it is not at all uncommon to find clinical pictures in which, over the course of time, one is followed by the other and then returns to the previous pattern.

The greatest uncertainty concerns the possible continuities and discontinuities between what used to be called eating disorders of childhood (such as pica, regurgitation, and extreme faddiness) and anorexia/bulimia nervosa. It is highly likely that, although these will be treated as separate diagnoses, they will be put in the same cluster in DSM-5 and ICD-11. There are few longitudinal data that provide a good answer on continuities and discontinuities, but the existing evidence suggests that there is a complex mixture of both (Uher & Rutter, 2012b).

Substance abuse and dependency

This is another group of disorders that seem to show a complex mixture of continuities and discontinuities over both the span of behavioral variation and over developmental course. The existing classification in ICD-10 has as its prime feature the specific drug or substance that is involved. That seems all back to front, as it is the nature of the particular problem that should constitute the prime basis and not the drug. That would be so in any case, but the argument is greatly strengthened by the universal observation that it is usual for substance abuse to involve several, often many, different substances (Weinberg, Harper, & Brumback, 2008). The notion that typically it is just one substance is simply wrong. At one time, it was assumed that there was a hierarchy wherein "softer" drugs were always taken first before proceeding to "harder" drugs, such as opiates or cocaine. Robins' pioneering studies of soldiers in Vietnam indicated how mistaken this was (Robins, 1993; Robins, Davis, & Goodwin, 1974). The soldiers tended to start with hard drugs, and she concluded that this was because they were more readily available in Vietnam than were some of the softer drugs (which, of course, is not the case in the general population). A particular continuity issue concerned the question of whether the use of certain drugs constitutes a "gateway" to the use of other, more serious substances. Clearly there is no necessary progression of this kind, but careful studies have indicated that marijuana does have this gateway function in some instances (Lynskey, Vink, & Boomsma, 2006; Stenbacka, Allebeck, & Romelsjoe, 1993). It is highly unlikely that the gateway constitutes any pharmacological pathway for the reasons already given. It is probably a question of attitudes, availability, or peer group pressures.

A further continuity/discontinuity question concerns the links between occasional experimental use of substances and gross dependence on or abuse of them. For obvious reasons, experimental use is likely to be the starting point, but the evidence also suggests that environmental factors play a major role in occasional, experimental use, whereas genetic factors have a more substantial role with respect to abuse and dependency (Goldman & Ducci, 2007).

Another key issue concerns possible discontinuities according to age. The best documented example of this kind concerns the effects of cannabis in increasing the liability to schizophreniform disorders (Arseneault et al., 2002; Casadio, Fernandes, Murray, & Di Forti, 2011; Rutter, in press). Cannabis taken only for the first time in adult life is not associated with any substantial increase in risk. By contrast, cannabis use that begins in childhood or adolescence, particularly if it is heavy regular use, is associated with a substantial increase in liability. This does not seem to be a purely social phenomenon, because the same does not apply to other substances, but it does apply to animal models (Rutter, in press) when cognitive functioning is treated as a dependent variable (as there is not a satisfactory rodent model of schizophrenia).

It used to be said that the early use of alcohol was associated with an increased risk of alcoholism in later life (Grant, Stinson, & Harford, 2001). General population studies do show such a statistical association, but the evidence indicates

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that this is likely to be a function of shared liability rather than a causal effect (McGue, Iacono, Legrand, & Elkins, 2001). Thus, for example, if early use is associated with an earlier age of puberty, leading to an increase in drunkenness in adolescence, it is not associated with an increase in alcoholism in adult life (Stattin & Magnusson, 1990).

Pharmacological and behavioral addictions

A further continuity question concerns the continuities or discontinuities between the abuse of substances and behavioral addictions. Thus, should problematic gambling or gross internet dependency be viewed as part of the same group of problems as exemplified by the abuse or dependency of substances? The evidence suggests that there are commonalities, but there are equal differences (Grant, Potenza, Weinstein, & Gorelick, 2010; Petry, 2007; Potenza, 2006). The value of a DP perspective is that it forces one to ask questions rather than fall back on some theoretical position that it must be the one or it must be another.

Continuities and discontinuities with other disorders

It is beyond the scope of this paper to provide an exhaustive summary of the evidence on all mental disorders. However, it is clear that very similar questions arise with respect to, for example, obsessive–compulsive disorder (OCD), tics, elimination disorders, sleep disorders, and posttraumatic stress disorder (PTSD). Thus, OCD is a distinctive syndrome characterized by obsessional thoughts or compulsive acts. Such thoughts comprise ideas, images, or impulses that enter the individual's mind again and again in a stereotyped form. Compulsive acts are similarly rituals of stereotyped behaviors that the individual feels compelled to perform repeatedly, although they are not particularly enjoyable, nor do they result in the completion of inherently useful tasks (Leckman et al., 2010; Rapoport & Shaw, 2008). Such features are extremely common in the general population and many ritualistic and supposedly "magical" behaviors are part of normal development, as well as part of normal adult behavior (e.g., the ritualized behaviors of many top tennis stars). Thus, OCD appears to be a continuously distributed trait with biological continuity between normality and psychopathology. However, the normal ritualistic/magical behaviors seen in many young children do not seem to predict overt OCD when they are older. A further continuity issue concerns the possible links between OCD and other apparently similar disorders such as hoarding (Mataix-Cols et al., 2010; Pertusa, Frost, & Mataix-Cols, 2010; Phillips, Stein, et al., 2010), hair-pulling (Stein et al., 2010), and body dysmorphic disorder (Phillips, Wilhelm, et al., 2010). A DP perspective requires that questions about continuities/discontinuities be posed, but unfortunately, the empirical research findings do not provide any definitive answer. OCD features commonly co-occur with anxiety disorders and with tic disorders, and there is continuing uncertainty on the extent to which this co-occurrence reflects a shared underlying liability. There is some evidence that there may be qualitative differences between tic-related forms of OCD and causes of OCD without a personal or family history of tics (Hounie et al., 2006).

Similar issues apply to tic disorders, which span simple motor tics that are common in the general population and are associated with little social impairment, and the much less common, severe disorder usually referred to as Tourette syndrome, which involves a combination of vocal and multiple motor tics (Leckman & Bloch, 2008; Walkup, Ferrão, Leckman, Stein, & Singer, 2010). Once again, uncertainty remains on whether the difference in severity and frequency reflects quantitative or qualitative differences.

It may seem that elimination disorders should constitute the prototype of disorders that represent a quantitative rather than qualitative variation from normality. No babies are continent at birth, but almost all gain continence with increasing age, although there is substantial individual variation in the age when continence is achieved (Butler, 2008). It may be that nighttime wetting fits the normal variations model; but, even with that phenomenon, there is likely heterogeneity, with a sex difference between nighttime wetting and daytime wetting and variations in both according to the presence or absence of bladder dysfunction (Butler, 2008). Fecal soiling is both different and more complicated, with some varieties fitting the normal variation model and others (such as the deposition of feces in inappropriate places) obviously not. As with other disorders, there appear to be both continuities and discontinuities across the span of behavioral variation (Butler, 2008).

Much the same applies to sleep disorders (American Academy of Sleep Medicine, 2005; Dahl & Harvey, 2008). Thus, behavioral insomnia and nightmares are so common that, although they may be problematic, it is not obvious that they constitute a disorder. Sleep walking is less common and is not ordinarily part of normal development. Narcolepsy (which involves sleep attacks, sudden loss of muscle tone without change in consciousness, an inability to move after waking up, and dreamlike imagery before falling asleep) is the most clearly abnormal sleep phenomenon, and it is usually considered a chronic neurological disorder.

PTSD (probably better termed *hyperarousal/hypervigilance syndrome*) is a behavioral pattern that is characterized by specific features and that follows an exceptionally severe traumatic event that involves actual physical injury or the threat of actual violence or injury (Brewin, Andrews, & Rose, 2003; Zohar et al., 2009). It is not a developmental phenomenon, but it does arise in children as well as adults. In a sense, PTSD represents an understandable response to an extreme circumstance, but it is not a normal phenomenon outside of that situation. The pattern involves a mixture of personal liabilities and unusual experiences.

In short, in line with DP perspectives, each of these disorders includes a varied mixture of both continuities and discontinuities across the span of behavioral variation. Questions come up most obviously in relation to classification issues,

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but these are not just nosological matters, because the answers to the questions on continuities and discontinuities are fundamental to an understanding of the meaning of the disorders and the mediating mechanisms for them.

Overview

Continuities and discontinuities over the course of development

Putting together the findings as already discussed, there is reasonably consistent evidence for continuities between normality and psychopathology with respect to depression, conduct disorder, autism, schizophrenia, mild intellectual disability, and attention-deficit disorder. In contrast, there is substantial evidence showing discontinuities with respect to severe intellectual disability, social regulation disorder and attachment insecurity, autism (as possibly shown through brain imaging findings) and schizophrenia (also possibly shown via imaging findings). In other words, there are several sorts of psychopathology wherein there is evidence for both continuities and discontinuities. This applies to both autism and schizophrenia. This underlines the importance of the DP perspective involving acceptance of both continuities and discontinuities and not an assumption that there will always be the one and not the other.

Much the same applies to continuities and discontinuities over the course of development. Continuities are strongly evident with respect to antisocial behavior, depression, and ADHD, but even with these, there is also discontinuity. For example, distinctions have been shown between childhood restricted antisocial behavior, AL antisocial behavior, and LCP behavior (Moffitt, Caspi, Harrington, & Milne, 2002). Several studies, including the Dunedin study, have shown that in childhood there is an equal sex ratio for depression but during adolescence, a female preponderance develops and continues into adult life (Hankin et al., 1998). It is not quite clear why this occurs, and it is notable that this is the case despite a substantial continuity between depression in childhood and depression in adult life (Harrington, Fudge, Rutter, Pickles, & Hill, 1990). The key issue here is whether change is a function of puberty as observed or hormonal changes as measured. Of course, the two are closely interconnected, but they are not the same and the mechanisms will be different. The timing will also not be the same because the hormonal changes begin well before the observed pubertal changes. Angold, Costello, Erkanli, and Worthman (1999) showed that it was the hormonal changes that were critical and not puberty, each being controlled when looking at the other. In contrast, it cannot be the direct effect of hormones because therapeutic administration of hormones does not have much effect on mood. Rather, it seems that the hormonal changes provide the necessary precondition for other mechanisms to come into play (Angold, 2008).

The evidence for discontinuities over the course of development is most obvious in relation to the effects of unilateral brain damage in the dominant hemisphere with respect to effects on language and intelligence. Mention has already been made of the change in sex ratio for depression in adolescence, and there are also changes between childhood and adult life in drug response.

Continuities between normality and pathology

There are at least some half a dozen disorders for which there is substantial evidence for continuities between normality and pathology. This has long been evident in depression and conduct disorder where the decision as to when a disorder presents is arbitrary to some extent, because it is assessed on the basis of the overall level of functioning and persistence. Mild intellectual disability also shows substantial continuity in the great majority of cases, although there is a proportion (it remains uncertain how high a proportion this is) due to some pathogenic mutation. Attention-deficit disorder also largely functions as a dimension, although the possibility of an extreme variety that is qualitatively different cannot be firmly ruled out as yet.

Until fairly recently most people would have considered autism a disorder that is distinct from normality, but the evidence of the validity of a BAP indicates that the diagnosis extends much more broadly than used to be thought. Moreover, at least some cases (perhaps most) of the BAPS share the same genetic liability (LeCouteur et al., 1996). What remains uncertain is what it is that causes liability to lead to the overt handicapping disorder rather than to the much milder problems of the broader phenotype. Is that simply a function of degree of genetic liability or is there some kind of two-hit mechanism, and if there is, what is the other hit that is involved?

Somewhat similar questions arise with respect to schizophrenia. The psychosis due to manifest schizophrenia would seem to be quite different from normality, but the strong association with schizotypal disorder indicates that this, too, extends much more broadly than used to be thought. Moreover, the risk factors in neurodevelopmental impairment evident in early life similarly raise questions about how the disorder develops. It certainly is not an inevitable progression because even with the very high risk groups studied in adolescence/early adult life, only a proportion (albeit a sizeable proportion) go on to show overt schizophrenia. Again, what are the factors that take it over that threshold?

Discontinuities between normality and pathology

Severe intellectual disability is the most obvious diagnosis that shows very little continuity with normality. Variations in IQ within the normal range are not explicable in terms of the pathogenic mutations found with severe intellectual disability. Of course, even within the severe disability range, the degree of disability does have a prognostic value for the level of functional impairment in adult life, but for the most part, there is discontinuity with normality.

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Disinhibited social engagement disorder, previously termed social disinhibition disorder, seems qualitatively different from the variations in attachment security/insecurity seen in the general population. Not only is there a strong association with institutional deprivation but also the particular form of the disorder is evident more in the interactions with strangers than with parents, the opposite of what is found ordinarily.

Autism has been discussed in terms of continuities, but the imaging findings suggest that there may also be an important degree of discontinuity. The imaging findings, to date, are not of a kind to enable a definitive answer one way or the other on this point but there may be a qualitative difference. The same applies to schizophrenia. In summary, there are instances where elements of a disorder may show continuity and yet there may also be elements of discontinuity.

Numerous examples have been given of discontinuities over the course of development. This applies, for example, to the effects of unilateral brain damage in the dominant hemisphere in relation to aphasia, the change in sex ratio for depression in adolescence, the changes between childhood and adult life in drug response, the change in rate of side effects of psychotropic drugs, the change in pattern of ADHD, the risks for schizophreniform disorders stemming from heavy *cannabis* use, and several aspects of the course of eating disorders.

Most of the topics considered have provided evidence on mediating mechanisms. This is most obviously the case in the use of natural experiments to test for environmental mediation: the study of G×E, *r*GE, epigenetics, and the biological embedding of experiences. It has also been evident in the effects on children of parental mental disorder that differ according to the child disorder being considered and the different models of reactivity distress. In many instances, the answers have not provided a definitive delineation of the mechanisms involved; but the use of a DP perspective has meant that this is a major aim, and the research has given rise to important findings.

Research Challenges

DP is characterized by using continuities and discontinuities across the span from normality to overt disorder, and the span across the course of development in order to ask, and seek to answer, questions about the mediating mechanisms and processes involved in the continuities and discontinuities found. There is no shortage of reports describing continuities and discontinuities, but there is a paucity of investigations into the mediators of both. The first need, therefore, is to better delineate what needs explaining. For example, there is currently much psychopathological interest in spectrum concepts (e.g., with respect to autism, schizophrenia, OCDs, and eating disorders), but there has been very little attempt to determine the possible processes that may be involved. Thus, how does BAP become transformed into autism "proper," how do the various developmental precursors of schizophrenia lead to manifest schizophrenia, and with respect to both autism and schizophrenia, why does the progression occur in some individuals but not in others? In much the same way, why does a childhood onset of antisocial behavior predispose to persistence into adult life, yet this progression applies to less than half of childhood-onset cases? Are genetic or environmental influences largely concerned with LCP?

It is clear that depressive disorders become more prevalent in adolescence and that this increase is associated with the development of a female preponderance. Why does this happen? What are the mechanisms that underlie the development of a female preponderance of emotional disorders in adolescence but a male preponderance of most (but not all) neurodevelopmental disorders in early childhood? Theorizing has tended to focus on the sex difference found in different disorders (such as the suggestion that autism represents extreme maleness), but DP findings suggest that this focus is too narrow. Why does the male preponderance apply to syndromes as seemingly diverse as dyslexia, ADHD, and autism?

The research world has thus far paid little attention to understanding the mechanisms involved in age differences in response to drugs and to lateralized brain injury. Why do stimulants have similar beneficial effects in both those with ADHD and normal individuals and have similar effects in both children and adults yet show important differences in side effects? Why are stimulants recreational drugs in adults but seemingly not so in children? Of course, one answer is that children do not like the feelings brought on by stimulants, but why is that so? Why do young people differ from adults in their response to tricyclic antidepressants but not to selective serotonin reuptake inhibitors? Why are adverse side effects to psychotropic medication greater in children than in adults?

Although it has long been apparent that individuals of all ages actively process their experiences, the mechanisms involved have been very little explored with respect to either the processing or the effects of this on individual differences in behavioral outcomes. Similar questions arise on children's shaping and selecting of experiences and, especially, on the ways in which their behavior has evocative effects. DP perspectives indicate that the greatest need is to understand the behavioral mediation of these evocative effects.

A key issue with respect to the persistence of environmental effects beyond the duration of the risk experience concerns the biological embedding of experiences, in other words, how environments "get under the skin." It is clear that epigenetic changes represent one such possibility (Rutter, 2012d) but so do changes in the neuroendocrine system (Gunnar & Vazquez, 2001, 2006), neural effects, and altered mental models. The first challenge is to determine which of these mainly accounts for the effects on behavior and the second is to determine whether the biological features account for individual differences in response, for example, between the sensitizing and steeling (strengthening) effects of stress.

In this connection, it is essential to investigate the huge individual differences in people's responses to all forms of

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environmental hazard. It is clear that, even with severe trauma such as child abuse, some individuals are surprisingly resistant to ill effects (Rutter, 2012c, 2013). Part of the explanation is to be found in the operation of G × E (Rutter, in press) but that is by no means the only mechanism (Rutter, 2013). In addition, it seems that although the main focus has been on response to stress, the main G × E is seen with maltreatment in early childhood and not acute stress. This suggests a biological pathway beginning early and extending into adult life where the psychopathology becomes apparent. Moreover, although most research reports concern the response to *adverse* experiences, evolutionary concepts and a growing body of evidence suggest that the phenomenon concerns a sensitivity to positive and negative environments (Ellis, Boyce, Belsky, Bakermans-Kranenburg, & van IJzendoorn, 2011). As already noted, the G × E findings apply to individuals *without* psychopathology as well as to those with disorder. DP concerns, to understand *both* continuities and discontinuities over behavior and over the life course, come to the forefront. Similar issues arise with respect to the need to find out whether the particular unusual responses to institutional deprivation (Rutter & Sonuga-Barke, 2010) also apply to deprivation in family settings.

The epidemiological/longitudinal study findings on G × E with respect to psychopathology rightly received a lot of attention, but as the investigators themselves appreciated, the evidence does not show the biological pathways involved. Of course, G × E does suggest that the pathway for the gene effects and the pathway for the environment effects are likely to be closely related. In order to determine the biological processes, human experimental approaches and animal models are needed and are being used (Rutter, in press-b). Natural experiments also have an important role to play. The DP challenge is to make maximal use of diverse, experimentally oriented research strategies to understand the biology.

Finally, there is the fundamental challenge of understanding the neural underpinning of the workings of the mind. Aspects of that issue have been noted in relation to G × E, but they apply most strikingly in relation to brain plasticity and the recovery of function after serious brain injury. Neuroscience findings (Bavelier, Levi, Li, Dan, & Hensch, 2010) have shown that plasticity continues into adult life, although it diminishes with age. Most crucially, it has shown that there are influences that can extend or curtail plasticity. The possible practical implications were highlighted by Taub's finding (Taub, 2000; Taub, Ramey, DeLuca, & Echols, 2004) that intensive massed practice of the damaged limb led to improved functioning in both brain-injured adults and children with cerebral palsy. The suggestion was that this might reflect neural changes, but did it? A recent experimental study with rodents (van den Brand et al., 2012) showed, following transaction of the spine, that there was new growth of neurons and increased synaptic connections. The DP challenge is to determine if mediation of psychological change can be due to induced brain changes. Of course, the answers will also have implications for the lateralized brain injury effect differences according to age.

Conclusions

To return to the points made in the introductory section, DP is not a theory and it is not a discipline. It is not a theory because it does not propose an overall explanatory account. It is not a discipline because it does not refer to a definable body of knowledge, and it does not involve a single profession. Rather, it constitutes a conceptualization that leads to crucial questions on continuities and discontinuities and which is dedicated to the discovery of mediating mechanisms. Much has been achieved but many challenges remain.

DP research employs both categorical and dimensional approaches, although the latter predominate. There are those who would like to argue that the distinction between categorical and dimensional approaches is something that can be settled by the appropriate statistical analyses. However, nearly half a century ago Zubin (1967) pointed out that nearly all categories can be transformed into dimensions, and conversely nearly all dimensions can be translated into categories. The question is not what is the reality in nature but, rather which is more useful in relation to the particular questions being addressed. It will be appreciated that, although this article has been solely concerned with mental disorders, much the same issues arise in relation to internal medicine when dealing with multifactorial disorders such as coronary artery disease, hypertension, and atopic disease. Each of these gives rise to similar questions about continuities and discontinuities and in many instances both are found. For example, hypertension is obviously a dimensional feature but once the changes that are characterized by the label of malignant hypertension arise, the course becomes a different one. DP is concerned with psychopathology, but the principles are likely to apply across medicine and across biology.

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