Seventh Edition

Rutter's Child and Adolescent Psychiatry and Psychology

Edited by

Anita Thapar • Daniel S. Pine • Samuele Cortese • Cathy Creswell

Tamsin J. Ford • James F. Leckman • Argyris Stringaris

WILEY Blackwell

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Foreword

Mike's mission to research child development began in the early 1960s. He commenced this journey with immense excitement, energy, and determination. This never wavered throughout his long career. He was greatly influenced by his mentor, Aubrey Lewis, the first child psychiatrist in the UK, who taught Mike to "think deeply, challenge accepted wisdom and never accept anything without careful scrutiny of the available empirical evidence." These words of wisdom guided Mike in all his endeavours and can clearly be seen in the rigour he applied to all the editions of the textbook. In addition, Mike always approached his work with humanity and kindness. Mike was committed to ensuring that younger generations of child psychiatrists and child psychologists continued to take the discipline forward and it was with this in mind that the 1st edition of the textbook was born. I know that Mike would be very proud of this 7th edition and delighted that his mission to bring together and create, with worldwide researchers and clinicians, an extraordinary body of knowledge and understanding of child psychiatry, which continues beyond his lifetime. I am so pleased and proud that his work has helped others to make a lasting and profound impact on the mental health of children around the world.

Marjorie Rutter

Preface

This textbook was the brainchild of Mike Rutter, the forefather of modern child and adolescent psychiatry who died in 2021, aged 88 years. The 1st edition was published in 1976, and subsequent editions were led by Mike, until we were entrusted with the 6th edition. However, Mike remained much involved in supporting us as we prepared the 6th edition; with his death, he asked that we continue the tradition of the textbook. We have felt enormously honored and privileged to take the volume forward with the 7th edition. The textbook is renowned for the high standards created by Mike, and we have retained many of the approaches he initiated in the hope of sustaining this tradition. We insisted that chapters continue to be written by the field's most brilliant minds with a conceptual focus and a skilled appraisal of the most current scientific evidence. For clinical chapters, these experts viewed their material through the lens of clinical wisdom.

To achieve excellence, Mike initiated a review process that would have been envied by the editors of many peer-reviewed journals. We have retained the process for editing chapters in this 7th edition. This has included a review of chapter outlines, followed by two stages of critical review for full chapters by the editorial team. Naturally, this has entailed an enormous amount of work for the editorial team and authors. We have been extremely fortunate in having such a wonderfully enthusiastic, kind, and responsive editorial team: Samuele Cortese, Cathy Creswell, Tamsin Ford, James Leckman, Argyris Stringaris. This has made a task that could have been arduous one to relish. Accordingly, both of us have greatly enjoyed working with the team. What has been striking to us is not only the extremely high quality of author contributions but also the willingness of senior researchers and clinicians to take on editor peer reviews and repeatedly revise their chapters. We are very grateful for their contributions and their graciousness in response to our repeated requests for different material and changes when there are so many demands on people's time.

We have also introduced a number of changes. The title of the textbook now has been changed to *Rutter's Child and Adolescent Psychiatry and Psychology* to reflect the readership and authorship of the book. This honors the multidimensional nature of our evolving field, where many professionals, including psychologists, have been instrumental for our growth. Mike would have been immensely pleased. Other changes were inspired by two notable issues: growing use of digital media, as well as increasing reliance on the textbook for teaching and training materials. For these reasons, authors provided short video clips to give a taste of their chapter, as well as slides for teaching and training. For this extra material, we did not insist on a standard approach so readers can expect much variation in author styles. Naturally, this represented even more work for the authors. Once again, we are very grateful and hope readers share our enthusiasm.

Other novel features of the edition mirror changes in science and the needs of children around the world. It contains several new chapters, including one on public health that covers global threats including the COVID-19 pandemic, wars, and natural disasters. We are also delighted to include a chapter on low-resource settings that is of relevance to clinicians in high- as well as low-income countries. Such material is vital since most of the world's children, young people, and their families live in resource-poor settings. Other new topics include: diversity in relation to gender and sexual orientation, as well as a chapter on digital technology, given the growing interest in digital approaches to assessment and intervention. Finally, our "Clinical Syndromes" section has changed with a new chapter, on body dysmorphic disorder, and new clinical highlights. We realize that some readers might peruse the textbook while working in the clinic. Hence, new highlights appear as assessment and treatment "at a glance boxes" to facilitate ready access to this information.

One of us (DSP) was supported by the Intramural Research Program of the National Institute of Mental Health to undertake editorial duties, and the work for this book (AT) was supported by the Wolfson Foundation.

Our final thanks are to Caroline Warren at Cardiff University who played the key role in coordinating much of the textbook process. It has taken us three years of weekly meetings and heavy reliance on Caroline's truly unique skill and commitment. Delivering a textbook of this complexity requires monumental organization and planning; all the editors are grateful for her hard work, perseverance, and stellar administrative and organizational support.

Anita Thapar and Daniel S. Pine

About the Companion Website

This book is accompanied by a companion website:

www.wiley.com/go/thapar/rutterspsych7

This website includes:

- Videos
- PowerPoint slides

Scan this QR code to visit the companion website:

PART I

Conceptual issues and research approaches

A: Developmental psychopathology

CHAPTER 1

Development and psychopathology: a life course perspective

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Why is a life course perspective to mental health important?

A developmental perspective is essential for understanding mental health at different stages of the life course. Childhood and adolescence are critical life stages where mental health problems can have profound impacts on young people's healthy development, including their education, relationships, and developing self-concept. Evidence shows that mental health problems in children and young people are common. More than 1 in 10 children and young people have a diagnosable condition at any given time (Sadler *et al*., 2018).

Psychiatric conditions in childhood and adolescence often continue or recur across adult life, and they may also signal the emergence of new or additional forms of psychopathology. Indeed, an earlier onset often heralds a poorer long-term prognosis (de Girolamo *et al*., 2012). Conversely, the majority of adult mental health problems have their origins earlier in life; prospective cohorts indicate that more than 50% of young adults with a psychiatric condition first met criteria for a psychiatric diagnosis before age 15 (Kim-Cohen *et al*., 2003), while global epidemiological evidence indicates that the peak age of onset of most mental disorders ranges between 5 and 21 years (Solmi *et al*., 2022).

At the same time, *longitudinal research* shows that recovery and improvement of childhood psychopathology are commonplace. Evidence suggests that around half of children and adolescents with a psychiatric condition experience no psychiatric disorder or major functional impairment in young adulthood (Costello & Maughan, 2015). A question of particular concern for young people and their families is what can be done to improve outcomes. A developmental perspective is essential for understanding the mechanisms that underlie *continuities and discontinuities* in psychopathology. Understanding modifiable risk and protective factors is a necessary first step for developing more effective prevention and early intervention. *Developmental transitions*, such as starting or changing school, puberty, or entering adulthood, are of particular interest, given that these are times of significant challenge as well as potential opportunities for intervention. This chapter sets out key principles that underpin theory and research of developmental psychopathology, together with examples that highlight the utility of a developmental approach. The chapter is structured as follows: (i) introduction to key concepts; (ii) longitudinal methods for studying developmental change; (iii) five brief snapshots to highlight developmental understanding for anxiety, depression, neurodevelopmental conditions, psychosis, and behavior problems; (iv) types of mechanisms that account for developmental continuity and discontinuity; and (v) early adversity, adverse outcomes, and resilience.

What is developmental psychopathology?

Developmental psychopathology is a scientific approach that focuses on the development of the person over time and aims to understand the processes underlying typical and atypical development including reasons for continuity and discontinuity in patterns of maladaptive thoughts, feelings, and behavior. There is a recognition that particular emotions and behaviors may be normative at certain stages of development (e.g., temper tantrums in toddlers, crying in infants) or in some situations (anxiety and fear in traumatic or stressful situations). However, where such symptoms are disproportionate, sustained over time, and out-of-keeping with children's developmental stage then these can impact on functioning and healthy development and thus represent psychopathology.

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Companion website: www.wiley.com/go/thapar/rutterspsych7

Developmental psychopathology considers adaptation and maladaptation to be the product of a series of multifaceted influences involving genes and biological factors, as well as both current and earlier environmental experiences. These *multifactorial influences* act together in complex ways, and many causal processes are thought to act as a series of chain reactions operating over time. Development is therefore viewed as a dynamic interplay between inherited/biological factors and environmental context with the person playing an active role in this process (Rutter & Sroufe, 2000; Figure 1.1). Longitudinal and genetically sensitive study designs have highlighted the active role of the person in development by illustrating that characteristics of the person, including their inherited tendencies, shape exposure to particular types of experience, which then impact on developmental outcomes. For example, antisocial behavior is moderately heritable (Burt, 2022) and children with antisocial behavior frequently evoke hostile reactions from other people with whom they interact, with these hostile interpersonal exchanges serving to further exacerbate those behaviors in the child (Burt, 2022). These types of "person effects" on environmental circumstances also apply to individual differences in personality; for example, a child concentrating on an academic task may elicit positive responses from adults (e.g., teachers, parents) that sustain that behavior (Caspi *et al.*, 2005). In summary, developmental psychopathology views development as the outcome of multiple factors operating over time and considers the person an active participant in this process.

Individuals add meaning to their experiences by cognitively and affectively processing them, which shapes how individuals respond to their experiences. For instance, children with a tendency toward antisocial behavior are more likely to attribute hostile intent to others, which over time can lead to the further development of antisocial behavior (Dodge, 2006). Another example relates to the development of working models of early attachment relationships that are posited to act as a template for later relationships and are "carried forward" into later development, via cognitive–affective representations (Waters & Waters, 2006).

Genetic and biological influences on development and sensitive periods

Many aspects of development are influenced by genetic and biological factors. Infancy is a period of profound cognitive and temperamental development and much of this development is driven by brain development and *maturational processes* that are under genetic control. Longitudinal studies over childhood, adolescence, and adult life show normative brain anatomical changes. These include increases in white matter volumes (myelinated axons) during childhood and adolescence as well as inverted U-shaped developmental changes of gray matter volumes (cell bodies of neurons) that are brain region specific (Lenroot & Giedd, 2006; Bethlehem *et al.*, 2022). Gray matter maturation occurs first in brain areas that support the primary functions of motor and sensory systems with association areas that integrate primary functions maturing later. These maturational changes are protracted with some changes, such as the remodeling of gray and white matter, continuing well into adult life (into the 30s). Other earlier maturational changes include the proliferation and organization of neurons and synapses during early prenatal life (Lenroot & Giedd, 2006).

Twin studies illustrate genetic influences on indices of white and gray matter volume and the stability of these measures in longitudinal studies during childhood and adolescence (Maggioni *et al.*, 2020). However, as with other traits, these

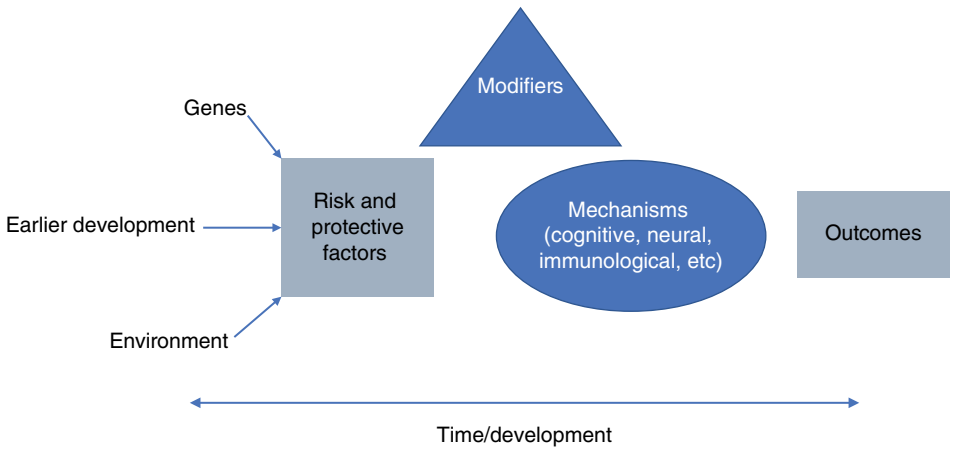

Figure 1.1 Development of the person in a multifactorial system. The time/development axis is double arrowed to represent the possibility of bidirectional influences where, for instance, one developmental outcome may influence factors, mechanisms, and/or modifiers at a later developmental point. Single arrows on risk and protective factors indicate a range of types of influence. *Source:* Adapted from Rutter & Sroufe (2000).

studies also indicate variability in changes of brain morphology over time with heritability measures changing depending on the brain region studied. This is consistent with longitudinal twin studies for a range of abilities and traits which highlight that new genetic influences can emerge at certain developmental stages—so called "genetic innovation"—and that the effect of earlier genetic influences can diminish over time; known as "genetic attenuation" (Bergen *et al.*, 2007). Sroufe *et al.* (2005) highlight three principles of development: (i) that the "organism develops as a whole;" (ii) that development is "characterized by emerging complexity;" and (iii) that differentiation and refinement can only operate on previous structures. The last principle highlights the fact that early damage can have profound effects on developmental outcomes. Examples of this are the *teratogenic effects* of thalidomide, rubella, high levels of alcohol, and Zika virus infection on the developing fetus (Thapar & Rutter, 2009; Rasmussen *et al.*, 2016). This principle of development also suggests the importance of the timing of certain exposures and that certain periods of development, often in early life, can act as *sensitive periods* of development where certain inputs are expected from the environment for normative development to occur (see the penultimate section on early adversity later in this chapter).

Important features of a life course approach

A fundamental aspect of developmental psychopathology is taking a *life course approach*. The central developmental challenges for an individual will include the primary concerns of the developing person or the most pressing capacities to be acquired, and these will vary across different developmental periods. For example, during the first year of life, establishing an effective attachment relationship with a caregiver may be a central concern whereas in early adult life this may be establishing a meaningful romantic relationship.

Several important features of the developmental psychopathology perspective emerge because of the life course approach. These include the concepts of *developmental snares*, *resilience* and/or *recovery*, and *turning points*. Developmental snares are events or experiences that "trap" people in particular circumstances which then further disadvantage individuals. As one example, Moffitt *et al.* (2011) examined self-control (the ability to control immediate urges or impulses in preference for longer-term goals) in a longitudinal cohort study. Children with lower self-control tended to do less well in terms of social and occupational outcomes in adult life compared to children higher on self-control. Although there was a main effect of childhood self-control on adult outcomes, the authors found that part of the effect of low childhood self-control on adult outcomes was due to children "making mistakes" as teenagers (e.g., unplanned pregnancy, leaving school early) which then impacted on adult outcomes such as earnings, health, and criminal convictions in adult life (Moffitt *et al.*, 2011). Children with the lowest self-control experienced more of these snares and were more likely than children with better self-control to show poor adult outcomes.

Resilience is often defined as a better-than-expected outcome following experience of adversity, and *recovery* as a period of adaptive development following a period of maladaptive development. The use of a life course perspective therefore allows resilience to be viewed as a developmental process and implies that development is not static, and that change is possible. Indeed, change is always possible in a developmental psychopathology perspective but becomes more or less likely depending on a range of factors including the characteristics of the individual, their environment, inherited and biological liabilities, and earlier experiences (Rutter & Sroufe, 2000). Concepts related to recovery and resilience are those of transition and turning points. Periods of transition can act as *turning points* (e.g., starting a new school, parenthood, marriage) by involving heightened susceptibility to either adaptive or maladaptive changes. For instance, in examining continuities between childhood and adult life, Rutter (1989) describes instances where negotiating transition points effectively can set in motion chains of events that have positive effects over extended periods of time. One classic example involved examining the adult outcomes of women who were raised in institutions as children following breakdown of parenting in the family (Rutter, 1989). A series of interrelated events were identified that were associated with good social functioning and parenting in adult life. Positive school experience, planning for work and marriage, marriage for positive reasons, and marital support were protective events associated with positive outcomes in this vulnerable group of young women who had experienced early adversity. Indeed, longitudinal research highlights the importance of planful behavior and problem-solving as well as interpersonal support in predicting positive outcomes following adversity (Rutter, 1989). More detailed examination, utilizing longitudinal and behavior genetic designs, supports the view that turning points such as marriage may have a causal effect on trajectories of psychopathology but that the nature of any protective (or indeed risk) effects is not uniform, with variation according to the characteristics of a partner (Kendler *et al.*, 2017). The normative educational transition from primary/elementary to secondary/high school can also act as a turning point. There are opportunities for some children to form new and more supportive friendships (Shell *et al.*, 2014; Ng-Knight *et al.*, 2019). Evidence that transition periods involve heightened susceptibility to change has led to suggestions that such periods are potentially useful points to introduce intervention programs (Vitaro & Tremblay, 2008).

Methods that are useful for the study of developmental psychopathology

Longitudinal studies with repeated assessments of psychopathology are critical for examining factors that shape children's development, as insights about developmental patterns and

underpinning etiological processes are often misleading when based on cross-sectional data (Kraemer *et al.*, 2000). Longitudinal studies differ in the extent to which they aim to provide a picture for the whole population or for particular groups of interest, the length of follow-up, and number of assessments over the follow-up period, as well as the breadth and depth of assessments of psychopathology and associated aspects of children's development, biology, and social environment.

Population birth cohorts, in principle, provide a picture of normative development unaffected by clinical referral, diagnostic, or other selection biases. With assessments often starting at or before birth, population cohorts provide opportunities to observe how psychopathology first develops. They help identify groups of children most at risk of developing mental health difficulties and developmental periods when risk is most strongly observed. Comparisons *between* population cohorts can address questions related to population differences in mental health. This includes testing whether and why rates of psychopathology have changed over time or differ between countries, and whether outcomes for children with mental health problems have improved or worsened (Collishaw, 2015; Sellers *et al.*, 2019).

There are challenges and limitations to population cohort studies that are important to consider. Longitudinal population cohorts typically experience sample drop-out which can introduce bias due to factors related to the probability of participants staying in the study. A further challenge is that population cohorts are costly and often broad-ranging in focus, limiting detailed phenotyping and density of repeated measurement. Guarding against the exclusion of high-risk and typically disadvantaged groups is a particular concern. Children from ethnic minority backgrounds, with disabilities, who are LGBTQ+, and those educated in non-mainstream school settings are very often underrepresented in population cohorts. Where they are included, it is often not possible to draw reliable conclusions because sample sizes are small. New cohorts have begun to adapt their methods to overcome some of these difficulties; for example, by oversampling families from socioeconomically disadvantaged or ethnic minority groups and by ensuring that a diverse range of young people's voices is heard when designing study assessments. Inevitably, each population cohort represents a picture of life course development that is particular to one generation of children.

An important complement to population cohorts are longitudinal studies of patient samples or high-risk groups that allow for more focused investigation of specific groups of vulnerable children. An example of this approach is the prospective investigation of children at high familial risk where one aim is to better understand the emergence of conditions such as depression, bipolar disorder, or psychosis (Rice *et al.*, 2017). Other examples include in-depth clinical studies of children with rare conditions such as childhood-onset schizophrenia (Driver *et al.*, 2013), or children with genetic conditions such as carriers of copy number variations that confer increased susceptibility to a range of mental health problems (Chawner *et al.*, 2019).

Developmental "catch-up" studies are sometimes a more pragmatic way to investigate life course outcomes for children in high-risk groups. Examples include a study of maltreatment which used record-based data to identify a sample with documented childhood maltreatment together with a matched control group to investigate adult psychosocial outcomes (Widom *et al.*, 2007) and a very long-term follow-up (up to age 70 years) of a juvenile delinquency sample first studied in the 1940s (Laub & Sampson, 2006). Registry studies which include anonymous data from routine social, health, and educational records are another way in which historical data on certain exposures (e.g., death of a parent, perinatal complications, potential maltreatment) can be used to generate insights about a life course perspective.

Prospective studies provide contemporaneous assessments of experiences at each time point in a longitudinal study, whereas *retrospective assessment* refers to the measurement of experiences that occurred in the past. Prospective and retrospective studies present distinct challenges in study design, implementation, and interpretation. It is now clear that there is often little agreement between prospective and retrospective assessments of experiences such as childhood maltreatment, and prospective and retrospective data can lead to different conclusions, including the estimation of lifetime prevalence of disorder (Reuben *et al.*, 2016).

Prospective study designs are often essential for assessing the temporal ordering of events, and to assess developmental change in psychopathology and associated risk and prospective factors. However, retrospective data can provide an important complement, particularly in circumstances where there are ethical or practical challenges in assessing aspects of children's lives contemporaneously, or where it is likely that informants might underreport certain experiences such as child maltreatment. There is very little evidence that participants in research studies fabricate or exaggerate problematic childhood experiences. Instead, an important bias can be that adults who are functioning well often underreport adverse childhood experiences (Hardt & Rutter, 2004).

A critical consideration in observational studies of developmental psychopathology is the extent to which it is possible to make *causal inferences* (Chapter 13). It is always important to consider alternative explanations for findings, including the possibilities of reverse causation, measured or unmeasured confounding, and collider bias. *Triangulation* of findings using study designs and statistical approaches with different strengths, limitations, and underlying assumptions is strongly advised (Hammerton & Munafò, 2021).

Thinking to the future, there are important decisions for scientists and research funders in how to prioritize the development of research infrastructure and how to maximize the utility of available data for scientific and clinical discovery. There is a need to overcome common limitations of traditional research designs. For example, a recent study demonstrates how the utility of population-wide, cross-sectional school questionnaire

data can be enhanced by linkage with national patient record data, enabling longitudinal analysis of clinically relevant outcomes at scale. The study showed that bullying and loneliness are common experiences and strongly predict subsequent clinical presentation for self-harm (John *et al*., 2023).

Patterns of continuity across childhood, adolescence, and adulthood

Developmental life course studies involve tracking individuals over time and are crucial for understanding influences on continuity and discontinuity of psychopathology, as well as for describing the typical developmental sequences of different types of psychiatric disorder. Patterns of continuity can be described as *homotypic* (where the same disorder persists over different developmental periods) or *heterotypic* (where an earlier disorder is followed by a disorder of a different kind). Such patterns of continuity can reflect continuation of the same underlying illness/maladaptation process, one disorder acting as a risk factor for another disorder, or that manifestation of the same underlying illness/maladaptation looks different at different developmental stages. Sankey diagrams are a data visualization technique to show flow (e.g., of energy, material, individuals) through a series of stages, and these have been applied in psychopathology research to visualize patterns of continuity and discontinuity (Caspi *et al*., 2020) or co-occurrence of disorders over time (Plana-Ripoll *et al*., 2019). In Figure 1.2, we present a *Sankey diagram* based on data from the Early Prediction of Adolescent Depression study (Rice *et al*., 2017) which is tracking the development of psychopathology in high-risk offspring of depressed parents. The figure illustrates patterns of continuity and discontinuity of disorder over time in that sample. This indicates a range of developmental pathways characterized by homotypic and heterotypic continuities, emergence of new difficulties, and recovery from earlier difficulties. Indeed, life course studies can identify influences on the 'persistence of' compared to 'recovery from' mental health disorders over time, whether etiological influences vary as a function of age of onset of a disorder, the childhood antecedents of adult-onset conditions, and the broader psychosocial and functional outcomes of psychopathology. Developmental life course studies have also highlighted the phenomena of *equifinality*, which refers to the fact that different sets of risk and protective factors can lead to the same outcome in different individuals, and *multifinality*, which refers to the fact that the same sets of risk and protective factors can lead to different outcomes in different individuals (Cicchetti & Rogosch, 1996; and see Figure 1.3).

In the next sections, we give a brief overview of studies focusing on different types of psychopathology to illustrate how a

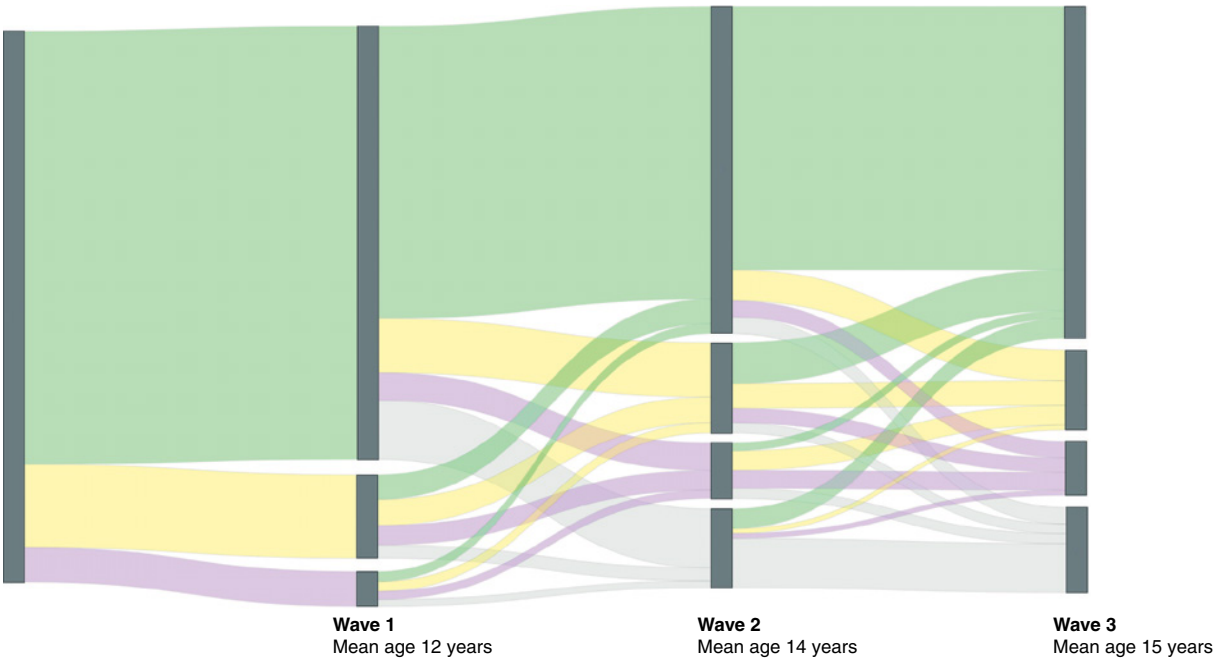

Figure 1.2 Homotypic and heterotypic continuity over time. Sankey diagram illustrating rates of anxiety (yellow), depressive (blue), behavioral (red), ADHD (purple), and no (green) disorders over a four-year follow-up period including three assessment waves in the Early Prediction of Adolescent Depression study (Rice et al., 2017). Homotypic continuity is demonstrated by the same-colored band over assessment wave (e.g., yellow to yellow shows continuity in anxiety; red to red shows continuity in behavioral disorders). Heterotypic continuity is demonstrated by changes in colors over assessment waves (e.g., red to blue shows behavioral to depression). Recovery is shown by offset of disorder (e.g., yellow to green shows recovery from earlier anxiety). Gray denotes missing data. *Source:* Diagram created by Dr Vicky Powell using information from the Early Prediction of Adolescent Depression study via a Sankey tool on this website http://sankey-diagram-generator.acquireprocure.com/.

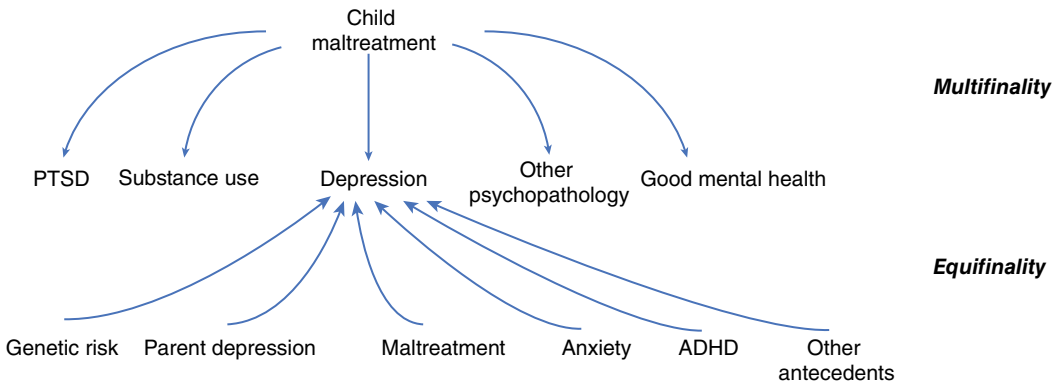

Figure 1.3 Multifinality and equifinality. Multifinality: a variety of endpoints are possible following the same beginning. Equifinality: the same endpoint can be reached through a variety of paths and from a variety of beginnings. *Source:* Adapted from Cicchetti & Rogosch (1996).

developmental approach can be helpful for understanding mental health, and to draw attention to some of the paradigmatic issues described earlier, as well as to commonalities and distinctions between different forms of psychopathology in terms of their developmental picture—age at onset, developmental course, and predictors of persistence and long-term outcomes. It is worth bearing in mind that psychopathology exists on a spectrum. While there is evidence for a dimensional approach for all of the disorders we consider, we focus primarily on disorder or clinically elevated symptoms in the examples provided.

Anxiety

There is marked heterogeneity in age of onset between different forms of anxiety disorder (Chapter 59). Separation anxiety and specific phobias typically begin in childhood, social anxiety has a median age of onset in early adolescence, while agoraphobia, panic disorder, and generalized anxiety disorder typically emerge in late adolescence or young adulthood (Beesdo-Baum & Knappe, 2012; Solmi *et al.*, 2022). It is noteworthy, that later onset anxiety disorders reflect more generalized forms of anxiety than those with a typical childhood onset which are characterized by circumscribed fears. It is important, however, not to view anxiety disorders in isolation from one another. Prospective longitudinal studies highlight that anxiety disorders in childhood or adolescence strongly predict anxiety disorders in adulthood. There is little specificity in continuities for particular anxiety disorders (with the exception of specific phobia), suggesting that continuities in anxiety reflect developmental change in the manifestation of anxiety (Gregory *et al.*, 2007).

Child and adolescent anxiety in the general population is associated with a broad range of problematic life course outcomes, notably depression, as well higher risk of substance use, behavioral problems, disrupted educational and occupational trajectories, impaired relationships, and poor physical health (Copeland *et al.*, 2014). Adolescent anxiety also contributes to long-term risk of suicide ideation and attempts, over and above other psychopathology such as depression (Pickles *et al.*, 2010). Anxiety may also be one developmental precursor of severe adult psychiatric disorders such as major depression, bipolar disorder, or schizophrenia, at least in children at already high familial risk of these conditions (Sandstrom *et al.*, 2019). However, the vast majority of children with anxiety do not go on to experience severe mental illness as adults.

There is heterogeneity in the persistence of anxiety and in broader life course outcomes. Anxiety is moderately heritable, and although current evidence is limited as to the role of genetic influences on homotypic and heterotypic continuities in anxiety, evidence does suggest that there are both age-specific genetic influences on anxiety, as well as common genetic factors predicting continuity in anxiety over time (Waszczuk *et al.*, 2016). Follow-up studies of children and adolescents with anxiety provide consistent evidence that persistence of anxiety across the life course is predicted by severity and duration of avoidance behaviors, degree of functional impairment, cognitive factors such as catastrophizing, and higher behavioral inhibition (Beesdo-Baum *et al.*, 2012; Hovenkamp-Hermelink *et al.*, 2021). Behavioral inhibition and avoidance likely shape children's environments, influencing parenting style and contributing to social risk exposures such as peer exclusion and victimization (Degnan *et al.*, 2010).

Finally, considerable progress has been made in understanding cognitive and neurobiological features of anxiety (Chapters 12 and 59; LeDoux & Pine, 2016). Developmental research holds the promise of elucidating their role as vulnerabilities, manifestations, or consequences of anxiety.

Depression

Several lines of evidence including from epidemiological, twin, and treatment studies suggest developmental differences in the etiology of depressive symptoms and depressive disorder between childhood and adolescence (Chapter 63). Indeed, twin studies suggest that depressive symptoms are moderately heritable in adolescence but that the influence of genetic factors is negligible in childhood. Thus, evidence for genetic innovation where "new" genetic influences become active during adolescence has been identified in longitudinal twin studies (Kendler *et al.*, 2008; Lau & Eley, 2010). It has been suggested that genetic innovation over the transition from childhood to adolescence may be partly due to genetic influence on hormonal changes associated with puberty, structural brain changes, and increased gene–environment correlation associated with increasing independence in selecting environments as children grow up. The correlation between changes in hormones associated with puberty leading to changes in the social environment has been highlighted as involved in the increase in depressive symptomatology seen in females around adolescence (Ge *et al.*, 1996). Thus, girls who began puberty earlier experienced greater psychological distress than on-time or later maturing peers and were more vulnerable to deviant peer pressure and paternal hostility. Several studies also highlight that females may experience increases in psychosocial stressors during adolescence, particularly interpersonal stressors, and may also be particularly sensitive to negative affect following these types of stressors (Oldehinkel & Bouma, 2011; Ordaz & Luna, 2012). Long-term studies highlight that the risk effects of early puberty for females are mostly time-limited and tend to dissipate by early adult life (Copeland *et al.*, 2010). It is also noteworthy that exposure to social stressors is correlated with genetic liability for depression (Rice *et al.*, 2003). This means that those with higher genetic liability are also more likely to be exposed to stressors.

Depression shows strong associations with anxiety (Copeland *et al.*, 2009). The finding that anxiety typically precedes depression in longitudinal studies is also consistent with the possibility that some forms of anxiety may be early developmental manifestations of depression (Rutter *et al.*, 2006). Consistent with the suggestion of common etiological influences for anxiety and depression, evidence from genetically sensitive family designs including twin studies shows considerable genetic overlap between depression and anxiety, particularly with generalized anxiety disorder (Silberg *et al.*, 2001; Kendler *et al.*, 2022). Thus, it is possible that these common genetic influences are expressed phenotypically in different ways at different stages of development.

An important feature of depression is whether it persists over time. Studies of adults (Eaton *et al.*, 2008) and adolescents (Patton *et al.*, 2014) in community studies suggest that one-off episodes of depressive disorder that do not recur are relatively common. Nevertheless, clinical follow-up studies of individuals treated for depressive disorder during childhood and adolescence report high recurrence rates of 70% within five years (Harrington *et al.*, 1997). This highlights depression as a condition that can desist or persist although it is worth noting that subthreshold symptomatology can be present between full-blown episodes (Judd *et al.*, 1998). When depression in young people does persist, it is associated with particularly poor mental health, educational, and functional outcomes (Weavers *et al.*, 2021). Systematic reviews indicate that approximately 5–12% of young people in the general population show persistently elevated depressive symptom trajectories (Musliner *et al.*, 2016; Schubert *et al.*, 2017). Persistent trajectories have been associated with an earlier age of onset, inherited vulnerabilities, and exposure to a range of stressors both chronic and acute, as well as female sex, minority status, and lower socioeconomic status (Musliner *et al.*, 2016; Schubert *et al.*, 2017; Rice *et al.*, 2019; Weavers *et al.*, 2021).

Neurodevelopmental disorders (NDDs)

The developmental course of NDDs (Chapters 51–57) can be distinguished from other forms of psychopathology in that most have an early onset of symptoms and demonstrate a steady stable course of symptoms. This points to the predominant importance of early etiological influences, whether genetic or environmental in nature (Nigg *et al.*, 2020).

The course of most NDDs is marked by apparent improvement in core symptoms, and rates of NDDs are higher in childhood than in adulthood. Estimates of diagnostic persistence vary considerably according to methods of assessment and whether clinical or population cohorts are considered (Thapar *et al.*, 2017). Specific symptoms also vary in the extent to which they show developmental change; for example, for children with attention deficit hyperactivity disorder (ADHD) symptoms of hyperactivity show greater attenuation with age than symptoms of inattention (Pingault *et al.*, 2015). Improvement in neurodevelopmental symptoms might reflect neurocognitive maturation, the development of adaptive mechanisms (or adaptation of environmental contexts to children's needs), and for some, the beneficial effects of treatment. Nevertheless, continuities in neurodevelopmental symptoms are observed from childhood into adulthood (Faraone *et al.*, 2006; Maughan *et al.*, 2009), and as discussed later in the chapter, impacts on psychosocial functioning and health persist across the life course. It is clear, that for most individuals with an NDD, this is not a childhood-limited condition that will naturally resolve with time.

Recent developmental research also demonstrates the possibility of late-emerging neurodevelopmental problems. Epidemiological studies with repeated assessments across development have shown that for some individuals symptoms of ADHD or autism spectrum disorder (ASD) are first reported in adolescence or in adulthood (Moffitt *et al.*, 2015; Caye *et al.*, 2016). It remains unclear how far the emergence of "late-onset" neurodevelopmental conditions reflects measurement artifacts such as variability in informant reports, the influence

of adolescent- or adult-onset conditions such as depression, or "masking" of childhood symptoms due to earlier protective influences, nor is it clear whether late-onset neurodevelopmental problems are typical or atypical in terms of etiology (Livingston *et al*., 2019; Riglin *et al*., 2022).

Individuals with neurodevelopmental conditions are at increased risk of experiencing peer victimization, loneliness, and educational underachievement. They are also at substantially heightened risk of other mental health problems including anxiety, depression, substance-use disorders, conduct problems, and self-harm (Caye *et al*., 2016), and risk of death by young adulthood (Dalsgaard *et al*., 2015). Longer-term impacts on health are less well-established, and different mechanisms may account for morbidity associated with neurodevelopmental conditions in early and later life (e.g., accidents vs. cardiovascular health risks).

Psychosis

A developmental approach has been a crucial part of theories about the genesis of schizophrenia (Howes & Murray, 2014; see also Chapter 56). Schizophrenia is a highly heritable condition and hundreds of genetic variants have been identified as showing robust associations with schizophrenia (Trubetskoy *et al*., 2022). Nonetheless, it has long been hypothesized that schizophrenia involves an important neurodevelopmental component (Weinberger, 1987). This is based on the following observations: that early prenatal and perinatal complications are associated with an increased risk for schizophrenia, that individuals who develop schizophrenia have elevated rates of neurological, cognitive, and social problems as children, and that structural brain differences can be observed when schizophrenia onsets in the absence of any evidence of neurodegenerative abnormalities in post-mortem studies (Howes & Murray, 2014). A prominent view of the development of schizophrenia is that multiple "hits" are required to generate the condition and that these involve genetic and environmental insults experienced throughout development (Davis *et al*., 2016; Owen *et al*., 2016). The role of a range of early adverse experiences has been highlighted including prenatal infection, perinatal complications, childhood adversity, and stress associated with migration and urban living (Howes & Murray, 2014). However, there is also strong evidence for disrupted brain development that is apparent early in life, and recent evidence highlights reduced neuronal connectivity in frontal cortex and hippocampal brain areas which begins *in utero* and continues through development (Hall & Bray, 2022). It is understood that such early neuronal developmental aberrations will involve genetic influences (Howes & Murray, 2014). It has been suggested that later maturational events in brain development such as synaptic pruning or environmental events or insults such as trauma exposure and cannabis use can elicit psychopathology because of the effects they have on an already vulnerable neuronal/cognitive system. Thus, exposures that are experienced during adolescence or young adulthood may lead to the onset of psychotic symptomatology because the exposure means that preexisting neuronal/cognitive vulnerabilities manifest themselves. Studies of childhood trauma have indicated that this may include potentially causal associations on psychosis (Heins *et al*., 2011) as well as gene–environment correlation whereby genetic loading for schizophrenia is associated with an increased likelihood of experiencing particular traumatic events (Sallis *et al*., 2021).

Studies of subclinical psychotic symptoms illustrated that these are relatively common during adolescence (Jones *et al*., 2016). For many individuals these symptoms are transient and remit, but in others they are persistent and associated with an increased risk for psychosis and other psychiatric disorders (Catalan *et al*., 2021). Psychotic symptoms in adolescents have shown strong association with co-occurring anxiety and depression (Jones *et al*., 2016; Catalan *et al*., 2021) and an inconsistent association with genetic risk for schizophrenia (Jones *et al*., 2016).

Behavioral problems

Developmental models of antisocial behavior have played a major role in guiding policy and practice, including emphasis on early parenting interventions (Leijten *et al*., 2019). Moffitt's (1993) model of antisocial behavior distinguishes two common developmental subtypes: early-onset life course persistent and adolescent-limited antisocial behavior. Studies of children's normative development in contrast illustrate that physical aggression is common among very young children and typically reduces by school entry (Tremblay, 2010). Longitudinal and intergenerational studies suggest multiple factors act as predictors of the onset and persistence of behavioral problems, including children's callous emotional traits, harsh parenting, parental antisocial behavior, maltreatment, poverty, and deviant peer relationships (Basto-Pereira & Farrington, 2022). Research highlights the importance of person–environment interplay. For example, adoption studies point to genetically influenced traits and behaviors evoking more hostile parenting (Harold *et al*., 2017). In adolescence and young adulthood, assortative selection into more problematic peer and romantic relationships, as well as problematic substance use, act as potential "developmental snares" that reinforce antisocial behavior (Quinton *et al*., 1993; Hussong *et al*., 2004). Conversely, the transition to adulthood also offers opportunities for desistance of antisocial behavior, for example, due to changes in peer affiliations, establishing a supportive romantic relationship, or starting a new job (Laub & Sampson, 2006).

Behavioral problems are associated with a wide variety of adverse outcomes, including elevated risks of mental health problems, substance misuse, educational and occupational difficulties, and poor health (Colman *et al*., 2009), but mechanisms of risk remain to be fully clarified. One complication is that