CHAPTER 1

Development and psychopathology: a life course perspective

Stephan Collishaw and Frances Rice
Wolfson Centre for Young People's Mental Health, Division of Psychological Medicine and Clinical Neurosciences, School of Medicine, Cardiff University, UK

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.

Rutter's Child and Adolescent Psychiatry and Psychology, Seventh Edition.
Edited by Anita Thapar, Daniel S. Pine, Samuele Cortese, Cathy Creswell, Tamsin J. Ford, James F. Leckman, and Argyris Stringaris.

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

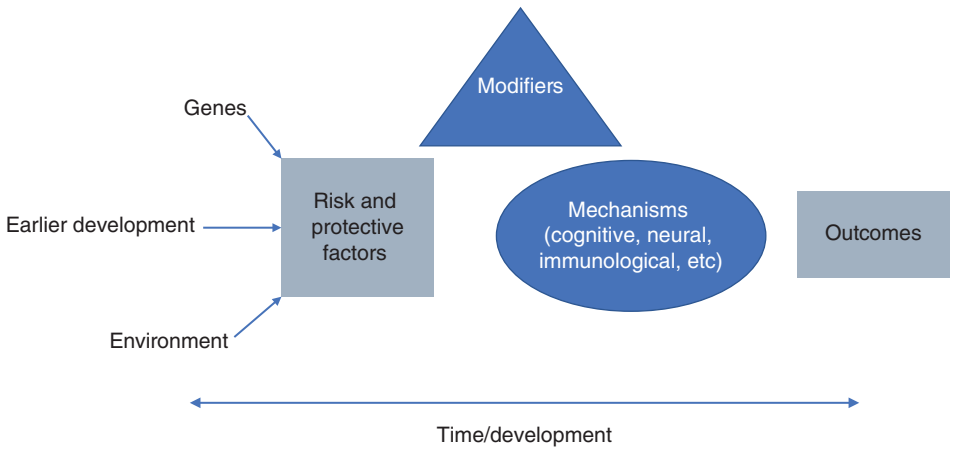

Figure 1.1 Development of the person in a multifactorial system. The time/development axis is double arrowed to represent the possibility of bidirectional influences where, for instance, one developmental outcome may influence factors, mechanisms, and/or modifiers at a later developmental point. Single 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

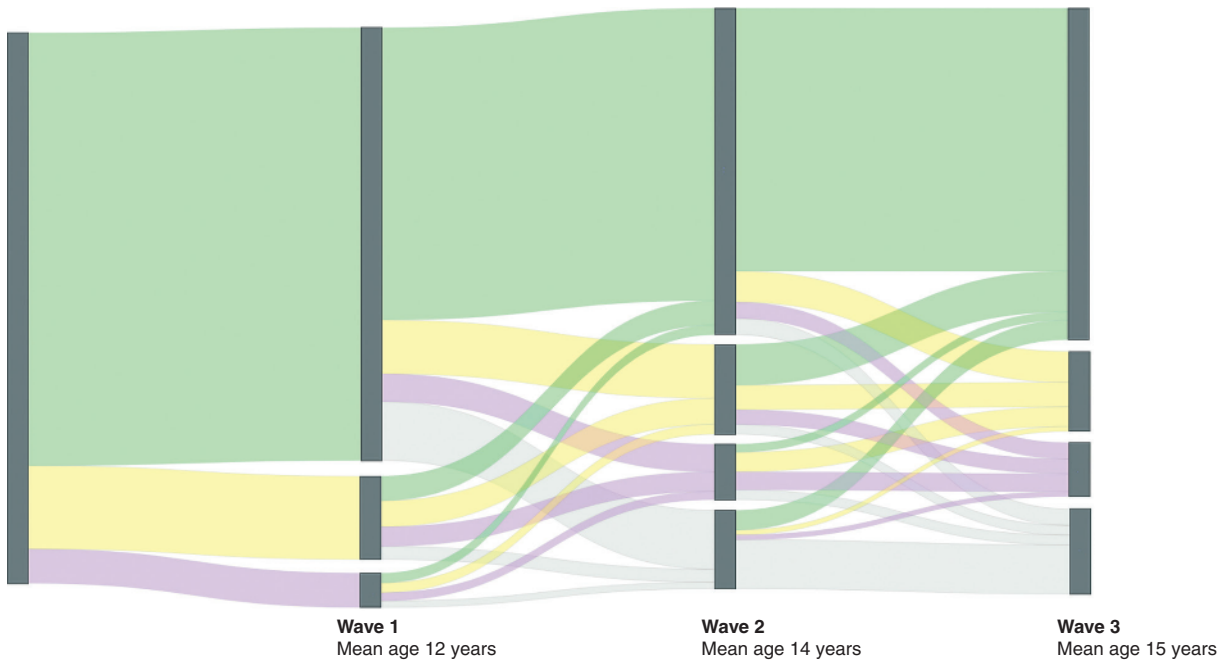

Figure 1.2 Homotypic and heterotypic continuity over time. Sankey diagram illustrating rates of anxiety (yellow), depressive (blue), behavioral (red), ADHD (purple), and no (green) disorders over a four-year follow-up period including three assessment waves in the Early Prediction of Adolescent Depression 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/.

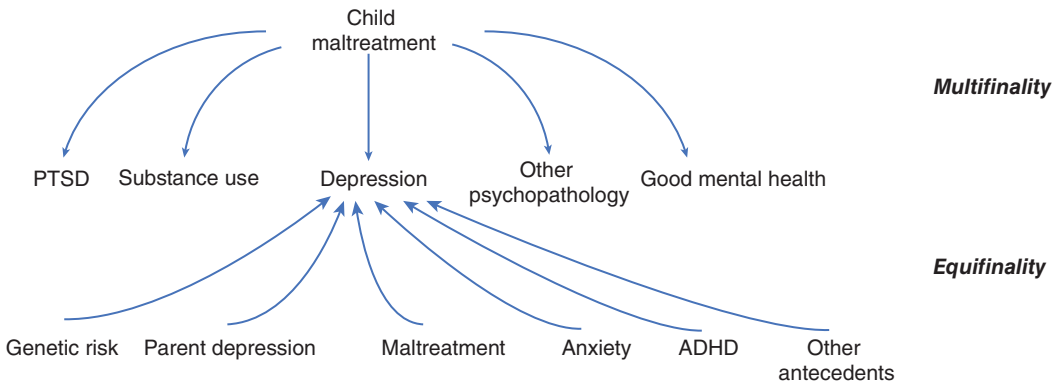

Figure 1.3 Multifinality and equifinality. Multifinality: a variety of endpoints are possible following the same beginning. Equifinality: the same endpoint can be reached through a variety of paths and from a variety of beginnings. *Source:* Adapted from Cicchetti & Rogosch (1996).

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

traditional diagnostic specifications of behavioral disorders include a broad range of symptoms with differing developmental features and implications. Conduct disorder symptoms show stronger homotypic continuity than do symptoms of oppositional defiant disorder (ODD) which more strongly predict later emotional disorders (Rowe *et al*., 2010). Research is increasingly considering individual "symptoms," for example, irritability is now viewed as an important transdiagnostic marker of mood dysregulation with links not just to behavioral disorders, but also neurodevelopmental conditions, anxiety, depression, and suicide/self-harm (Brotman *et al*., 2017).

Different types of developmental association

As highlighted by these brief snapshots, a developmental approach has been instrumental in improving our understanding of psychopathology, highlighting early life, childhood, and adolescence as important developmental periods, and drawing attention to developmental connections that very often cross traditional diagnostic boundaries (Chapter 9). Developmental studies reveal different types of longitudinal associations between variables. One useful distinction is between associations that are "distal," i.e., have remote or indirect causal influence on a disorder and those that are "proximal" which have a more direct impact on causal chains of events (Rutter, 2009). *Distal associations* are often thought to influence behavior mainly via more proximal factors. For example, there is a strong association between low socioeconomic status and psychopathology in children, but these effects appear to be driven in part by more proximal features of children's family and social contexts such as parenting (Bifulco *et al*., 2002). Understanding *proximal processes* is clearly important because they can explain how and why psychopathology arises. There has been much interest in identifying proximal explanatory mechanisms for psychopathology that transcend traditional diagnostic boundaries. Research to identify potentially *transdiagnostic mechanisms* influencing psychopathology has tended to focus on patterns of neural circuitry and cognitive–affective processing (Insel *et al*., 2010). For instance, Harvey *et al.* (2004) asked which cognitive and behavioral processes (e.g., selective attention, overgeneral memory, repetitive negative thinking) were shared across a range of disorders. A number of models including the Research Domain Criteria (RDoC; Insel *et al*., 2010) and the Hierarchical Taxonomy of Psychopathology (HiTOP; Kotov *et al*., 2017) specify transdiagnostic processes (e.g., negative valence systems, positive valence systems, cognitive systems) and transdiagnostic structures (e.g., internalizing, externalizing, thought disorder, detachment) to psychopathology. While there are strengths and weaknesses of both a disorder-oriented and a transdiagnostic approach the hope is that identifying transdiagnostic risk and protective mechanisms can improve understanding about processes, contribute to knowledge about the taxonomy of psychopathology, and help develop treatments that are effective in targeting more than one primary problem (Dalgleish *et al*., 2020).

Early life experience and long-term implications for mental health

Early life experiences, both negative and positive, are associated with potentially long-lasting consequences for development of psychopathology. Certain groups are at particularly elevated risk of developing mental health difficulties. These include children: with a parent with mental illness; exposed to parental conflict; growing up in poverty; who have experienced trauma, maltreatment, or peer victimization; and who experience discrimination due to race, religion, sexuality, or gender identity, as well as children with special educational needs, or chronic physical health conditions. Adverse childhood experiences are common, and both retrospective and prospective reports of adversity demonstrate strong associations with adult mental health (Newbury *et al*., 2018). It is also clear that adverse childhood experiences often do not occur in isolation, that the cumulative burden of adversities is an important predictor of long-term outcomes, and that both chronic stress and severe acute events can have long-lasting consequences for children's development (Lacey *et al*., 2020). Several important questions arise in the interpretation of long-term associations between early adversity and psychopathology.

First, it is important to understand whether identified childhood risk factors exert causal effects on children's outcomes (see Chapter 13). Alternative explanations such as confounding, reverse causation, and possibilities of collider bias need to be considered. A variety of research designs that approximate *"natural experiments"* can help tease apart alternative explanations (Thapar & Rutter, 2019). Examples of such designs include comparisons of children born through *in vitro* fertilization who are related or unrelated to their mothers to test the role of *in utero* exposures such as maternal smoking (Rice *et al*., 2009), *regression discontinuity* designs to examine the role of factors that are quasi-randomly assigned to children in the population such as relative age in the school year (Broughton *et al*., 2023), and the impact of *universal interventions* such as poverty reduction (Costello *et al*., 2003).

Second, it is important to understand the developmental mechanisms by which particular early-life risk factors impact on children's mental health. For example, observational studies suggest an increased risk of emotional and behavioral difficulties among children who have experienced parental separation. While parental separation is often stressful for children, of particular relevance to children's psychosocial outcomes are the proximal features of children's family relationships that impact children prior to, during, and subsequent to the break-up of a parental relationship (Harold & Sellers, 2018). There are also important unresolved questions about the psychological and

biological processes through which early adversity impacts children's development, including impacts on the set-points and functioning of neurophysiological reactivity, as well as consequences for children's development of emotion regulation, self-concept, and social information processing (Cicchetti, 2016).

Third, early-life adversities do not exert deterministic effects on psychopathology. Children exposed to even the most severe stressors show variability in outcomes, with resilience and recovery not uncommon (Rutter, 2006). Reducing risk exposure is important but not always possible. An additional priority therefore is identifying modifiable protective factors that can buffer risk and promote recovery. Multifactorial explanations for resilience are required, and research demonstrates that affective, cognitive, behavioral, social, and contextual factors act together to predict resilience in the context of adversity (Figure 1.4). An important conclusion is that specific protective factors on their own are rarely sufficient to promote resilience and recovery in the context of severe adversity. Furthermore, a developmental perspective holds that patterns of resilience will change over time, influenced by previous experiences of successfully (or unsuccessfully) negotiating similar challenges, as well as by maturation and the specific challenges facing an individual at different developmental stages. Furthermore, salient protective factors will vary between individuals and for differing developmental outcomes.

A developmental perspective is also useful for considering the importance of *timing of exposure* to stress. There are sensitive periods in development during which experience can have larger impacts on development of particular traits than during other periods. The English and Romanian Adoptees (ERA) study, for example, examined the development of children who experienced severe early institutional deprivation and were then adopted into socioeconomically advantaged families (Sonuga-Barke *et al.*, 2017). The study highlighted profound immediate impacts of early deprivation on social and cognitive development, as well as longer-term impacts on mental health. At the same time, many children showed evidence of recovery and catch up, especially those who were adopted early, suggesting that both the timing and the duration of exposure to early stress can be important.

Conclusion and priorities for the future

Developmental studies have improved our understanding of how common mental health conditions develop and progress, possible reasons for homotypic and heterotypic continuities over the life course, and of factors that promote resilience and recovery. A priority for the future is to ensure that this rich understanding of life course development can inform more effective approaches to both the prevention of mental health problems and the provision of tailored support for children with mental health and neurodevelopmental conditions as they grow up. Other priorities for the future include incorporating multiple approaches and measurements of important potential drivers of psychopathology into longitudinal studies in efficient and

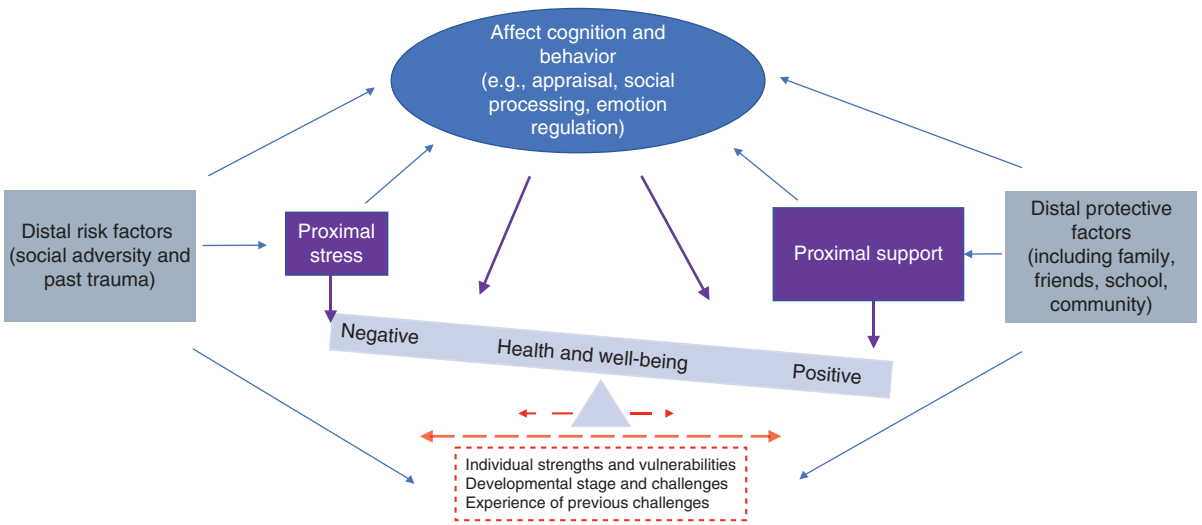

Figure 1.4 Resilience—dynamic interplay of person and context. Vulnerability and resilience in the context of stress varies according to children's underlying strengths and vulnerabilities, their developmental stage, and past (un)successful negotiation of stressful situations. In the context of greater vulnerability, greater levels of support are needed to promote resilience. The balance of proximal stressors and supports is influenced by contexts of adversity and available support systems, in interplay with children's own appraisal, emotional responses, and behaviors. *Source:* Adapted from National Scientific Council on the Developing Child (2015).

creative ways. Understanding routes to adaptive and maladaptive outcomes in vulnerable populations will also be important for the development of policies to support mental health. This includes developing an understanding of how psychopathology is shaped by society via a more in-depth understanding of population differences, cohort effects, and how socioeconomic and cultural influences impact psychopathology. Understanding long-term outcomes of psychopathology across multiple domains of functioning will be important for interventions that impact on the outcomes that matter most to those affected by psychopathology.

References

Basto-Pereira, M. & Farrington, D.P. (2022) Developmental predictors of offending and persistence in crime: a systematic review of meta-analyses. *Aggression and Violent Behavior* **65**, 101761.

Beesdo-Baum, K. & Knappe, S. (2012) Developmental epidemiology of anxiety disorders. *Child and Adolescent Psychiatric Clinics of North America* **21**(3), 457–478. doi:10.1016/j.chc.2012.05.001.

Beesdo-Baum, K. *et al.* (2012) The natural course of social anxiety disorder among adolescents and young adults. *Acta Psychiatrica Scandinavica* **126**(6), 411–425.

Bergen, S.E. *et al.* (2007) Age-related changes in heritability of behavioral phenotypes over adolescence and young adulthood: a meta-analysis. *Twin Research and Human Genetics: The Official Journal of the International Society for Twin Studies* **10**(3), 423–433. doi:10.1375/twin.10.3.423.

Bethlehem, R.A.I. *et al.* (2022) Brain charts for the human lifespan. *Nature* **604**(7906), 525–533. doi:10.1038/s41586-022-04554-y.

Bifulco, A. *et al.* (2002) Childhood adversity, parental vulnerability and disorder: examining inter-generational transmission of risk. *Journal of Child Psychology and Psychiatry* **43**(8), 1075–1086. doi:10.1111/1469-7610.00234.

Brotman, M.A. *et al.* (2017) Irritability in youths: a translational model. *The American Journal of Psychiatry* **174**(6), 520–532. doi:10.1176/appi.ajp.2016.16070839.

Broughton, T. *et al.* (2023) Relative age in the school year and risk of mental health problems in childhood, adolescence and young adulthood. *Journal of Child Psychology and Psychiatry* **64**(1), 185–196. doi:10.1111/jcpp.13684.

Burt, S.A. (2022) The genetic, environmental, and cultural forces influencing youth antisocial behavior are tightly intertwined. *Annual Review of Clinical Psychology* **18**, 155–178.

Caspi, A. *et al.* (2005) Personality development: stability and change. *Annual Review of Psychology* **56**, 453–484.

Caspi, A. *et al.* (2020) Longitudinal assessment of mental health disorders and comorbidities across 4 decades among participants in the Dunedin birth cohort study. *JAMA Network Open* **3**(4), e203221. doi:10.1001/jamanetworkopen.2020.3221.

Catalan, A. *et al.* (2021) Annual research review: prevention of psychosis in adolescents—systematic review and meta-analysis of advances in detection, prognosis and intervention. *Journal of Child Psychology and Psychiatry* **62**(5), 657–673. doi:10.1111/jcpp.13322.

Caye, A. *et al.* (2016) Predictors of persistence of ADHD into adulthood: a systematic review of the literature and meta-analysis. *European Child & Adolescent Psychiatry* **25**(11), 1151–1159. doi:10.1007/s00787-016-0831-8.

Chawner, S.J. *et al.* (2019) Genotype–phenotype associations in children with copy number variants associated with high neuropsychiatric risk in the UK (IMAGINE-ID): a case-control cohort study. *The Lancet Psychiatry* **6**(6), 493–505. doi:10.1016/S2215-0366(19)30123-3.

Cicchetti, D. (2016) Socioemotional, personality, and biological development: illustrations from a multilevel developmental psychopathology perspective on child maltreatment. *Annual Review of Psychology* **67**, 187–211. doi:10.1146/annurev-psych-122414-033259.

Cicchetti, D. & Rogosch, F.A. (1996) Equifinality and multifinality in developmental psychopathology. *Development and Psychopathology* **8**(4), 597–600.

Collishaw, S. (2015) Annual research review: secular trends in child and adolescent mental health. *Journal of Child Psychology and Psychiatry* **56**(3), 370–393.

Colman, I. *et al.* (2009) Outcomes of conduct problems in adolescence: 40 year follow-up of national cohort. *BMJ (Clinical Research Edition)* **338**, a2981. doi:10.1136/bmj.a2981.

Copeland, W. *et al.* (2010) Outcomes of early pubertal timing in young women: a prospective population-based study. *The American Journal of Psychiatry* **167**(10), 1218–1225. doi:10.1176/appi.ajp.2010.09081190.

Copeland, W.E. *et al.* (2009) Childhood and adolescent psychiatric disorders as predictors of young adult disorders. *Archives of General Psychiatry* **66**(7), 764–772. doi:10.1001/archgenpsychiatry.2009.85.

Copeland, W.E. *et al.* (2014) Longitudinal patterns of anxiety from childhood to adulthood: the Great Smoky Mountains study. *Journal of the American Academy of Child and Adolescent Psychiatry* **53**(1), 21–33. doi:10.1016/j.jaac.2013.09.017.

Costello, E.J. & Maughan, B. (2015) Annual research review: optimal outcomes of child and adolescent mental illness. *Journal of Child Psychology and Psychiatry* **56**(3), 324–341. doi:10.1111/jcpp.12371.

Costello, E.J. *et al.* (2003) Relationships between poverty and psychopathology: a natural experiment. *JAMA* **290**(15), 2023–2029. doi:10.1001/jama.290.15.2023.

Dalgleish, T. *et al.* (2020) Transdiagnostic approaches to mental health problems: current status and future directions. *Journal of Consulting and Clinical Psychology* **88**(3), 179–195. doi:10.1037/ccp0000482.

Dalsgaard, S. *et al.* (2015) Mortality in children, adolescents, and adults with attention deficit hyperactivity disorder: a nationwide cohort study. *The Lancet* **385**(9983), 2190–2196.

Davis, J. *et al.* (2016) A review of vulnerability and risks for schizophrenia: beyond the two hit hypothesis. *Neuroscience and Biobehavioral Reviews* **65**, 185–194. doi:10.1016/j.neubiorev.2016.03.017.

de Girolamo, G. *et al.* (2012) Age of onset of mental disorders and use of mental health services: needs, opportunities and obstacles. *Epidemiology and Psychiatric Sciences* **21**(1), 47–57. doi:10.1017/s2045796011000746.

Degnan, K.A. *et al.* (2010) Temperament and the environment in the etiology of childhood anxiety. *Journal of Child Psychology and Psychiatry* **51**(4), 497–517. doi:10.1111/j.1469-7610.2010.02228.x.

Dodge, K.A. (2006) Translational science in action: hostile attributional style and the development of aggressive behavior problems. *Development and Psychopathology* **18**(3), 791–814. doi:10.1017/s0954579406060391.

Driver, D.I. *et al.* (2013) Childhood onset schizophrenia and early onset schizophrenia spectrum disorders. *Child and Adolescent Psychiatric Clinics* **22**(4), 539–555.

Eaton, W.W. *et al*. (2008) Population-based study of first onset and chronicity in major depressive disorder. *Archives of General Psychiatry* **65**(5), 513–520.

Faraone, S.V. *et al*. (2006) The age-dependent decline of attention deficit hyperactivity disorder: a meta-analysis of follow-up studies. *Psychological Medicine* **36**(2), 159–165. doi:10.1017/S003329170500471X.

Ge, X. *et al*. (1996) Coming of age too early: pubertal influences on girls' vulnerability to psychological distress. *Child Development* **67**(6), 3386–3400. doi:10.1111/j.1467-8624.1996.tb01919.x.

Gregory, A.M. *et al*. (2007) Juvenile mental health histories of adults with anxiety disorders. *American Journal of Psychiatry* **164**(2), 301–308. doi:10.1176/ajp.2007.164.2.301.

Hall, J. & Bray, N.J. (2022) Schizophrenia genomics: convergence on synaptic development, adult synaptic plasticity, or both? *Biological Psychiatry* **91**(8), 709–717. doi:10.1016/j.biopsych.2021.10.018.

Hammerton, G. & Munafò, M.R. (2021) Causal inference with observational data: the need for triangulation of evidence. *Psychological Medicine* **51**(4), 563–578. doi:10.1017/S0033291720005127.

Hardt, J. & Rutter, M. (2004) Validity of adult retrospective reports of adverse childhood experiences: review of the evidence. *Journal of Child Psychology and Psychiatry* **45**(2), 260–273. doi:10.1111/j.1469-7610.2004.00218.x.

Harold, G.T. & Sellers, R. (2018) Annual research review: interparental conflict and youth psychopathology: an evidence review and practice focused update. *Journal of Child Psychology and Psychiatry* **59**(4), 374–402. doi:10.1111/jcpp.12893.

Harold, G.T. *et al*. (2017) How can genetically informed research help inform the next generation of interparental and parenting interventions? *Child Development* **88**(2), 446–458. doi:10.1111/cdev.12742.

Harrington, R. *et al*. (1997) Psychiatric disorders in the relatives of depressed probands. I. Comparison of prepubertal, adolescent and early adult onset cases. *Journal of Affective Disorders* **42**(1), 9–22. doi:10.1016/s0165-0327(96)00091-2.

Harvey, A. *et al*. (2004) *Cognitive Behavioral Processes Across Psychological Disorders: A Transdiagnostic Approach to Research and Treatment*. Oxford University Press, Oxford.

Heins, M. *et al*. (2011) Childhood trauma and psychosis: a case-control and case-sibling comparison across different levels of genetic liability, psychopathology, and type of trauma. *The American Journal of Psychiatry* **168**(12), 1286–1294. doi:10.1176/appi.ajp.2011.10101531.

Hovenkamp-Hermelink, J.H.M. *et al*. (2021) Predictors of persistence of anxiety disorders across the lifespan: a systematic review. *The Lancet Psychiatry* **8**(5), 428–443. doi:10.1016/S2215-0366(20)30433-8.

Howes, O.D. & Murray, R.M. (2014) Schizophrenia: an integrated sociodevelopmental-cognitive model. *The Lancet*. doi:10.1016/S0140-6736(13)62036-X.

Hussong, A.M. *et al*. (2004) Substance abuse hinders desistance in young adults' antisocial behavior. *Development and Psychopathology* **16**(4), 1029–1046. doi:10.1017/s095457940404012x.

Insel, T. *et al*. (2010) Research domain criteria (RDoC): toward a new classification framework for research on mental disorders. *The American Journal of Psychiatry* **167**(7), 748–751. doi:10.1176/appi.ajp.2010.09091379.

John, A. *et al*. (2023) Self-harm, in-person bullying and cyberbullying in secondary school-aged children: a data linkage study in Wales. *Journal of Adolescence* **95**(1), 97–114.

Jones, H.J. *et al*. (2016) Phenotypic manifestation of genetic risk for schizophrenia during adolescence in the general population. *JAMA Psychiatry* **73**(3), 221–228. doi:10.1001/jamapsychiatry.2015.3058.

Judd, L.L. *et al*. (1998) A prospective 12-year study of subsyndromal and syndromal depressive symptoms in unipolar major depressive disorders. *Archives of General Psychiatry* **55**(8), 694–700. doi:10.1001/archpsyc.55.8.694.

Kendler, K.S. *et al*. (2008) A developmental twin study of symptoms of anxiety and depression: evidence for genetic innovation and attenuation. *Psychological Medicine* **38**, 1567–1575.

Kendler, K.S. *et al*. (2017) The role of marriage in criminal recidivism: a longitudinal and co-relative analysis. *Epidemiology and Psychiatric Sciences* **26**(6), 655–663.

Kendler, K.S. *et al*. (2022) An extended Swedish adoption study of anxiety disorder and its cross-generational familial relationship with major depression. *The American Journal of Psychiatry* **179**(9), 640–649. doi:10.1176/appi.ajp.21111110.

Kim-Cohen, J. *et al*. (2003) Prior juvenile diagnoses in adults with mental disorder: developmental follow-back of a prospective-longitudinal cohort. *Archives of General Psychiatry* **60**(7), 709–717. doi:10.1001/archpsyc.60.7.709.

Kotov, R. *et al*. (2017) The hierarchical taxonomy of psychopathology (HiTOP): a dimensional alternative to traditional nosologies. *Journal of Abnormal Psychology* **126**(4), 454–477. doi:10.1037/abn0000258.

Kraemer, H.C. *et al*. (2000) How can we learn about developmental processes from cross-sectional studies, or can we? *American Journal of Psychiatry* **157**(2), 163–171. doi:10.1176/appi.ajp.157.2.163.

Lacey, R.E. *et al*. (2020) Adverse childhood experiences and adult inflammation: single adversity, cumulative risk and latent class approaches. *Brain, Behavior, and Immunity* **87**, 820–830.

Lau, J.Y.F. & Eley, T.C. (2010) The genetics of mood disorders. *Annual Review of Clinical Psychology* **6**, 313–337.

Laub, J.H. & Sampson, R.J. (2006) *Shared Beginnings, Divergent Lives: Delinquent Boys to Age 70*. Harvard University Press, Cambridge, MA.

LeDoux, J.E. & Pine, D.S. (2016) Using neuroscience to help understand fear and anxiety: a two-system framework. *The American Journal of Psychiatry* **173**(11), 1083–1093. doi:10.1176/appi.ajp.2016.16030353.

Leijten, P. *et al*. (2019) Meta-analyses: key parenting program components for disruptive child behavior. *Journal of the American Academy of Child and Adolescent Psychiatry* **58**(2), 180–190. doi:10.1016/j.jaac.2018.07.900.

Lenroot, R.K. & Giedd, J.N. (2006) Brain development in children and adolescents: insights from anatomical magnetic resonance imaging. *Neuroscience & Biobehavioural Reviews* **30**, 718–729.

Livingston, L.A. *et al*. (2019) Compensatory strategies below the behavioural surface in autism: a qualitative study. *The Lancet Psychiatry* **6**(9), 766–777. doi:10.1016/S2215-0366(19)30224-X.

Maggioni, E. *et al*. (2020) Twin MRI studies on genetic and environmental determinants of brain morphology and function in the early lifespan. *Neuroscience & Biobehavioural Reviews* **109**, 139–149.

Maughan, B. *et al*. (2009) Persistence of literacy problems: spelling in adolescence and at mid-life. *Journal of Child Psychology and Psychiatry, and Allied Disciplines* **50**(8), 893–901. doi:10.1111/j.1469-7610.2009.02079.x.

Moffitt, T.E. (1993) Adolescence-limited and life course-persistent antisocial behavior: a developmental taxonomy. *Psychological Review* **100**(4), 674.

Moffitt, T.E. *et al.* (2011) A gradient of childhood self-control predicts health, wealth, and public safety. *Proceedings of the National Academy of Sciences* **108**, 2693–2698.

Moffitt, T.E. *et al.* (2015) Is adult ADHD a childhood-onset neurodevelopmental disorder? Evidence from a four-decade longitudinal cohort study. *The American Journal of Psychiatry* **172**(10), 967–977. doi:10.1176/appi.ajp.2015.14101266.

Musliner, K.L. *et al.* (2016) Heterogeneity in long-term trajectories of depressive symptoms: patterns, predictors and outcomes. *Journal of Affective Disorders* **192**, 199–211. doi:10.1016/j.jad.2015.12.030.

National Scientific Council on the Developing Child (2015) *Supportive Relationships and Active Skill-Building Strengthen the Foundations of Resilience: Working Paper 13*. Available: https://developingchild.harvard.edu/resources/supportive-relationships-and-active-skill-building-strengthen-the-foundations-of-resilience/ [5 September 2024].

Newbury, J.B. *et al.* (2018) Measuring childhood maltreatment to predict early-adult psychopathology: comparison of prospective informant-reports and retrospective self-reports. *Journal of Psychiatric Research* **96**, 57–64. doi:10.1016/j.jpsychires.2017.09.020.

Ng-Knight, T. *et al.* (2019) 'Best friends forever'? Friendship stability across school transition and associations with mental health and educational attainment. *The British Journal of Educational Psychology* **89**(4), 585–599. doi:10.1111/bjep.12246.

Nigg, J.T. *et al.* (2020) Development of ADHD: etiology, heterogeneity, and early life course. *Annual Review of Developmental Psychology* **2**(1), 559–583. doi:10.1146/annurev-devpsych-060320-093413.

Oldehinkel, A.J. & Bouma, E.M. (2011) Sensitivity to the depressogenic effect of stress and HPA-axis reactivity in adolescence: a review of gender differences. *Neuroscience and Biobehavioral Reviews* **35**(8), 1757–1770. doi:10.1016/j.neubiorev.2010.10.013.

Ordaz, S. & Luna, B. (2012) Sex differences in physiological reactivity to acute psychosocial stress in adolescence. *Psychoneuroendocrinology* **37**(8), 1135–1157. doi:10.1016/j.psyneuen.2012.01.002.

Owen, M.J. *et al.* (2016) Schizophrenia. *The Lancet* **388**(10039), 86–97. doi:10.1016/S0140-6736(15)01121-6.

Patton, G.C. *et al.* (2014) The prognosis of common mental disorders in adolescents: a 14-year prospective cohort study. *The Lancet* **383**(9926), 1404–1411.

Pickles, A. *et al.* (2010) Predictors of suicidality across the life span: the Isle of Wight study. *Psychological Medicine* **40**(9), 1453–1466. doi:10.1017/S0033291709991905.

Pingault, J.B. *et al.* (2015) Genetic and environmental influences on the developmental course of attention-deficit/hyperactivity disorder symptoms from childhood to adolescence. *JAMA Psychiatry* **72**(7), 651–658. doi:10.1001/jamapsychiatry.2015.0469.

Plana-Ripoll, O. *et al.* (2019) Exploring comorbidity within mental disorders among a Danish national population. *JAMA Psychiatry* **76**(3), 259–270. doi:10.1001/jamapsychiatry.2018.3658.

Quinton, D. *et al.* (1993) Partners, peers, and pathways: assortative pairing and continuities in conduct disorder. *Development and Psychopathology* **5**(4), 763–783.

Rasmussen, S.A. *et al.* (2016) Zika virus and birth defects—reviewing the evidence for causality. *New England Journal of Medicine* **374**, 1981–1987. doi:10.1056/NEJMsr1604338.

Reuben, A. *et al.* (2016) Lest we forget: comparing retrospective and prospective assessments of adverse childhood experiences in the prediction of adult health. *Journal of Child Psychology and Psychiatry, and Allied Disciplines* **57**(10), 1103–1112. doi:10.1111/jcpp.12621.

Rice, F. *et al.* (2003) Negative life events as an account of age-related differences in the genetic aetiology of depression in childhood and adolescence. *Journal of Child Psychology and Psychiatry* **44**(7), 977–987. doi:10.1111/1469-7610.00182.

Rice, F. *et al.* (2009) Disentangling prenatal and inherited influences in humans with an experimental design. *Proceedings of the National Academy of Sciences of the United States of America* **106**(7), 2464–2467. doi:10.1073/pnas.0808798106.

Rice, F. *et al.* (2017) Antecedents of new-onset major depressive disorder in children and adolescents at high familial risk. *JAMA Psychiatry* **74**(2), 153–160. doi:10.1001/jamapsychiatry.2016.3140.

Rice, F. *et al.* (2019) Characterizing developmental trajectories and the role of neuropsychiatric genetic risk variants in early-onset depression. *JAMA Psychiatry* **76**(3), 306–313. doi:10.1001/jamapsychiatry.2018.3338.

Riglin, L. *et al.* (2022) "Late-onset" ADHD symptoms in young adulthood: is this the same as child-onset ADHD? *Journal of Attention Disorders* **26**(10), 1271–1282.

Rowe, R. *et al.* (2010) Developmental pathways in oppositional defiant disorder and conduct disorder. *Journal of Abnormal Psychology* **119**(4), 726–738. doi:10.1037/a0020798.

Rutter, M. (1989) Pathways from childhood to adult life. *Journal of Child Psychology and Psychiatry* **30**, 23–51.

Rutter, M. (2006) Implications of resilience concepts for scientific understanding. *Annals of the New York Academy of Sciences* **1094**, 1–12. doi:10.1196/annals.1376.002.

Rutter, M. (2009) Understanding and testing risk mechanisms for mental disorders. *Journal of Child Psychology and Psychiatry, and Allied Disciplines* **50**(1–2), 44–52. doi:10.1111/j.1469-7610.2008.01976.x.

Rutter, M. & Sroufe, L.A. (2000) Developmental psychopathology: concepts and challenges. *Development and Psychopathology* **12**, 265–296.

Rutter, M. *et al.* (2006) Continuities and discontinuities in psychopathology between childhood and adult life. *Journal of Child Psychology and Psychiatry* **47**(3–4), 276–295.

Sadler, K. *et al.* (2018) *Mental Health of Children and Young People in England, 2017*. NHS Digital.

Sallis, H.M. *et al.* (2021) Genetic liability to schizophrenia is associated with exposure to traumatic events in childhood. *Psychological Medicine* **51**(11), 1814–1821. doi:10.1017/S0033291720000537.

Sandstrom, A. *et al.* (2019) Offspring of parents with schizophrenia, bipolar disorder, and depression: a review of familial high-risk and molecular genetics studies. *Psychiatric Genetics* **29**(5), 160–169.

Schubert, K.O. *et al.* (2017) Depressive symptom trajectories in late adolescence and early adulthood: a systematic review. *The Australian and New Zealand Journal of Psychiatry* **51**(5), 477–499. doi:10.1177/0004867417700274.

Sellers, R. *et al.* (2019) Cross-cohort change in adolescent outcomes for children with mental health problems. *Journal of Child Psychology and Psychiatry* **60**(7), 813–821.

Shell, M.D. *et al.* (2014) Anxious solitude and the middle school transition: a diathesis × stress model of peer exclusion and victimization trajectories. *Developmental Psychology* **50**(5), 1569–1583. doi:10.1037/a0035528.

Silberg, J.L. *et al.* (2001) Genetic and environmental influences on the temporal association between earlier anxiety and later depression in girls. *Biological Psychiatry* **49**(12), 1040–1049. doi:10.1016/s0006-3223(01)01161-1.

Solmi, M. *et al*. (2022) Age at onset of mental disorders worldwide: large-scale meta analysis of 192 epidemiological studies. *Molecular Psychiatry* **27**, 281–295.

Sonuga-Barke, E.J.S. *et al*. (2017) Child-to-adult neurodevelopmental and mental health trajectories after early life deprivation: the young adult follow-up of the longitudinal English and Romanian Adoptees study. *The Lancet* **389**(10078), 1539–1548. doi:10.1016/S0140-6736(17)30045-4.

Sroufe, L.A. *et al*. (2005) *The Development of the Person: The Minnesota Study of Risk and Adaptation from Birth to Adulthood*. The Guilford Press, London.

Thapar, A. & Rutter, M. (2009) Do prenatal risk factors cause psychiatric disorder? Be wary of causal claims. *British Journal of Psychiatry* **195**, 100–101. doi:10.1192/bjp.bp.109.062828.

Thapar, A. & Rutter, M. (2019) Do natural experiments have an important future in the study of mental disorders? *Psychological Medicine* **49**(7), 1079–1088.

Thapar, A. *et al*. (2017) Neurodevelopmental disorders. *The Lancet Psychiatry* **4**(4), 339–346. doi:10.1016/S2215-0366(16)30376-5.

Tremblay, R.E. (2010) Developmental origins of disruptive behaviour problems: the 'original sin' hypothesis, epigenetics and their consequences for prevention. *Journal of Child Psychology and Psychiatry, and Allied Disciplines* **51**(4), 341–367. doi:10.1111/j.1469-7610.2010.02211.x.

Trubetskoy, V. *et al*. (2022) Mapping genomic loci implicates genes and synaptic biology in schizophrenia. *Nature* **604**(7906), 502–508. doi:10.1038/s41586-022-04434-5.

Vitaro, F. & Tremblay, R.E. (2008) Clarifying and maximising the usefulness of targeted preventive interventions. In: *Rutter's Child and Adolescent Psychiatry*. (eds M. Rutter, *et al.*), 5th edn. Blackwell, Oxford. pp. 989–1008.

Waszczuk, M.A. *et al*. (2016) The stability and change of etiological influences on depression, anxiety symptoms and their co-occurrence across adolescence and young adulthood. *Psychological Medicine* **46**(1), 161–175. doi:10.1017/S0033291715001634.

Waters, H.S. & Waters, E. (2006) The attachment working models concept: among other things, we build script-like representations of secure base experiences. *Attachment & Human Development* **8**(3), 185–197.

Weavers, B. *et al*. (2021) The antecedents and outcomes of persistent and remitting adolescent depressive symptom trajectories: a longitudinal, population-based English study. *The Lancet Psychiatry* **8**(12), 1053–1061. doi:10.1016/S2215-0366(21)00281-9.

Weinberger, D.R. (1987) Implications of normal brain development for the pathogenesis of schizophrenia. *Archives of General Psychiatry* **44**, 660–669.

Widom, C.S. *et al*. (2007) A prospective investigation of major depressive disorder and comorbidity in abused and neglected children grown up. *Archives of General Psychiatry* **64**(1), 49–56.