Depression During Childhood and Adolescence

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Abstract
Information on depression during childhood and adolescence is reviewed from a developmental psychopathology perspective. The chapter covers descriptive and diagnostic features, as well as considering key concerns in the assessment of depression from childhood through adolescence. Issues of developmental epidemiology, including prevalence rates, developmental trajectories, sex differences, and developmental patterns of sequential comorbidity, are reviewed. Multiple risks and vulnerabilities, with particular attention to the developmental relevance of these factors and processes in children and adolescence, are considered. Finally, future directions are emphasized, including continued investigation of transdiagnostic versus specific etiologies, employing multiple levels of analysis, and translating risk knowledge into personalization of evidence-based, developmentally sensitive assessment and intervention approaches.

Key Words: children and adolescents, developmental psychopathology, description, epidemiology, risk and vulnerability, assessment and intervention

Introduction
Depression is one of the most commonly occurring of the major psychiatric disorders, and depression over the lifespan often begins in adolescence. It is a prototypical multifaceted disorder that profoundly affects individuals’ emotions, thoughts, sense of self, behaviors, interpersonal relations, productivity, and life satisfaction. Indeed, given the multiple effects that depression has, it has been ranked as the fourth leading cause of disability and premature death worldwide (Murray & Lopez, 1996). This chapter focuses on three main issues for child and adolescent depression: (1) description, diagnosis, and assessment; (2) developmental patterns of depression over time; and (3) various risks and vulnerabilities to depression among youth.

Description, Diagnosis, and Assessment
Issues in Youth Depression
Depression as Mood, Syndrome, and Disorder
Depression can refer to three hierarchically arranged constructs: mood, syndrome, and clinical disorder (Petersen et al., 1993). At the level of mood, depression connotes feeling sad, unhappy, or irritable without any additional symptoms or time frame. At the syndrome level, depression signifies the mood component along with typically occurring symptoms (e.g., vegetative, cognitive, affective symptoms) without a minimum time frame or clinical impairment. At the disorder level, depression is defined according to the official psychiatric classification system (the Diagnostic and Statistical Manual of Mental Disorders, 5th ed., DSM-V;
American Psychiatric Association, 2013), in which particular symptoms are required for a minimum duration with impairment. Relatively more research focuses on and assesses depression at the mood and syndrome level, which implies a dimensional perspective on depression at a latent level (see Hankin, Fraley, Lahey, & Waldman, 2005, for evidence). Although there are likely continuities among depression conceptualized and studied at the mood, syndrome, and disorder level, research is still needed to verify that findings (e.g., risk factors) obtained at one level (e.g., syndrome) apply to other levels (e.g., disorder with impairment and minimum duration) as there may be discontinuities such that some findings may pertain to depression at the syndrome, but not disorder, level.

With respect to youth, DSM-V asserts that an episode of major depression can be diagnosed on the basis of the same symptoms in childhood and adolescence and in adulthood, except that irritability can be applied as a mood symptom along with depressed, sad mood and anhedonia (loss of pleasure) in youth. DSM-V states that dysthymia in youth has the same symptom profile as in adults, but there is a minimum 1-year duration in youth compared with 2 years in adulthood. Although in some research, Major Depressive Disorder is considered separately from Dysthymia, most studies with youth either combine them to examine clinically significant depressive disorders or focus on depressive symptoms more broadly. As such, this chapter reviews evidence for depression generally, as opposed to a specific diagnosis of Major Depressive Disorder or Dysthymia.

**Manifestation and Presentation of Depression in Children and Adolescents**

DSM-V now notes that the nature of depression in youth may be slightly different from that of adults. Hypersomnia is more likely among children, whereas melancholia and psychomotor problems are observed more among adolescents. Other research suggests additional potential developmental differences in depression presentation as the syndrome and predominant symptoms of depression may differ as a function of development given cognitive, social, emotional, and biological changes that transpire throughout childhood and adolescence (Weiss & Garber, 2003). Very young children, especially preschoolers, tend not to report depressed mood or hopelessness, and younger children are more likely to describe somatic symptoms of depression (Weiss & Garber, 2003). Other symptoms, such as anhedonia and psychomotor retardation, tend to increase and become more prevalent with the transition from childhood into adolescence, whereas the symptoms of somatic complaints and the physical appearance of looking depressed tend to decrease with age. Still it is clear that children who fit DSM-V criteria for clinical depression can be identified.

**Assessment Approaches and Issues Specific to Youth Depression**

Many instruments are available to assess depression in children and adolescents (Klein, Dougherty, & Olino, 2005). Some of these map directly onto DSM-V's definition and criteria for youth depression, whereas others are related to but are substantially different in approach from the DSM-V conceptualization of depression as a clinical disorder. These tools include brief, reliable, valid, normed questionnaires that can be completed in a short time frame by the adolescent (e.g., the Children's Depression Inventory; Mood and Feelings Questionnaire) or the parent (e.g., Child Behavior Checklist). Adolescents who score higher than a cut point that is based on normed national samples can be evaluated further with more thorough diagnostic assessments. These include clinical rating scales (e.g., Children's Depression Rating Scale, CDRS) as well as semistructured interviews (e.g., K-SADS) in which a trained professional interviews the adolescent (and often a caretaker), on the basis of which a diagnosis of a depressive disorder can be given.

**Developmental Epidemiology of Depression**

**Prevalence of Depression in Children and Adolescents: Developmental Trends**

The prevalence of depression has been examined in many studies with different age groups, methods, and samples. In this chapter, representative community samples are emphasized for estimating rates of depression because samples drawn from psychiatric clinics may be biased in various ways (e.g., actively treatment seeking, greater symptom severity, and higher comorbidity rates), and these biases can inflate rates and comorbidity levels of depression. Cross-sectional studies of adolescent self-reported depressive mood and symptoms (i.e., less than a clinically significant depressive disorder) indicate that between 20% and 50% of adolescents report significant symptom levels of depression (Petersen et al., 1993). Prospective longitudinal studies of self-reported depressive symptoms show that the average levels of depressive mood and symptoms rise substantially from relatively low levels in
childhood to much higher levels starting in middle adolescence (Ge, Lorenz, Conger, & Elder, 1994). It is important to note that elevated rates of depressed mood or symptoms do not merely indicate typical, benign adolescent “moodiness” or “turmoil,” but rather represent a substantial risk for later, clinically significant depressive disorder (Pine, Cohen, Cohen, & Brook, 1999) and impaired functioning.

Cross-sectional (e.g., Merikangas et al., 2010) and longitudinal (e.g., Hankin et al., 1998) studies of diagnosed clinical levels of depression show that the rates of depression are generally low in children and increase to near-adult prevalence levels in adolescence. Preadolescent school-aged children tend to have low lifetime prevalence rates (i.e., a depressive episode determined to have occurred at some point over the child’s life up to the time of diagnostic assessment) of depression (<3%), and lifetime rates of depression are higher among adolescents (i.e., ages 13–17 years) (11.7% overall; 8.7% with severe impairment; Merikangas et al., 2010). There is a dramatic surge in rates of depression within this broad 13–17 year age range, as indicated by an increase in rates from 3% in early adolescence (age 13 years) to 17% by the end of adolescence (age 18 years) based on prospective, repeated diagnostic interview research on the development of clinical depression (Hankin et al., 1998).

**Depression Trajectories: Continuity, Discontinuity, and Recurrence Patterns**

Depressed mood at younger ages carries a risk for the development of depressive disorder later in life (Reinherz, Giaconia, Hauf, Wasserman, & Paradis, 2000). Compared with the continuity of depression from childhood into adulthood, there is much stronger continuity for depression from adolescence into adulthood (Pine et al., 1999). Most depressed prepubertal children do not grow up to become adults with major depression. That there is less continuity in depression from childhood into adulthood as there is from adolescence into adulthood is consistent with the notion that there are important developmental differences (e.g., symptoms and potential causes) between depressions that arise during childhood and those that develop in adolescence or adulthood (Jaffee et al., 2002).

A frequently stated truism is that depression is a chronic, recurrent disorder. This can be evaluated in two ways. First, many depressed adults previously were depressed in childhood or adolescence, and their current adult depressive episode is a recurrence. For example, in a prospective follow-back study (Kim-Cohen et al., 2003), in which an entire birth cohort of individuals was followed for 26 years, the majority of depressed adults at age 26 years (75%) had previously had a depressive disorder in childhood or adolescence. Moreover, approximately half of adults with a diagnosis of depression experience a recurrence within 2 years and more than 80% within 5–7 years (Judd, 1997). Approximately 40% of youth experience a depression recurrence over 3–5 years (Lewinsohn, Clarke, Seeley, & Rohde, 1994). Second, recurrences can occur from either an acute or chronic illness perspective (Monroe & Harkness, 2011). Within the acute illness view, a recurrent episode occurs due to a new instance of the illness, rather than a continuation of a previous disorder, whereas the chronic illness perspective suggests that there is a stable liability, including a trait-like, enduring vulnerability prior to the first episode that persists into later recurrent episodes. Although approximately 60% of individuals experience a recurrent episode, this also means that 40% of individuals who experience a first episode never experience a second (Monroe & Harkness, 2011).

This highlights the likely different trajectories emanating after a first episode of depression and the existence of both continuity (recurrence) and discontinuity (only one episode) in depression. Consistent with this multiple trajectories perspective, most studies have found evidence for four latent trajectory groups of depressive symptoms in youth (e.g., Costello, Swendsen, Rose, & Dierker, 2008). They identified a “severe” group of adolescents who experience persistently high level of depressive symptoms, a group consistently low in symptoms, as well as a “stable moderate” and an “increasing over time” group. The severe group of youth reporting high symptoms that are maintained over time is consistent with the chronic depression characterization. Future research is needed to identify risk factors that best differentiate these trajectories, especially focusing on discontinuities after a first episode, to identify those who develop a chronic pattern versus a single lifetime episode.

**Emergence of Sex Differences in Depression in Early Adolescence**

Depression is more common in women than in men (see Chapter 4). Various studies have examined the emergence of this sex difference developmentally from childhood through adolescence and into adulthood. More girls than boys report depression starting in early adolescence (around ages 12 and 13 years; Ge et al., 1994). Longitudinal
Developmental Patterns of Sequential Comorbidity in Youth Depression

Depression commonly occurs with other disorders. Angold, Costello, and Erkanli (1999) showed that depression is associated at greater than chance levels with anxiety disorders (median odds ratio = 8.2), behavioral disorders (OR = 6.6), and attention deficit hyperactivity disorder (ADHD) (OR = 5.5).

In addition to concurrent diagnostic cooccurrence, there are developmental patterns of sequential comorbidity. Children with depression are more likely to have a cooccurring diagnosis of separation anxiety disorder, whereas older adolescents are more likely to exhibit an eating disorder and substance use comorbidities. Although anxiety has been found in many studies to precede later depression (e.g., Kim-Cohen et al., 2003), other studies have not replicated this pattern but instead have shown that depression can precede anxiety (Moffitt et al., 2007) or that homotypic (the same underlying process and the same manifestation of construct), relative to heterotypic (same underlying process but different manifestation of construct), continuity is more common (Keenan, Feng, Hipwell, & Klostermann, 2009). There may be three developmental patterns to anxiety and depression (Cummings, Caporino, & Kendall, 2014): (1) anxiety precedes depression, (2) anxiety and depression cooccur simultaneously, and (3) depression comes before anxiety. Future research is needed to differentiate among these different developmental patterns, which may differ across the anxiety disorders (e.g., generalized anxiety, social anxiety, panic) and at different ages. Similar inconsistent patterns have been observed with externalizing behaviors. Some research shows that earlier externalizing behaviors tend to predict later depressive symptoms (Kim-Cohen et al., 2003), whereas other studies show the opposite (Wolff & Ollendick, 2006).

Risk and Vulnerability Factors for Youth Depression

There are many risk factors and processes that enhance vulnerability for youth's likelihood of developing depression, including stressful life events and traumas, genetic influences, temperament, cognitive factors, biological risks, and interpersonal influences (see Abela & Hankin, 2008 for reviews). This section provides brief reviews of these various risks with a focus on how they apply to children and adolescents within a developmental psychopathological framework.

Stressful Life Events and Trauma

Stressful life events play a substantial contributory role in the development of depression from childhood through adulthood (Grant et al., 2003). Almost all individuals with a depressive disorder will have encountered at least one significant negative life event in the month prior to the onset of depression. Additionally, longitudinal studies have demonstrated that experiencing stressors precedes the initial elevation, recurrence, and exacerbation of depression (e.g., Ge et al., 1994).

In addition to the perspective that stressors precede and contribute to depression (i.e., stress exposure model), a complementary perspective suggests that the stress–depression relationship is not a static, unidirectional one, but rather a bidirectional, transactional process. The stress generation hypothesis (Hammen, 1991) suggests that some individuals, because of personality characteristics (e.g., neuroticism), behaviors (e.g., excessive reassuring seeking), or mood/emotions (e.g., being depressed) generate stressful circumstances and additional events for themselves. Transactionally, then, these dependent stressors (those events that are, at least partly, based on individuals’ behaviors) can lead to further increases in depression (Hankin & Abramson, 2001). Cross-sectional and longitudinal—including multiwave (e.g., Hankin, Mermelstein, & Roesch, 2007)—studies show that depressive symptoms, in particular, but also other individual differences (e.g., interpersonal and cognitive risks, temperament, psychopathology), are associated with subsequently occurring self-generated, dependent stressors (Liu & Alloy,
Genetic Influences

Having a parent with a history of major depression is one of the strongest predictors of depression in youth (Beardslee, Versage, & Gladstone, 1998). Behavior genetic studies with children and adolescents have found depression to be moderately heritable (~40%; Lau & Eley, 2010; Rice, Harold, & Thaper, 2002). Evidence from twin research also suggests that depressive symptoms are heritable starting in adolescence (after age 11 years) and continuing throughout adulthood, whereas shared common family environment, but less so latent genetic risk, is linked with depression in childhood (before age 11 years; Rice et al., 2002). Other research suggests developmental differences in genetic influences; genetic liability increases the risk both of depression and for experiencing stressors for girls after, but not before, puberty (Silberg et al., 1999). Finally, some of the etiological risk factors for depression, including exposure to stressful life events (Thapar, Harold, & McGuffin, 1998), cognitive risks (Lau, Rijndijk, & Eley, 2006), and temperament (e.g., negative emotionality, Lau & Eley, 2010), are themselves moderately heritable.

Research has also revealed gene–environment interactions (G×E). Some individuals are more likely to become depressed in the face of certain environmental risks because of genetic liability. Whereas G×E has been shown using behavioral genetic designs, the most specific evidence to date for G×E comes from molecular genetic studies that include assessments of stressors. Many specific genetic polymorphisms have been investigated in G×E for depression. A functional polymorphism in the promoter region of the serotonin transporter (5-hydroxytryptamine, 5-HTT) interacting with stressors has been most studied (see Chapter 9, Karg, Burmeister, Shedden, & Sen, 2011, for reviews). Such research with youth has been supportive, especially when strong measurement of stress (e.g., contextual stress interviews; Hammen, Brennan, Keenan-Miller, Hazel, & Najman, 2010; Jenness, Hankin, Abela, Young, & Smolen, 2011) is used and when idiographic (i.e., within person changes over time) stressors are examined (Hankin, Jenness, Abela, & Smolen, 2011).

Temperament

A prominent model (Rothbart & Bates, 1998) postulates three temperament dimensions: negative emotionality (NE), positive emotionality (PE), and effortful control (EC). NE involves a tendency toward negative emotions (e.g., discomfort, fear,
anger, sadness) and stress, and PE characterizes the extent to which an individual is outgoing, sociable, and receptive to reward. Both NE and PE represent affective reactive dimensions of temperament. Finally, EC taps into self-regulatory, executive functioning (EF), and control processes (e.g., task persistence, attentional control, planfulness, inhibitory control) that can modulate the more affective/reactive dimensions of NE and PE.

A robust finding is that depression is related, both concurrently and prospectively, to negative emotionality (Ormel et al., 2013). PE (lower levels) is also related to depression, especially in the context of high NE (Compas, Connor-Smith, & Jaser, 2004). Finally, initial research finds that the three-way interaction (high NE, low PE, and low EC) may constitute the highest temperamental risk to depression (Vasey et al., 2013). In essence, reactive temperament risk is a function of high NE × low PE, but this synergistic affective reactivity likely contributes to depression only among individuals with low EC who lack resources or skills to inhibit or interrupt the more toxic combination of high NE and low PE.

**Biological Risks**

Numerous aspects of biological vulnerability to depression have been investigated (see Chapter 9). This section briefly focuses on research highlighting (1) neuroendocrine dysregulation in response to stressors and (2) dysregulated brain regions associated with depression.

In humans stressful environmental challenges activate the hypothalamic–pituitary–adrenal (HPA) axis, and dysregulation of this human stress response may constitute a biological vulnerability to depression. Although many studies of depressed adults have demonstrated cortisol hyperreactivity in response to challenge, fewer studies have investigated the role of cortisol, and the direction of effects, among depressed youth (Guerry & Hastings, 2011; Lopez-Duran, Kovacs, & George, 2009). Developmentally, HPA axis dysregulation among youth may be more complex and nuanced than the standard hypercortisolism-to-challenge pattern found in depressed adults. Research with postpubertal adolescents (Hankin, Badanes, Abela, & Watamura, 2010; Rao, Hammen, Ortiz, Chen, & Poland, 2009) shows that depressed adolescents exhibit cortisol hyperreactivity in response to challenge in a manner similar to adults; younger prepubertal children tend to demonstrate cortisol hyporesponsivity to laboratory stressors (Hankin, Badanes, et al., 2010; Luby et al., 2003). Such research suggests a developmental switch in HPA axis dysregulation around puberty. Finally, findings from the few longitudinal studies that have been conducted suggest that HPA axis dysregulation predicts later symptoms and the course of depression (Badanes, Watamura, & Hankin, 2011; Rao, Hammen, & Poland, 2010).

In addition, findings from neuroimaging studies suggest a generally consistent picture of how particular brain regions and neural circuits are connected in such a way as to confer risk to depression (Disner, Beevers, Haigh, & Beck, 2011). In short, limbic areas (e.g., the amygdala), which are frequently found to be associated with high negative emotion and affectivity, tend to be overactive, whereas various interconnected prefrontal cortex (PFC) areas (e.g., dorsolateral, ventromedial, orbitofrontal), which commonly are found to be associated with EF and cognitive control, are underactivated. Moreover, some empirical evidence indicates that there is a strong functional connectivity (inverse association) between PFC and limbic areas. The PFC, as a broad area linked with higher order planning, EF, and cognitive control, can moderate more affective, limbic-driven activity. Research indicates that children (Tomarken, Simien, & Garber, 1994) of depressed mothers, who are at high risk for depression but are not yet depressed, have left-frontal underactivity. Moreover, other prominent brain regions are dysregulated in depression. These include the anterior cingulate, which has been associated with emotion processing, attention, and more general cognitive control (Hamilton, Chen, Thomason, Schwartz, & Gotlib, 2011), and reward processing areas (e.g., nucleus accumbens and ventral striatum; Forbes et al., 2006).

Still, despite the exciting potential of biological vulnerabilities, most of the research to date has employed cross-sectional designs. Retrospective, cross-sectional studies cannot disentangle biological factors as a cause, correlate, or consequence of depression, nor can they clearly establish whether putative biological indices constitute a relatively stable vulnerability for depression. Developmentally sensitive, prospective studies are needed.

**Cognitive Risks**

Four social–cognitive vulnerability factors (see Chapter 12) have received the most attention and support in predicting youth depression: (1) negative inferential styles about causes, consequences, and the self, (2) dysfunctional attitudes and maladaptive
schemata, (3) the tendency to ruminate in response to depressed mood, and (4) self-criticism. Several prospective studies have shown that these cognitive factors confer vulnerability to depression, ranging from depressive symptoms to disorder, among children and adolescents from diverse cultural backgrounds (for review, see Hankin, Snyder, & Gulley, in press). Additionally, research investigating the emergence, consolidation, and developmental origins of these cognitive risks suggests that by early adolescence they stabilize into relatively enduring, trait-like risks to depression (Hankin, 2008). Various environmental (e.g., IPV, poor parenting) and genetic factors can contribute to the development of these social–cognitive risks (Hankin et al., in press).

In addition, information processing biases have been investigated as correlates of youth depression. Attentional bias to negative emotion, especially sad and angry faces, is linked with clinical depression (Hankin, Gibb, Abela, & Flory, 2010) and may be engendered by negative parenting styles and behaviors (Gulley, Oppenheimer, & Hankin, 2014). Other cognitive and information processing risks, including executive function (EF) impairments and aspects of memory processes (e.g., autobiographical memory), have been shown to be disrupted concurrently among depressed youth (Hankin et al., in press). However, unlike the evidence with social–cognitive risks as vulnerability based on prospective designs, the majority of the evidence regarding information processing biases, EF, and memory has come from cross-sectional designs.

**Interpersonal Influences**

This review focuses attention on interpersonal risks (see Chapter 14), including excessive reassurance seeking, social support, and attachment patterns, that can be characterized as manifesting themselves within the individual. Other interpersonal relationships, including those with parents and peers, are not covered.

Excessive reassurance seeking (ERS) involves persistently seeking assurances from others that you are loveable and worthy, even when such assurance has been provided. Cross-sectional studies show that higher levels of reassurance seeking are associated with higher levels of depressive symptoms in both children and adolescents (Starr & Davila, 2008). Few longitudinal studies have been conducted (Starr & Davila, 2008), but some show that ERS does not predict prospective increases in symptoms and may be a concomitant of depression rather than a vulnerability (Oppenheimer, Technow, Hankin, Young, & Abela, 2013).

Social support is a multidimensional concept that is defined as the availability of a network of people on whom a person can rely in times of need. Adolescent depression is related to lower levels of family support (Sheeber, Hops, Alpert, Davis, & Andrews, 1997). Longitudinal research shows that adolescents’ perceptions of low parental support predicted future depression, whereas initial depression predicted decreased peer support (Stice, Ragan, & Randall, 2004); so it is clearly important to consider who provides the support. Longitudinal research shows the importance of carefully considering, measuring, and analyzing the buffering effects of social support and developmentally relevant stressors in predicting prospective changes in depressive symptoms. Positive parental support can protect against depression-eliciting effects of peer stressors (Hazel, Oppenheimer, Technow, Young, & Hankin, 2014).

Attachment patterns are thought to derive primarily from the quality and the quantity of contact a child has with his or her caregivers. Sensitive and responsive parents are likely to have children with secure attachment, whereas anxious and avoidant attachment patterns can result and serve as risk to multiple psychopathologies, including depression. Several cross-sectional (Davila, Ramsay, Stroud, & Steinberg, 2005), as well as longitudinal (e.g., Abela et al., 2005; Lee & Hankin, 2009), studies have demonstrated that attachment insecurity is associated with depressive symptoms in adolescent samples and that this relation may be mediated by cognitive risks as well as interpersonal stress generation processes.

**Summary of Risk: Multilevel Integrative Example Synthesizing Risk**

It is clear that stressful environments precede and contribute to increases in youth depression, and many vulnerabilities can potentiate this link. But it is doubtful that any single vulnerability will prove necessary or sufficient to account for the heterogeneity and varied developmental pathways leading to depression. A complete causal understanding of the development of depression across the lifespan will likely involve a multifaceted, integrative, developmentally sensitive vulnerability–stress approach in which vulnerabilities are coherently integrated across multiple levels of analysis. As but one example of this approach, Hankin (2012) articulated a multiple levels of analysis model of vulnerabilities to depression that integrates accessible, observable
factors (e.g., cognitive and temperament risks), intermediate processes and endophenotypes (e.g., information processing biases, biological stress physiology, and neural activation and connectivity), and genetic influences. Available evidence suggests that these various levels connect together (e.g., genetics with information processing biases and neural connectivity; biological risks with cognitive vulnerabilities) and each of these connections is linked to youth depression.

**Future Directions**

Among many potential future directions in the inquiry in depression among children and adolescents, this section focuses on four cross-cutting translational issues. First is investigating the degree to which the risk factors and etiological models presented and reviewed herein are specific to depression or represent more transdiagnostic predictors of broader-based psychopathology. The degree of specific, relative to transdiagnostic, prediction may also depend on which level is considered based on more recent hierarchical structural models of psychopathology (e.g., general internalizing; distress vs. fear-based disorders; depression vs. social phobia; Lahey, Applegate, Waldman, Hankin, & Rick, 2004).

Second is a continued emphasis on etiological, multiple levels of analysis models (e.g., Hankin, 2012) that highlight which coherent risks can be the focus of targeted intervention. Such work can inform the development of new, or the refinement of existing, evidence-based interventions.

Third is identifying those youth at elevated levels of risk, using evidence-based assessment methods and practical, clinically relevant assessment procedures for those youth who are most susceptible to a chronic trajectory of recurrent and chronic depression, beginning in childhood and adolescence, that would persist over the life-course. Given that most individuals' depression remits even without treatment, and not all individuals experience relapse, an evidence-based assessment of risk can focus needed clinical resources on those individuals at greatest risk for this chronic depression trajectory profile over time. For example, initial prospective research shows that youth with a history of depression who experience dependent stressors over time tend to exhibit persistently higher depressive symptoms (Technow, Hazel, Abela, & Hankin, 2015). Also, youths' prior minor depression and parental depression history predict recurrent episodes (Pettit, Hartley, Lewinsohn, Seeley, & Klein, 2013).

Last is the development and testing of individualized depression interventions, including treatments and prevention programs, based on matching and targeting evidence-based interventions to particular youths' vulnerabilities. Several evidence-based interventions for youth depression have been developed and been demonstrated as efficacious in randomized clinical trials [e.g., cognitive–behavioral therapy (CBT), interpersonal therapy (IPT), pharmacotherapy], yet a significant minority of depressed youth does not show clinically significant improvement. A promising future direction to improve upon this present modest treatment efficacy is personalized intervention. Initial findings suggest the capacity of such an approach. For example, adolescents who exhibit higher levels of hopelessness were significantly more likely to benefit from a CBT-based depression prevention (Gillham et al., 2012). Similarly, consideration of moderators (e.g., comorbid conditions, IPV history, hopelessness) can improve efficacy of standard evidence-based treatments for adolescent depression and should be considered in clinical decision making based on a personalized treatment approach (Asarnow et al., 2009).

**Conclusions**

Depression among children and adolescents is a serious mental health concern that contributes significant distress and impairment to the youth, their families, and their friends. There are clear developmental issues that need to be explicitly considered when studying, assessing, and treating depression among children and adolescents; knowledge of assessment methods, vulnerabilities, theoretical models, and treatments cannot simply be extended downward from the adult literature. Instead, a developmental psychopathology perspective is essential for advancing knowledge of youth depression in ways that can make a significant impact in reducing the risk for a potentially lifelong, chronic trajectory leading to recurrent episodes of depression over the lifespan. Such developmentally sensitive knowledge, especially when considered from and integrated into a multiple levels of analysis perspective, can be used to meaningfully inform the creation of new and the refinement of present evidence-based interventions, both treatment and prevention, to bend trajectories of depression risk and episode occurrence when these patterns have been demonstrated to occur throughout childhood and adolescence.
References


