



Review Article

Unpacking adolescent depression: A narrative review from a transdiagnostic perspective using the dimensional RDoC framework

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ABSTRACT

Background: Adolescent depression is a unique presentation that is highly heterogeneous, yet few studies have utilized longitudinal multimodal assessment approaches to develop a lifespan-oriented view of the etiology, course, and severity of this condition. Consequently, diagnostic criteria for mood disturbances may disadvantage adolescents by overlooking key developmental factors relevant to depression risk, delaying access to early interventions. This narrative review examines transdiagnostic factors of adolescent psychopathology, including sleep health and its effects on brain development and functioning on biological and psychosocial levels. Building on this work, a novel and dimensional framework of adolescent depression is proposed in the context of relevant Research Domain Criteria (RDoC) systems.

Methods: This review is based on scientific peer-reviewed publications, identified via keyword searches from Google Scholar, PubMed, ProQuest, and Connected Papers.

Results: There is some evidence to support a bidirectional relationship between sleep health and risk of depression development in adolescents. Furthermore, hormonal changes during adolescence and genetic risk for developing depression may specifically influence neuronal architecture and functioning underlying depressive presentations.

Limitations: Participant samples of chosen studies varied in terms of adolescent age ranges, with data on adolescent depression and associated factors being largely correlational.

Conclusions: The reviewed literature provides support for a multimodal approach to characterize adolescent depression, yet few studies have utilized quantifiable, transdiagnostic constructs within the RDoC systems. Developmentally oriented methodological designs and constructs, such as pubertal timing, should be emphasized to tie underlying biological mechanisms and sleep health to adolescent risk and resilience over time.

1. Introduction

Depression occurring during the period of adolescence is a poorly understood psychiatric condition that can manifest in diverse ways. Depressive symptoms are some of the most common psychiatric issues experienced in adolescents, with symptoms reaching clinical significance for 25% of adolescents worldwide in 2020 (Racine et al., 2021). Youth can experience a variety of preclinical depressive symptoms

before meeting the criteria for clinical depression. Vulnerabilities and preclinical features are often specific to this developmental age period during which depression, including its prodromal symptoms of depressed mood and loss of interest, is not a monolithic disorder, but rather, is a nuanced condition that is influenced by puberty and the dynamic interactions of psychological, environmental, and biological factors (Chahal et al., 2020; Dahl and Lewin, 2002). These nuanced features may not be adequately captured by categorical classification

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systems that typically require the presence of symptoms that are more commonly experienced by adults to be diagnosed, e.g., anhedonia, concentration difficulties, and loss of interest (Rice et al., 2019), and fail to account for adolescent depressive symptoms or constructs such as perceived loneliness and self-hatred (Manfro et al., 2023).

To address this gap, this narrative review will unpack adolescent depression by employing a transdiagnostic perspective and integrating literature on social and psychological factors with biological factors, brain development, and sleep. Attempting to review adolescent psychopathology along the depression symptom continuum, we specifically highlight the role of sleep as a developmentally sensitive, modifiable risk marker. Sleep has been found to be a significant risk factor for depressive symptoms (Jääkallio et al., 2024; Marino et al., 2022). Sleep is a fundamental, ubiquitous process that sustains positive mood, attention, and optimal cognitive functioning for adolescents, influencing academic success and the perceived quality of interpersonal relationships (Åslund et al., 2023; Lo et al., 2016; Urrila et al., 2017). Experiencing a combination of later bedtimes, shorter sleep duration, and poor perceived quality of sleep over time may increase negative affect and impact cognitive ability, putting adolescents at unique risk of developing depressive symptoms (Åslund et al., 2023; Bauducco et al., 2024; Lemke et al., 2023). Sleep and biological factors (Chiang et al., 2019) interact with psychosocial stressors, such as academic stress (Yan et al., 2018), early life adversity (Zajkowska et al., 2021), and peer interactions (Wang, et al., 2023) to increase risk for depression (Dahl and Lewin, 2002). Some evidence suggests that pre-existing sleep problems increased the vulnerability to psychological distress and risk for the development of depressive symptoms during the COVID-19 pandemic (Kiss et al., 2022). Emerging young adults with a short sleep duration before the pandemic, for example, had a greater increase in pandemic-period depressive symptoms (Alzueta et al., 2023). As adolescent depression is associated with reduced quality of life (McLeod et al., 2016; Zisook et al., 2007), suicidal ideation (Gijzen et al., 2021), substance use (Schuler et al., 2015; Wilkinson et al., 2016), and difficulties with educational attainment (Clayborne et al., 2019), peers, and family life (Breslau et al., 2017), understanding the transdiagnostic features that mark vulnerabilities and subthreshold symptoms may inform timely intervention and the prevention of clinical depression and lifetime consequences.

The goal is to understand adolescent depression from a multifaceted lens using transdiagnostic systems such as the National Institute of Mental Health's (NIMH) Research Domain Criteria (RDoC) framework (Dell'Acqua et al., 2023; Insel et al., 2010). Accurately assessing for depression in adolescence is complicated by the heterogeneity, severity, comorbidity, and duration of symptoms (APA, 2022; Cai et al., 2020; Chahal et al., 2020; Rice et al., 2019). Adding to this is the fact that adolescence itself is a time of dynamic change across multiple domains including biologically-driven changes in sleep, cognitive and psychosocial development. The trajectories of depression in adolescence are further influenced by pubertal processes due to hormonal and genetic factors (Rice et al., 2019b; Stumper and Alloy, 2023). Also, increased brain plasticity during adolescence (Laube et al., 2020; McLaughlin and Gabard-Durnam, 2022; Piekarski et al., 2017) links neurobiological sensitivity to the onset of depression symptoms in some youth, potentially underlying changes in the brain's frontal lobe cortical thickness (Bos et al., 2018) and cortico-striatal circuit connectivity and activity during reward processing (Ladouceur et al., 2019; Rappaport et al., 2020).

As multiple factors intersect to influence adolescent depression, this review will utilize a transdiagnostic lens using the RDoC framework. We briefly introduce categorical classification systems, describe a profile of adolescent depression and how the research domain criteria can be utilized in a dimensional transdiagnostic approach. We next review the literature and provide a model for how sleep, biological, and psychosocial constructs are related to depression in youth. The model includes the use of targeted interventions to improve youth outcomes that may be

especially pertinent as adolescence serves as unique period of opportunity to address vulnerabilities related to mood and sleep changes. The findings of this review will illuminate the complexities of adolescent depression to assist in developing more effective preventive and early intervention strategies in clinical, familial, and educational settings.

2. Methods

We conducted a search of the literature on adolescent depression and variables relevant to the RDoC Matrix. Relevant studies were identified through exploratory searches of Google Scholar, Connected Papers, ProQuest, and PubMed, and supplemented by manual reference-list screening of key articles. Searches for articles were conducted from March to November 2024, and key search terms included combinations of keywords and synonyms related to “adolescence”, “depression”, “mood”, “sleep”, “RDoC” and associated terminology with biological (e.g., “genetics”, “puberty”, “epigenetics”, “hormones”, “neural plasticity”, “DMN”, “SN”, “CCN”), cognitive (e.g., “rumination”, “internalization”, “executive functioning”), and social factors and outcomes (e.g. “early life stress”, “familial depression”, “COVID-19”, “development”, “depression risk”, “treatment outcomes”). Search terms were based on the main concepts of the review topic, and terminology and search combinations varied across databases to optimize relevance and retrieval of literature. Articles published from 2014 to 2024 were prioritized, although earlier papers were included where relevant. Studies that reported utilizing “adolescent” aged samples or data were included, allowing variability in included participant ages. Studies pertaining to adolescent depression in the reviewed literature included youth samples assessing for risk and vulnerability factors during development, as well as the presence and severity of subthreshold depressive symptoms (often referred to generally as ‘depression’) and current or prior clinical diagnoses, such as Major Depressive Disorder (MDD). Multiple studies also assessed for the presence of other psychiatric diagnoses and comorbid symptoms in their samples, yet the main focus of this review is on depression. Depression studies were excluded with only adult (aged 18 and older) samples and data. Conflicting evidence and discrepancies in the literature were interpreted in the context of their sample characteristics, methodology, and relevance to review objectives. As this was a narrative review, exhaustive search strategies and predefined inclusion/exclusion criteria in alignment with a formal systematic review methodology were not applied. The aim of this review was to provide a focused and interpretive synthesis of the available literature rather than a comprehensive systematic assessment. Table 1.

3. Diagnostic orientation to adolescent depression

3.1. Categorical classification of adolescent depression

Depressive disorders are commonly defined as a cluster of psychiatric conditions characterized by sad, empty, or irritable mood states (APA, 2022; WHO, 2022). Through the use of categorical diagnostic classification systems, such as the Diagnostic and Statistical Manual of Mental Disorders (5th ed., text revision; DSM-5-TR; American Psychiatric Association [APA], 2022) and the International Classification of Diseases and Related Health Problems (11th ed.; ICD-11; World Health Organization [WHO], 2022), extensive heterogeneity of symptoms and disorder comorbidity has been documented (Avenevoli et al., 2015; Micheline et al., 2021). For example, anxiety disorders are highly comorbid with adolescent MDD (Avenevoli et al., 2015; Korczak and Goldstein, 2009) and depressive symptoms (Costello et al., 2003), suggesting that categorical criteria for youth depressive disorders are ill-defined and overlap with symptoms categorized to other psychiatric conditions. Variability in adolescent depression presentation can potentially increase youths' chances of a depressive episode being overlooked or not being taken seriously to warrant intervention if symptoms are considered subthreshold, which then may increase a

Table 1

Sources used for review.

- *Google Scholar*. Keywords: adolescence, adolescent, depression, sleep, genetics, dimensional, rumination, DMN, SN, CCN, neural plasticity, internalization, hormones, epigenetics, puberty, depression risk, RDoC, negative valence system, positive valence system, arousal and regulatory system, cognitive systems, social processes.
- *Connected Papers*. Keywords: adolescence, adolescent, depression.
- *ProQuest*. Keywords: adolescent, depression.
- *PubMed*. Keywords: adolescent, depression, sleep, executive functioning, salience network, default mode network, neural plasticity, depression risk, treatment outcomes, circadian rhythms, chronotype, mood, sleep quality, early life stress, familial depression, COVID-19, pandemic, development, RDoC.

youth's risk for meeting diagnostic criteria for a depressive disorder in adulthood (Bertha and Balázs, 2013; Naicker et al., 2013). Therefore, alternative methods of assessment are necessary to accurately identify depressive symptoms for adolescents as a form of early psychiatric intervention.

3.2. A profile of adolescent depression

Manifestations of depression appear to vary based on age: adolescents and adults have different prevalences of depressive symptoms. A combination of irritability (Bernaras et al., 2019), loss of energy, insomnia, and appetite and weight changes were observed more frequently in youth diagnosed with major depression, compared with adults, who are typically characterized by anhedonia (i.e., loss of interest and pleasure) and concentration difficulties (Rice et al., 2019). Depressive symptom network approaches to psychopathology further reveal that perceived loneliness, pessimism, and self-hatred are prevalent in depressed adolescents aged 13–19 years (Mullarkey et al., 2019), with loneliness being directly related to affective depressive symptoms and mediating somatic symptoms (Grygiel et al., 2024). Similarly, adolescent-onset depression diagnoses heighten the risk of recurrent depression in adulthood compared to childhood-onset depression diagnoses (Copeland et al., 2021). As such, adolescent-onset depression presents with its own prognostic outcomes and warrants further diagnostic consideration as an age-specific condition. The transition from the DSM-IV TR to the DSM-5 attempted to account for variability in depressive symptomology found in children and adolescents while reducing the over-diagnosis of bipolar disorder by introducing disruptive mood dysregulation disorder (DMDD) as a depressive disorder in its 2013 revision (DSM-5-TR; Rodríguez-Testal et al., 2014; Roy et al., 2014). After its introduction, the prevalence of DMDD increased annually in association with the clinical use of mood disorder not otherwise specified (NOS) in youths (Le et al., 2020), indicating an ongoing and dynamic impact in conceptualizing depression in early life. However, the prevalence of DMDD was found to significantly decrease from childhood to adolescence (Althoff et al., 2016), suggesting that depression in adolescence presents unique features that are different from child- and adult-onset depression. Accordingly, ongoing diagnostic efforts would benefit from separately targeting child-, adolescent-, and adult-onset depression to address unique dimensional characteristics of depression across age ranges. Furthermore, categorical systems lack information on neurodevelopmental underpinnings of adolescent depression.

3.3. Research domain criteria (RDoC)

In contrast to categorical diagnostic systems, NIMH's RDoC framework offers a transdiagnostic perspective of adolescent depression. It focuses on brain-behavior relations by examining constructs (i.e., processes, mechanisms, behavioral elements, and responses of human experience) across major domains of human functioning: Arousal/Regulatory; Social Processes; Cognitive; Positive Valence; and Negative Valence Systems (see Appendix Table 1; NIMH, n.d.). The major domains in the framework are designed to correspond to a multidimensional view of human behavior and development, including cognitive processes (Cognitive Systems), responses to interpersonal settings (Social Processes), arousal and homeostatic regulation (Arousal and

Regulatory Systems), and responses to situations that are aversive (Negative Valence Systems) or positive and motivational (Positive Valence Systems) (NIMH, n.d.). The Sensorimotor domain was added later to highlight the role of disruptions in motor systems within overall psychopathology (NIMH, n.d.) but is not further discussed in this review on adolescent depression. Domain constructs are measured via units of analysis that tend to be neurobiological (i.e., molecules, cells, genes, neural circuitry, and physiology) but also include observable behaviors, self-reports, and experimental paradigms (Glenn et al., 2017; NIMH, n.d.). Thus, psychopathological issues in adolescence are viewed as forms of neurodevelopmental disorders (Casey et al., 2014) likely influenced by disruptions in neurocircuitry (Insel et al., 2010), subsequently creating a cascade of disruptions across multiple units of analysis (Woody and Gibb, 2015). These units of analysis provide rich explanations about the complexity of psychopathology compared to traditional diagnostic categories (Insel et al., 2010), especially in the context of depression (Dean and Keshavan, 2017) and how brain circuits involved in the constructs, such as of 'loss' or 'acute threat' within Negative Valence Systems, are implicated in the presentation of depression in youth (Henje Blom et al., 2014).

The organization of RDoC fits well with a data-driven approach to determine patterns of risk in symptoms of depression using multiple measures, as seen with machine learning models that have been used to identify longitudinal patterns of overlapping symptoms between MDD and the RDoC negative valence construct (i.e., sadness, fear, anxiety, loss, threat) (Paschali et al., 2022). The Arousal/Regulatory System of the RDoC Matrix warrants particular mention when researching adolescent depression development, as its constructs of circadian rhythms, arousal, and sleep-wakefulness can be measured by units of analysis corresponding to affective states, mood, fatigue, and disruptions in sleep length and quality (NIMH, n.d.). As depressed adolescents demonstrate a unique clinical presentation characterized by insomnia, irritability, and loss of energy (Bernaras et al., 2019; Rice et al., 2019), investigating the role of impaired sleep in depression provides a mechanistic understanding of biological features linked to the Arousal/Regulatory System during adolescence. However, the RDoC units of analysis for sleep-wakefulness (e.g., sleep quantity, timing, and wakefulness) and circadian rhythm constructs (e.g., chronotype and morningness-eveningness preference, longitudinal actigraphy) within the Arousal and Regulatory Systems (Micheline et al., 2021) are under-studied in youth depression. Several longitudinal studies show that sleep disturbance increases risk for depression (Lovato and Gradisar, 2014). Of the studies that have examined units of analysis of sleep-wakefulness, eveningness preference predicted subsequent depression, and chronotype was significantly associated with depression symptoms (Bauducco et al., 2024; Lemke et al., 2023), supporting the formulation that sleep is a modifiable behavior as an intervention target to reducing risk for depression (Blake et al., 2019; Blake and Allen, 2020; Lovato and Gradisar, 2014; Orchard et al., 2020).

4. Sleep

Prior to reviewing the literature on sleep and depression, it should be noted that there are differences in the methodological approaches to the study of sleep quality. These include both subjective and objective measures. Subjective measures of sleep range from validated questionnaires such as the Pittsburgh Sleep Quality Index (PSQI, Buysse et al.,

1989), Pediatric Sleep Questionnaire (PSQ, [Chervin et al., 2000](#)), the Insomnia Severity Index (ISI, [Bastien et al., 2001](#)), specifically for measuring insomnia symptoms, or Sleep Disturbance Scale for Children (SDSC; [Bruni et al., 1996](#)) among others, to daily sleep diaries assessing daily variation of perceived sleep. The gold-standard objective measurement of sleep is polysomnography (PSG), which relies on electroencephalographic recordings of brain activity combined with electro-oculographic and electromyographic measurements of eye and muscle activity, respectively, for precise wakefulness and sleep stage differentiation between N1, N2, N3, and rapid eye movement (REM) sleep, and their distinct pattern of change in sleep cycles across the night (e.g., [Baker et al., 2016](#); [de Zambotti et al., 2022](#)). PSG has been applied to study sleep architecture in adolescence, such as changes during puberty or with increasing age ([Baker et al., 2022](#); [de Zambotti et al., 2015](#); [Scholle et al., 2011](#)). PSG is highly involved for the participants and clinician/researcher alike and is labor-intensive. It requires trained overnight personnel, adequate laboratory equipment, and clinical sleep diagnostic expertise. Typically, PSG recordings are made in a sleep lab and include only a couple of nights of recording. As such, PSG is not well-suited for tracking changes in sleep quality or continuity from night-to-night. In contrast, research-grade actigraphy (e.g. wrist-worn accelerometers) and commercial multisensor wearables (e.g., those placed on wrist or finger that include photoplethysmography sensors for detecting pulse rate in addition to accelerometry sensors for detecting movement) can be used to track sleep-wake activity objectively over many nights in the home environment. Actigraphy and wearable sleep technology have good performance for detecting sleep, although their performance in detecting wake is poorer, compared with PSG ([de Zambotti et al., 2024](#)). In addition, it should be kept in mind that commercial wearables often rely on proprietary algorithms, and their limitations should be acknowledged when used in research or clinically ([de Zambotti et al., 2024](#)). A major advantage of actigraphy or wearable sleep technology is that the devices can be used to track sleep objectively over a long time period, which represents an important advance in objective sleep assessment, including tracking night-to-night variability in sleep, that can complement self-report measures. Self-report assessments of sleep with diaries and questionnaires are easy to assess, function as screening tools, and can provide clinically relevant information. Questionnaires represent an important aspect of subjective sleep quality, with diaries further adding information on the daily variation of perceived sleep. Subjective sleep measures can be particularly relevant to mood, along the positive and negative valence RDoC features ([NIMH, n.d.](#)). Objective measure from actigraphy and PSG recordings may tap more into the arousal/regulatory aspects of physiological sleep health and be able to evaluate nuanced biologically-driven vulnerabilities related to puberty and adolescent depression. Here, using a trans-diagnostic RDoC approach can explain why objective actigraphy and PSG sleep quality assessments may not necessarily overlap with subjective self-assessments of sleep quality ([Kiss et al., 2025](#)), and that they can differ across adolescence and along the depression continuum ([Biddle et al., 2015](#)).

4.1. Sleep and depression

Sleep is a universal, physiological process imperative to physical and mental wellness ([Firth et al., 2020](#); [Medic et al., 2017](#); [Yang et al., 2022](#)). Healthy sleep, as characterized by getting enough sleep, at appropriate times, and in the absence of sleep disturbance ([Paruthi et al., 2016](#); [Orchard et al., 2020](#)), is critical for optimal mood, cognitive function, and behavioral regulation ([Gregory and Sadeh, 2016](#); [Kelly et al., 2022](#)). Due to the fundamental need for healthy sleep to ensure normal human functioning, the relationship between sleep difficulties and the onset and severity of psychopathology during adolescence is an active area of research ([Alvaro et al., 2017](#); [Åslund et al., 2023](#); [de Bruin et al., 2018](#)). Many studies have examined how a broad cluster of ‘sleep problems’ relate to and predict depressive symptoms ([Gregory and Sadeh, 2012](#);

[Ho et al., 2022](#)). For example, poor sleep quality disrupts brain functioning of reward pathways via reduced ventral striatum activation to reward anticipation and receipt of feedback ([Hasler et al., 2012](#); [Holm et al., 2009](#)). Variability in sleep can impact white matter development ([Telzer et al., 2015](#)), synaptic homeostasis ([Tononi and Cirelli, 2006](#)), and neural response and network efficiency ([Robinson et al., 2018](#); [Tashjian et al., 2018](#); [Telzer et al., 2013](#)), indicating that sleep is crucial for brain development and maintenance of brain plasticity across the lifespan ([Graven and Browne, 2008](#)). Accordingly, experiencing a combination of later bedtimes, shorter sleep duration, and perceived poor quality of sleep may increase negative affect and deficits in cognition, putting adolescents at unique risk of developing depressive symptoms ([Åslund et al., 2023](#); [Bauducco et al., 2020, 2024](#); [Lemke et al., 2023](#)), altogether highlighting sleep as a focal risk factor ([Jääkallio et al., 2024](#); [Marino et al., 2022](#); [Medic et al., 2017](#); [Newton et al., 2019](#)).

4.2. Sleep mechanisms of risk in adolescent depression

Mechanisms of how disturbed sleep increases risk, and conversely, how healthy sleep protects against adolescent depression are multifactorial, including shared genetic roots ([Dueck et al., 2017](#); [Gregory et al., 2009](#); [Tahmasian et al., 2020](#)), atypical interactions between circadian and sleep processes ([Jääkallio et al., 2024](#); [Scott et al., 2021](#)), and overlap between neural regulatory systems for sleep, arousal and affect ([Casement et al., 2016](#); [Dahl, 1996](#); [Galván, 2020](#)). Sleep patterns undergo considerable change during adolescence due to factors that are biological (e.g., normative delays in circadian phase, slower collection of homeostatic sleep pressure) and environmental (e.g., early school start times, extracurricular activities, academic, social, and cultural pressures) factors, leading to eveningness preference clashing with early school start times and reducing time for sleep ([Colrain and Baker, 2011](#); [Crowley et al., 2018](#); [Knutson and Lauderdale, 2009](#)). Multiple lifestyle factors and experiences can impact adolescents’ quality of sleep, including exercise, caffeine intake, and electronic display use ([Gillis et al., 2021](#); [Paulich et al., 2021](#); [Tomanic et al., 2022](#)). As sleep plays a key role in the formation of memories ([Walker and Stickgold, 2006](#)) and emotion regulation ([Deliens et al., 2014](#)), disruptions in sleep have been associated with lower positive affect ([Short et al., 2020](#)), rumination ([D. Jamieson et al., 2021](#)), and poor emotion regulation ability to negative affect ([Palmer et al., 2018](#)). As such, sleep loss and adolescent depression may mutually interact through multiple avenues to impact executive functioning ([Ji et al., 2021](#)) and emotional processing ([Short et al., 2020](#)) via dysregulation of brain processes underwriting top-down emotion regulation behavior ([Jamieson et al., 2020](#)).

4.3. The directionality of sleep and adolescent depression

As in adults, sleep disturbances in adolescents are noted to be highly comorbid with depression ([Asarnow and Mirchandaney, 2021](#); [Roberts and Duong, 2013](#)), with nearly 73% of adolescents presently experiencing MDD also experiencing sleep disturbances ([Liu et al., 2007](#)). Evidence from longitudinal studies suggest sleep problems as a vulnerability factor for developing depressive symptoms ([Gregory et al., 2009](#)) and diagnoses in adolescents ([Orchard et al., 2020](#); [Roberts and Duong, 2014](#)). Data from the Adolescent Brain Cognitive Development (ABCD) Study cohort showed that sleep disturbance at 9–10 years old, while controlling for baseline mental health symptoms, predicted depressive symptom scores at Year 1 follow-up ([Goldstone et al., 2020](#)), supporting prior literature suggesting that sleep problems predict depression symptoms ([Gregory et al., 2009](#); [Orchard et al., 2020](#); [Roberts and Duong, 2014](#)). Furthermore, sleep may impact internalizing symptoms (e.g., anxiety, depression) in adolescents by influencing self-control (i.e., executive control; [Ye et al., 2024](#)), suggesting that sleep affects depression through complex cognitive processes. However, the directionality and strength of the relationship between sleep disturbances and

depressive symptom onset remain debated upon within the current literature and reflect age-based variations (Gregory et al., 2009; Marino et al., 2022).

In a pre- to young- adolescent sample, sleep problems at age 8 years were found to predict depression at age 10 years, yet the inverse was not found (Gregory et al., 2009). However, another study found bidirectional associations between depressive symptoms and disturbed sleep in child (5–8 years old) and early adolescent samples (10–12 years; Marino et al., 2022). Comparatively, a slightly older adolescent sample (12–13 years) demonstrated that depression symptoms were modestly associated with sleep only, supporting evidence for a unidirectional relationship (Marino et al., 2022). While both studies utilized cross-lagged statistical models to assess developmental links between depression and sleep, discrepancies in directional findings may be attributable to differences in methodology and participant samples. Notably, Marino and colleagues (2022) examined sleep-depression links within a much larger child ($n = 1689$) and adolescent ($n = 1113$) study sample than Gregory and colleagues (2009; $n = 300$). More notably, both studies stratified their age samples for statistical modeling differently, with Gregory and colleagues (2009) including a child-to-early adolescent sample (ages 8–10) within one statistical model, and Marino and colleagues (2022) investigating a wider span of development (ages 5–17) by conducting two separate models for children (ages 5–8) and adolescents (ages 10–17). Accordingly, while methodological variations in definitional age ranges likely influence the directionality and strength of sleep-depression associations, adolescence is still implicated as a vulnerable period for understanding longitudinal interactions between sleep and depression. This relationship may specifically present as bidirectional in adolescents (aged 11–17 years), with reduced quality of sleep being associated with a three-fold higher odds of meeting MDD criteria, and the presence of a major depressive episode (MDE) within the last 12 months being associated with a higher likelihood of sleep deprivation (<6 h of sleep on weeknights) (Roberts and Duong, 2014). These findings were still relevant after controlling for age, family income, gender (Roberts and Duong, 2014), further cementing the notion that sleep and the presence of depression symptoms are longitudinally bidirectional (Bai et al., 2020).

4.4. Effects of sleep interventions on depression

Targeting sleep difficulties can serve as a practical and promising point of intervention when conceptualizing and treating adolescent depression, especially because sleep interventions appear to be well-received amongst adolescents (Blake et al., 2016). Adolescents diagnosed with depression may self-report lower sleep quality compared to other youths (e.g., less sleep, later sleep onset; Orchard et al., 2020), making subjective sleep improvement an attractive variable to examine changes in adolescent depression research and interventions as it can be easily assessed daily (Blake et al., 2017). A clinical trial of sleep hygiene and sleep extension in adolescents with short sleep was found to improve their sleep quality and reduce depressive symptoms (Dewald-Kaufmann et al., 2014). Also, cognitive behavioral interventions, like Cognitive Behavioral Therapy for Insomnia (CBT-I), have been found to improve behavioral problems (Blake et al., 2017), sleep quality, anxiety, and chronic pain in adolescents with baseline sleep problems and mood symptoms at a clinical or subclinical level (Åslund et al., 2020). CBT-I may be especially effective for adolescents when incorporating age-related adaptations in its protocol, including the use of smart phone apps to reduce rumination (Sleep Ninja; Li et al., 2024), and age-appropriate psychoeducational materials about poor sleep and pre-bed electronic use (Clarke et al., 2015). Of note, CBT-I was specifically found to alleviate symptoms of both depression and anxiety (Åslund et al., 2020), while a novel virtual-reality sleep intervention reduced signs of physiological arousal associated with adolescent insomnia symptoms (i.e., heart rate, cortisol; de Zambotti et al., 2022). As shorter total sleep time and greater sleep disturbance at ages 9–10

have been found to significantly predict later depression symptoms during adolescence (Goldstone et al., 2020), sleep interventions may serve as unique paradigms to research adolescent depression through RDoC Arousal/Regulatory and Negative Valence Systems constructs (e.g., Arousal, Sleep-Wakefulness, Acute Threat; NIMH, n.d.). By further incorporating sleep interventions in investigations of adolescent depression through RDoC domains, multimodal methods may improve depressive diagnostic accuracy and better reach adolescents who would benefit from such a comprehensive and effortful intervention.

5. Biological considerations of adolescent depression

5.1. Adolescent developmental plasticity

Neural plasticity, as defined by the brain's ability to change and adapt its activation patterns and neuroarchitecture over time, is necessary for normative human development (Ismail et al., 2017; Sweatt, 2016). Neural changes during adolescence may account for the experience of depression in youths, as well as predispose them to depression in adulthood (Goff and Tottenham, 2015; Ho and King, 2021; Straub et al., 2019). Hormonal changes during this period are believed to incite increases and decreases in brain plasticity that impact development in various cognitive domains and neural circuits (Laube et al., 2020). This is achieved through plasticity mechanisms that are 'experience-driven' and promote adaptive learning with environmental exposure or are 'experience-expectant' and facilitate the input of environmental stimuli during developmental windows (Greenough et al., 1987; McLaughlin and Gabard-Durnam, 2022). Normative development is characterized by dynamic changes in subcortical and cortical regional volume and activity over time (Ostby et al., 2009) to enhance functional connectivity and neural signaling and transmission (Jolles et al., 2011). This functional refinement may be facilitated in adolescents via the increased pruning of excess synapses from childhood into adulthood (Tamnes et al., 2013; Huttenlocher and Dabholkar, 1997) in combination with increases and decreases in cortical white matter density (Perrin et al., 2009) and nonlinear changes in grey matter volume (Giedd et al., 1999). Synaptic pruning during pubertal maturation in adolescence is often reflected by reduced slow-wave sleep activity (SWA), as measured by electroencephalogram (EEG; Tarokh et al., 2016). Reductions in EEG power from pre- to post- puberty can be observed in resting and waking states, indicating that significant changes to sleep physiology during this time are closely linked to developmentally sensitive outcomes in youths (Tarokh et al., 2016). Furthermore, plastic effects on brain development have been heavily studied in the context of depression (Liu et al., 2017), with depressive symptoms in youths being associated with accelerated cortical thinning in the frontal lobe (Bos et al., 2018) and blunted striatal circuit activity in response to reward (Stringaris et al., 2015). Adolescence, therefore, is a critical window for intervention, which capitalizes on understanding pubertal processes and genetic influences to lessen the impact of neural vulnerability to depression.

5.2. Pubertal and hormonal changes

Puberty is a developmental process that facilitates sexual maturity, the onset of which triggers the activation of the hypothalamic-pituitary-gonadal (HPG) axis and results in the release of increased sex hormones such as estrogen and testosterone (Marceau et al., 2015). These hormonal shifts drive secondary sexual characteristics and impact brain development (Blakemore et al., 2010), notably in regions corresponding to decision-making, social processing, and emotional regulation (Vijayakumar et al., 2018). Early pubertal timing serves as a transdiagnostic risk factor for depression and is linked to internalizing symptoms in female individuals (Hamlat et al., 2019), such that female adolescents with a younger age of menarche were more likely to develop depression or experience greater depressive symptoms according to self-report measures (Hirtz et al., 2022). Indeed, transitioning to

adolescence is observed to increase depression rates amongst females (Costello et al., 2003). Female youth may specifically experience inter-correlated depressive symptoms and somatic complaints over time in relation to menstrual cycling (Beal et al., 2014), highlighting the unique role of hormonal fluctuations in adolescent depression risk. Hormones play an important role for mood management in adolescents and can influence depression prevalence during puberty. For example, elevated levels of androstenedione (a hormone involved in the production of testosterone and estrogen) have been observed in female adolescents with depressive symptoms (Hirtz et al., 2022). For male adolescents, high levels of estradiol (a hormonal product of androstenedione, estrone, and testosterone; Ishikawa et al., 2006; Ranganathan et al., 2019) were associated with depression symptoms (Chronister et al., 2021).

While hormonal changes in both male and female adolescents have been linked to the development of depression symptoms, there appear to be differential effects of sex-specific hormones on neural reward system circuitry between male and female youths (Chronister et al., 2021; Ladouceur et al., 2019). Reduced activity in the reward system's caudate and greater connectivity between the nucleus accumbens (NAcc) and putamen, associated with high levels of estradiol, have been observed in female adolescents, potentially underwriting symptoms of increased impulsivity and/or diminished feelings of pleasure (Ladouceur et al., 2019). This is further supported when considering the maturation of positive valence systems, in which NAcc activity in response to reward prediction errors decreases with age for female adolescents into young adulthood, with the inverse being found in male adolescents (Willinger et al., 2021). While cortico-striatal-thalamic network effective connectivity becomes more finely tuned with age (Willinger et al., 2021), sex-specific hormones likely are associated with changes in longitudinal reward processing and depression risk through early-in-life functional changes to positive valence system circuitry and activity. Cortisol, a hormone associated with stress response, can further influence the relationship between depression symptoms and the hormones testosterone and dehydroepiandrosterone (DHEA) in female youth (Chronister et al., 2021). As such, female adolescents may have increased risk of developing a depression diagnosis in early adolescence if they experience prolonged stress with simultaneously heightened levels of testosterone (Copeland et al., 2019), which may support non-linear 'peaks' in subcortical brain development (Vijayakumar et al., 2021).

Interactions between stress, environmental factors, and developmental hormones may appear differently for transgender and non-binary (TNB) youths compared to data on cisgender male and female adolescents, however these gender-minority populations appear to be under-represented in studies examining hormonal-level influences on depression (Nguyen et al., 2019). Hormonal research examining depressive symptoms in TNB youths, in contrast, appears to focus on the alleviation of psychosocial stressors and gender-dysphoria through the utilization of gender-affirming hormone therapy (GAHT) (also known as cross-sex hormonal therapy) (Achille et al., 2020; Chen et al., 2023; Green et al., 2022; Grannis et al., 2021; Kaltiala et al., 2020). Utilization of GAHT has been found to be associated with decreased depression severity, suicidality, suicide attempts, lower odds of experiencing depression, and less need for depression-specific psychiatric treatment in TNB populations (Grannis et al., 2021, 2023; Green et al., 2022; Kaltiala et al., 2020). Examinations of the effects of specific cross-sex hormonal interventions on psychopathology likewise focus on longitudinal trends in emotional and cognitive functioning within adult TNB populations (Nyugen et al., 2018; Nguyen et al., 2019), warranting further investigation in TNB youth.

5.3. Neuronal networks

Along with structural maturation, functional brain networks become more cohesive and defined throughout lifespan development, enabling

quick and specialized cognitive processing (Aboud et al., 2019; Gilmore et al., 2018; Goddings et al., 2019). Network changes allow for improved functional integration of information and increased capacity for higher-level cognition (Baum et al., 2020; Larsen and Luna, 2018), allowing adolescents to effectively engage in complex reasoning, abstract thinking, and deep reflection on information (Larsen and Luna, 2018). Atypical functional connectivity and activity patterns in intrinsic networks, such as the default mode network (DMN), cortical/cognitive control network (CCN), and the salience network (SN) have been implicated in youths with depression (Ho et al., 2015; Hou et al., 2024; Jacobs et al., 2014; Miller et al., 2015). Notably, intrinsic functional connectivity differences have been found to differentiate those with high versus low risk for depression and even predict depression onset in adolescents (Pawlak et al., 2022; Shapero et al., 2019).

The CCN has been found to aid in facilitating executive functioning (Breukelaar et al., 2017; Dwyer et al., 2014), while the DMN may promote self-referential thinking (Dalwani et al., 2014; Davey et al., 2016; Domakonda et al., 2019; Fuentes-Claramonte et al., 2019; Silveri et al., 2022) and memory processes (Chai et al., 2014; Müller et al., 2020; Patriat et al., 2016; Viard et al., 2019). Notably, the DMN may correspond with alterations in self-referential thinking in adolescent depression, as observed by its aberrant activation during the recall of negatively valenced autobiographical memories in a depressed youth sample (van Houtum et al., 2023). Accordingly, neurodevelopmental change in the CCN can be conceptualized through the Attention and Cognitive Control constructs of the RDoC Cognitive Systems, as attention is required to engage in executive functions pertaining to performance monitoring and goal and response selection (NIMH, n.d.). The DMN, in contrast, has been associated with self-referential thinking during rest (Davey et al., 2016), where DMN dynamically interacts with other networks underwriting declarative memory, perception and understanding of self, and worry and rumination across the Social Processes, Negative Valence, and Cognitive Systems. In tandem with weakened effective connectivity between the DMN and SN in adolescent MDD (Willinger et al., 2024), alterations in self-referential processing via an exaggerated self-focus likely result from network-wide functional changes in adolescent depression. Normative non-linear brain development during adolescence, as revied by Gogtay and Thompson (2010), likely compounds challenges depressed adolescents may face in effectively integrating emotional and self-referential stimuli across neural networks. Specifically, hormonal changes engender the early development of the limbic system (Crone and Dahl, 2012; Sisk and Foster, 2004) and protracted development of the 'socio-emotional' brain after puberty (Ahmed et al., 2015; Blakemore, 2008; Burnett et al., 2011). Similarly, the SN is predominantly implicated in detecting external and internal stimuli, which may promote deficits in behavior and self-awareness in depression (Ho et al., 2021; Schimmelpfennig et al., 2023; Webb et al., 2021).

5.4. Genetic heritability

Another way to potentially identify adolescent depression and approach early intervention is to consider high-risk factors, such as genetic predisposition. Genetic variations in the serotonergic 5-HTTLPR gene (5-HTTLPR) and neural plasticity brain-derived neurotrophic factor (BDNF) gene (Val66Met) appear to be heavily implicated in adolescent depression etiology (Xia and Yao, 2015), warranting consideration of the role of familial heritability. Familial studies reveal that children with a clinically depressed parent may be at increased risk of meeting criteria for a depressive disorder in their lifetime, with high familial genetic risk being associated with earlier onset and recurrence for major depression (Kendler et al., 2021; Mars et al., 2012). The presence of at least one biological parent with a history of recurring MDD or a diagnosis of MDD has also been linked to alterations in emotion and reward-processing neurocircuitry in their children (Gotlib et al., 2010) and may

constitute ‘high-risk’ status for MDD development. Adolescent cohort differences in familial MDD risk have revealed neuronal functional connectivity alterations in regions of the amygdala, dorsal striatum, lingual gyrus, fusiform gyrus, and postcentral gyrus (Fischer et al., 2022), regions involved in sensory perception, emotion, and salience processing. Furthermore, the presence of early and late adolescence-onset depressive symptoms has been associated with later risk for major depression (Rice et al., 2019b), with heritability of depression estimated to reach a heightened “peak” of risk at age 13 years for male adolescents and at age 18 years for female adolescents, regardless of pubertal status (Sallis et al., 2017). These findings imply that depressive symptom prevalence and related neuronal processes may be influenced by sex and age-related changes in the expression of depression-related genes during the period of adolescence (e.g., puberty onset; Sallis et al., 2017). Accordingly, understanding the intersection of familial genetic risk to depression, environmental influence on genetic expression (Alshaya, 2022; Sullivan et al., 2000), and puberty serves to further illuminate the nuances of adolescent psychopathology development.

5.5. Epigenetics of depression

Epigenetics refers to the study of long-standing changes in deoxyribonucleic acid (DNA) gene expression through transcriptional/post-transcriptional and translational/post-translational DNA mechanisms (Ochi and Dwivedi, 2023), which then affects the function and behavior of a given gene (Alshaya, 2022). Through an epigenetic approach, the nuanced influences that life stress can have on genetic predisposition and expression in the development of adolescent depression can be further explored and understood (Ämmälä et al., 2019; Shen et al., 2022; Talarowska, 2020). Epigenetic mechanisms, including noncoding ribonucleic acid (RNA) functions, histone and chromatin structure changes, and DNA methylation (DNAm), are neurodevelopmental processes occurring through interactions between genes and the environment to influence bodily functioning and health, such as in the context of depression development (Talarowska, 2020). These key epigenetic mechanisms have been heavily researched in animal models examining the expression of BDNF (Miao et al., 2020), an activity-dependent growth factor that is secreted in the brain and necessary for normative brain development in mammals (Park and Poo, 2013). The BDNF precursor (proBDNF) is secreted and converted into its mature form (BDNF) by enzymes, triggering a signaling cascade in the brain to regulate central nervous system functioning (Riffault et al., 2018). In humans, dysregulation of long non-coding RNA processes and over-methylation of BDNF genes are believed to reduce neural plasticity and increase susceptibility to depression development (Aljabali et al., 2024), while abnormal histone modification processes have been found to inhibit BDNF expression in rodents (Wu et al., 2022). Given that BDNF serum levels appear to differ between youth responders and non-responders diagnosed with MDD during treatment with selective-serotonin reuptake inhibitors (SSRIs), further investigating the role of BDNF in depression development during adolescence as a highly plastic developmental period is warranted. DNAm specifically allows for selective gene transcription (i.e., transcriptional silencing) necessary to support brain functioning and development across the lifespan (Yuan et al., 2024). Furthermore, stronger causal effects for DNAm to depression have been observed when considering polygenic risk, like the additive effect of genetic variance on gene expression (Shen et al., 2020; Talarowska, 2020).

Fluctuations in DNAm have been identified in male adolescents with disordered sleep and diagnosed depression (Ämmälä et al., 2019), and female adolescents endorsing MDD symptoms likewise were observed to have significantly lower levels of BDNF gene promoter methylation compared to an age-matched control group (Zwolińska et al., 2024). Following antidepressant treatment to target MDD symptoms in the female sample, methylation levels between the groups were no longer

statistically different than the non-depressed control group (Zwolińska et al., 2024), suggesting that pharmacological interventions can influence expressions of adolescent depression on an epigenetic level. Furthermore, daily interpersonal stress and sleep duration have been found to impact gene expression in adolescents (Chiang et al., 2019). These findings suggest that multi-level RDoC variables, such as sleep, environment, stress, and behaviors may interact with neural vulnerabilities of depression via genetic and familial depression risk to impact adolescent depression development. Accordingly, incorporating the RDoC framework into clinical research will enable nuanced neurodevelopmental and epigenetic insights into understanding adolescent depression development (Ämmälä et al., 2019; Shen et al., 2022; Zwolińska et al., 2024).

6. Psychosocial and environmental considerations of adolescent depression

6.1. Cognition and emotion in adolescent depression

Commonly identified cognitive symptoms of depression may be more prevalent in adults (e.g., concentration difficulties), yet adolescents show unique cognition-based predictors of depression that are associated with RDoC Negative Valence Systems, like difficulties with behavioral inhibition and cognitive flexibility (Paschali et al., 2022). Executive functioning is necessary for individuals to successfully plan, execute, and adapt goal-driven behaviors based on their internal and external environments (Kryza-Lacombe et al., 2021; Wagner et al., 2015). Disturbances in the development of executive functioning skills can manifest as increased impulsivity and ineffective emotional processing, which in turn have been related to self-harming behaviors and suicidality in depressed youths (Lockwood et al., 2017; McHugh et al., 2019; Robinson et al., 2019). Executive functioning abilities in youth have been related to frontal cortical development, where neurodevelopmental delays can impact risk-taking propensity in the context of emotion and reward processing (Casey and Jones, 2010; Wu et al., 2024), and behaviors that may become dysregulated in adolescent depression (O’Callaghan and Stringaris, 2019; Wu et al., 2024). Cognitive functioning, subsequently, may be posited as a risk factor for the development of adolescent depression symptoms within the RDoC Framework.

Cognitive and emotional aspects of depression in adolescents reflect constructs from the RDoC’s Positive Valence and Negative Valence Systems. The Positive Valence System underlies an individual’s engagement with positive environmental situations that promote achievement and reward attainment (Olino, 2018). Adolescent depression may affect the Positive Valence System by presenting as anhedonia (Olino, 2018), decreased positive affect, lower social activity, and failure to engage in reward-seeking behaviors (Forbes and Dahl, 2005). Comparatively, the Negative Valence System focuses on the processing of sadness, loss, threats, negative stimuli, and monitoring of errors (Kujawa and Burkhouse, 2017). In adolescents at increased risk of developing depression, this system may promote reduced emotion regulation and error-related negativity, and increased sensitivity to loss (Kujawa and Burkhouse, 2017). Indeed, error processing in loss-avoidance contexts appears impaired in adolescents diagnosed with MDD via aberrations in insular, habenular, and orbitofrontal cortex processing of loss feedback and loss outcomes (Willinger et al., 2022). Alterations in avoidance learning through functional changes in Negative Valence System neuronal correlates may serve as early mechanisms for the development of early-onset depression in adolescents (Willinger et al., 2022). Experiencing loss (e.g., separation from loved ones, loss of material possessions, jobs, career opportunities, health) has likewise been found to predict the onset of depression in adolescents and young adults (Asselmann et al., 2015). Integrating the Negative Valence System with the RDoC Social Processes domain is warranted, as affiliations, attachment, and understanding of others and self are encompassed in

this domain and are likely impacted during experiences of loss. Accordingly, dynamic interactions between the two RDoC valence systems can be utilized to illuminate the heterogeneity of adolescent depressive presentations beyond traditional classification systems.

6.2. Environmental pressures and risks

Environmentally based psychosocial stressors often constitute life situations that youth experience as non-controllable due to lack of autonomy (e.g. parenting behaviors; Cook et al., 2018) and likely contribute to a significant degree to the prevalence of MDEs (Lu, 2019). Forms of early life stress (ELS; e.g., abuse, neglect, community violence, poverty, foster home placement) may exacerbate youth-onset MDD risk (LeMoult et al., 2020) more so than youth-modifiable lifestyle factors. Furthermore, ELS may influence self-reported depression symptoms and trajectories from adolescence into adulthood (Desch et al., 2023). Youths who experienced early life stress were found to be 2.5 times more likely to develop MDD before age 18 years, with exposure to domestic violence, a death of a family member, or sexual, physical, and emotional abuse being significantly more associated with depression onset by childhood or adolescence (LeMoult et al., 2020). As such, these events may serve as forms of Adverse Childhood Experiences (ACEs) and subsequently exacerbate mood disturbances in a dose-dependent fashion beyond adolescence into adulthood (Desch et al., 2023; Giano et al., 2021). Functional and socioemotional impairments may even appear in subclinical adolescent depression, based on symptom severity, in turn reducing overall quality of life (Bertha and Balázs, 2013). Moreover, global events can impact depression prevalence among youths: peer separation and social isolation predicted worsening depressive symptoms in younger adolescents (11–14 years old) and older adolescents experienced a three-fold increase in developing depressive symptoms during the COVID-19 pandemic (Alzueta et al., 2023).

7. Conclusion and future directions

7.1. Main findings and implications

Even with this impressive body of work, relating the vast, multi-

factorial developmental data to a RDoC Framework is not yet fully realized in current research and is under-utilized in the clinical setting. The current review aims to promote this integrative framework by highlighting how distinct factors contribute to depression development risk during the period of adolescence, including interactions with sleep, puberty, and biological processes. Doing so adds unique developmental contributions to conceptualizing adolescent depression as an age-specific condition. Neural plasticity and pubertal hormones play a significant role in shaping trajectories for brain development and are necessary to consider when assessing for adolescent depression risk. Furthermore, support for researching genetic influences on adolescent depression development has been demonstrated through the exploration of familial genetic risk and epigenetic studies. Gene-by-environment interactions likely facilitate depression risk in adolescents through epigenetic mechanisms, such as DNAm; yet these mechanisms may also be utilized to alleviate symptoms through targeted interventions that also highlight cognitive and emotional RDoC constructs pertinent to adolescent depression. Evidently, a multimodal approach to assessing for adolescent depression is warranted to advance precision mental health approaches. As such, we propose that adolescent depression is the outcome of dynamic interactions among systems, specifically neurodevelopment, sleep, and puberty (see Fig. 1).

7.2. Future directions

Future directions for expanding and incorporating multimodal assessment techniques in adolescent depression research may be achieved through developmentally oriented methodological design. For example, few longitudinal studies exist that examine the relationships between individual-level changes in functional connectivity, neural network structure, and depressive symptoms (Chahal et al., 2020), warranting researchers and clinicians to transition to utilizing lifespan-informed views on depressive disorders in research. Neuroimaging-focused methodologies may specifically yield valuable clinical insights, as fronto-amygdalar functional connectivity during emotional facial processing may predict treatment responsiveness for depression in youths and young adults diagnosed with MDD (Jamieson et al., 2021). As such, investigating functional connectivity modulation

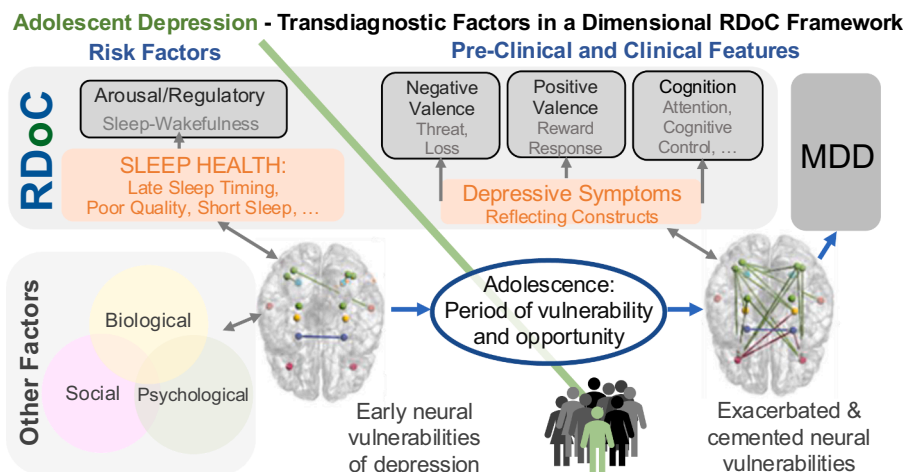


Fig. 1. Proposed model of how neurodevelopment, puberty, and sleep interact in the development and maintenance of adolescent depression. Adolescence is conceptualized as a unique neurodevelopmental period, with heightened vulnerability to depression and opportunity for positive change. From left to right: Unique features in adolescents across the depressive symptom continuum – Sleep health (duration, quality, timing) is emphasized as developmentally sensitive, modifiable risk marker, integrated within the RDoC Matrix in the Arousal/Regulatory System (notably with the Sleep-Wakefulness construct). Individual-level risk factors across social, biological, and psychological domains interact with poor sleep health (e.g., late sleep timing, short duration, poor quality) to shaping early neural vulnerabilities linked to depressive symptoms. Preclinical and clinical feature of depression can be understood using RDoC Positive and Negative Valence and Cognitive Systems with constructs encompassing, e.g., reward processing and fear, anxiety, loneliness, and loss experiences, internalization, attention, rumination, and executive functioning. Recognizing these risk factors and early symptoms could enhance our understanding of the onset and progression of major depressive disorder (MDD) in youth.

in the relevant networks, such as DMN, CCN, SN (Jamieson et al., 2021) and how they relate to respective RDoC features in depressed adolescents during intervention protocols is a promising area of ongoing research. Likewise, focusing on pubertal timing and age may be worthwhile in the context of adolescent depression, as research shows that associations between brain alterations and subsequent depression differ according to these factors (Toenders et al., 2019). As such, utilizing methodology to examine biological and environmental factors prior to, during, and following puberty may further illuminate how adolescent depression risk and resilience differs over time with continued development. There is need for the role of sleep processes to be further elucidated within adolescent depression development, as sleep has been demonstrated to be integral for averting depression, yet the relationship between sleep and other biopsychosocial factors is not yet fully understood. Both subjective sleep assessments and objective actigraphy or multisensory wearable technology can be easily utilized for research and clinical applications as they can be assessed in the home environment, across many nights and even years. They may therefore offer the potential for tracking sleep changes across adolescence in conjunction with mood changes as well as pubertal development. Accordingly, this review provides a novel model of mapping adolescent depressive risk, symptoms, and developmental processes onto RDoC domains to emphasize adolescence as a vulnerable yet dynamic period of change. Future models informed by research incorporating multimodal perspectives of adolescent depression development may further broaden our understanding via incorporating psychosocial and environmental stressors into sleep-depression relationships.

Author statement

The work described in the submitted manuscript has not been

Appendix

Table 1A
RDoC Matrix domains and constructs.

Domains	Negative Valence Systems	Positive Valence Systems	Cognitive Systems	Social Processes	Arousal and Regulatory Systems	Sensorimotor Systems
Constructs	Acute Threat ("Fear")	Reward Responsiveness	Attention	Affiliation and Attachment	Arousal	Motor Actions
	Potential Threat ("Anxiety")	Reward Learning	Perception	Social Communication	Circadian Rhythms	Agency and Ownership
	Sustained Threat	Reward Valuation	Declarative Memory	Perception and Understanding of self	Sleep-Wakefulness	Habit – Sensorimotor
	Loss		Language	Perception and Understanding of Others		Innate Motor Patterns
	Frustrative Nonreward		Cognitive Control Working Memory			

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CRediT authorship contribution statement

Lane M. Belmont-Olson: Writing – review & editing, Writing – original draft, Visualization. **Madison Wright:** Writing – original draft. **Eva M. Müller-Oehring:** Writing – review & editing, Supervision, Visualization. **Fiona C. Baker:** Writing – review & editing, Visualization. **Orsolya Kiss:** Writing – review & editing. **Kilian M. Pohl:** Writing – review & editing, Conceptualization. **Ian H. Gotlib:** Writing – review & editing. **Tilman Schulte:** Writing – review & editing, Supervision, Conceptualization.

Declaration of competing interest

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