

REVIEW ARTICLE

Advances in understanding adolescent emotion dysregulation: Mechanisms, risk factors, and interventions

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Abstract

Adolescence is a developmental period associated with increased vulnerability to emotion dysregulation (ED), reflecting interacting neurodevelopmental and psychosocial influences. This review synthesizes current theoretical models, risk factors, and interventions for adolescent ED, with a focus on integrative and transdiagnostic perspectives. The risk factors for ED in adolescents are multifaceted, involving biological factors, psychological factors, and social–environmental factors, all of which form a complex network of causes. Regarding interventions, neuromodulation techniques such as transcranial magnetic stimulation and transcranial direct current stimulation are emerging as effective approaches for targeting biological mechanisms of ED. These technologies can modulate brain activity to improve dysregulated emotional responses. Psychological interventions focus on the social and psychological factors influencing ED, effectively helping adolescents develop adaptive emotion regulation strategies. Additionally, family-based interventions are also critical in addressing the family dynamics that influence emotional regulation.

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Citation: Zhao C, Yang Z, Li L, Li K, Qian L. Advances in understanding adolescent emotion dysregulation: Mechanisms, risk factors, and interventions. *J Clin Basic Psychosom*.
doi: 10.36922/JCBP026150019

Received: April 11, 2026

Revised: July 8, 2026

Accepted: July 20, 2026

Published online: July 30, 2026

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Keywords: Emotion dysregulation; Adolescent psychiatry; Emotional regulation model; Neuromodulation techniques; Interventions

1. Introduction

Adolescence represents a critical developmental phase characterized by notable changes in physical, cognitive, social, and emotional domains. This period is associated with an increased susceptibility to emotional distress and the onset of various psychiatric conditions.¹ A key concept in elucidating this susceptibility is emotion dysregulation (ED), which refers to an individual's difficulty in flexibly adjusting emotional responses according to situational demands, leading to maladaptive reactions and an inability to sustain goal-directed behavior.^{2,3} Adolescents grappling with ED commonly display

mood fluctuations, a tendency toward intense and rapid emotional responses, and challenges in returning to a stable emotional state following provocation.⁴ In academic literature, ED is often used synonymously with “emotional regulation difficulties.”^{5,6} According to the multidimensional framework proposed by Gratz and Roemer⁶, ED encompasses six interrelated dimensions: lack of awareness of emotional responses, unclear emotional responses, inability to accept emotional responses, limited access to effective emotional regulation strategies, difficulty controlling impulses when experiencing negative emotions, and difficulty engaging in goal-directed behavior. Thus, ED represents a comprehensive, process-based, core construct that offers greater explanatory power than terms describing individual affective traits such as “irritability” or “emotional instability.”⁷ It provides a more systematic framework for understanding why adolescents are prone to emotional and behavioral difficulties.

The implications of ED during adolescence are significant and widespread. Emotion dysregulation is acknowledged as a transdiagnostic risk factor underlying various forms of psychopathology, including mood and anxiety disorders, personality disorders, eating disorders, and substance use disorders.⁸ Additionally, ED is closely associated with maladaptive coping strategies, such as non-suicidal self-injury (NSSI) and suicidality.^{9,10} The relationship between interpersonal challenges and these behaviors is intricate and often influenced by deficiencies in emotion regulation.¹¹ Given the profound influence of ED on adolescent well-being and future life course, a thorough comprehension of its mechanisms, risk factors, and effective interventions is imperative for public health.

Recent advances in neuroscience and clinical psychology have significantly improved our comprehension of adolescent ED. Neurodevelopmental models underscore the developmental gap between the swiftly evolving limbic system (the brain’s emotional hub) and the slower maturing prefrontal cortex (in charge of executive control and regulation), leading to a phase of heightened emotionality with reduced top-down control.^{12,13} Simultaneously, psychological models have elucidated the cognitive and behavioral mechanisms contributing to maladaptive emotional reactions. This expanding knowledge base has catalyzed the emergence of innovative interventions. While traditional psychotherapies remain fundamental in treatment, novel strategies such as neuromodulation techniques are gaining prominence.¹⁴ These techniques target the neural circuits involved in ED, presenting a promising option for patients unresponsive to standard therapies.

This review provides a systematic overview of the current understanding of adolescent ED, synthesizing

evidence across its neurobiological and theoretical mechanisms, multidimensional risk factors, and current and emerging interventions. This synthesis highlights the value of an integrated, multi-level approach to address this crucial challenge in adolescent mental health.

2. Aims and methods

This review aims to synthesize current evidence on the mechanisms, risk factors, and therapeutic interventions for adolescent ED through integrative and transdiagnostic frameworks. A targeted literature search was conducted in PubMed, PsycINFO, and Web of Science using combinations of keywords including “emotion dysregulation,” “adolescent,” “risk factors,” “intervention,” “neuromodulation,” and “psychotherapy.” This was supplemented by manual screening of reference lists from key studies. Selection criteria prioritized recent meta-analyses, systematic reviews, and high-quality empirical studies to provide a robust overview of the field.

3. Mechanisms of adolescent emotion dysregulation

To grasp the mechanisms underlying ED in adolescents, a holistic viewpoint is essential, merging neurobiological development with psychological models of emotion processing. Adolescence represents a distinct period marked by substantial brain reorganization, shaping a neurobiological terrain that is especially responsive to emotional fluctuations.

3.1. Neurodevelopmental imbalance

A prominent neurobiological model of adolescent ED suggests a transient imbalance in the development of crucial brain networks. This theory proposes that during adolescence, there is accelerated growth and heightened reactivity in subcortical limbic structures, particularly the amygdala, which processes emotions such as fear and threat.¹² In contrast, the prefrontal cortex (PFC), responsible for functions including impulse control, planning, and regulating emotional responses, matures more slowly, with development continuing until the early twenties. This developmental delay leads to a dominance of “bottom-up” processing, where intense emotional signals from the limbic system can overpower the still maturing “top-down” regulatory abilities of the PFC.^{12,13} Studies using neuroimaging techniques in adolescents diagnosed with borderline personality disorder (BPD), a condition characterized by severe ED, have identified structural changes, such as decreased amygdala volume as early as age 13, which can forecast future symptoms. These studies also reveal functional patterns of heightened amygdala activity alongside reduced PFC activity in individuals with BPD.¹⁵

However, the limbic–prefrontal imbalance model remains debated in explaining the mechanisms of adolescent ED. Longitudinal neuroimaging studies have yet to reach consensus on the temporal precedence of limbic hyperreactivity versus PFC maturation; some evidence suggests that PFC development may precede and predict limbic trajectories, rather than the reverse.¹⁶ Furthermore, cross-sectional age comparisons are limited in capturing within-individual developmental dynamics and cannot provide rigorous causal tests of this hypothesis.¹⁷

3.2. Theoretical models of emotion dysregulation

Several psychological models offer frameworks for comprehending the cognitive and behavioral aspects of ED.

3.2.1. The process model of emotion regulation

Gross's process model of emotion regulation views emotion regulation as a multistage sequential process comprising five key phases: situational selection, situational modification, attentional deployment, cognitive transformation, and response modulation.¹⁸ The model's core lies in elucidating that emotion regulation can intervene at different nodes within the emotion generation process. Subsequently developed extended process models further define emotion regulation as a cyclical decision-making process encompassing four phases: recognition, selection, implementation, and monitoring.^{19,20} This cycle continues until the discrepancy between the emotional state and the desired state is sufficiently reduced.^{21,22} This model explains how maladaptive cycles of ED are maintained. For instance, in depression, negative recognition biases lead to poor strategy choices (e.g., rumination), whose ineffective implementation reinforces initial negative appraisals, perpetuating dysregulation. Cross-diagnostic research confirms that psychopathology is closely linked to inflexible strategies such as excessive avoidance or suppression.²³ Despite its influence, the process model has drawn attention to its cross-cultural applicability. The adaptiveness of reappraisal versus suppression varies across cultural contexts; suppression, for instance, may be less maladaptive in collectivistic cultures, in which emotional restraint may be more strongly valued, than in Western individualistic cultures.²⁴

3.2.2. Metacognitive model

The metacognitive model of ED underscores the significance of individuals' metacognitive beliefs regarding their cognitive and emotional states in the emotional-regulation process.²⁵ According to this model, ED arises and is maintained through maladaptive metacognitive

beliefs, such as "I cannot control my feelings," which trigger a cognitive attentional syndrome (CAS).²⁶ CAS is characterized by persistent negative thinking, excessive self-focused attention, and rigid coping behaviors, which together consume cognitive resources and hinder flexible emotion regulation.²⁵ This process often becomes cyclical, as ongoing emotional struggles reinforce dysfunctional metacognitive beliefs, further entrenching ineffective regulation attempts.²⁷ Clinically, metacognitive therapy addresses this cycle by targeting underlying beliefs and reducing CAS, showing demonstrated efficacy in improving ED.²⁸

3.2.3. Cognitive maintenance model of emotion dysregulation

This multidimensional model explains the formation and persistence of ED by highlighting the central role of early attachment experiences in shaping core cognitive schemas (e.g., "I am unlovable"), which underlie emotional vulnerability. When interpersonal events are appraised as threatening (e.g., rejection), these schemas are activated, triggering intense negative emotions.²⁹ Emotion dysregulation is maintained through a combination of (i) secondary appraisals labeling emotions as unmanageable, (ii) maladaptive short-term coping strategies (e.g., excessive reassurance-seeking or emotional avoidance), and (iii) processes such as rumination that amplify emotional distress. These cognitive–behavioral patterns form a self-sustaining cycle.³⁰ This model clearly delineates pathways from early vulnerability to current maintenance factors, providing explicit operational targets for cognitive behavioral therapy (CBT).

3.2.4. The integrative ball-in-bowl metaphor model

This model offers an intuitive, dynamic framework by using the analogy of a ball moving within a bowl to represent emotional states.³¹ The bowl's shape represents an individual's psychophysiological regulatory capacity (e.g., shallow curvature may indicate slower recovery from emotional arousal). Its tilt corresponds to strategic regulation efforts: adaptive tilting (e.g., cognitive reappraisal) promotes equilibrium, while maladaptive tilting (e.g., suppression) exacerbates turbulence. The walls feature a gradient reflecting the individual's appraisal of emotional controllability, a process influenced by metacognitive beliefs. For the first time, this model integrates emotional appraisal, physiological regulation, and behavioral strategies into a unified dynamic system, illustrating how they jointly determine emotional experience. It thus provides a theoretical foundation for developing multi-level, integrated intervention strategies.

4. Risk factors for adolescent emotion dysregulation

The emergence of ED in adolescents is a multifaceted process influenced by biological predispositions, psychological traits, and social–environmental factors. These elements interact within a complex causal framework, where weaknesses in one aspect can heighten risks in another.

4.1. Biological factors

Genetic and neurobiological vulnerabilities contribute to traits, such as negative affectivity and poor impulse control, which are fundamental to ED. Evidence suggests a genetic basis for these traits.³² Additionally, the structural and functional characteristics of the adolescent brain create inherent vulnerability, which can be exacerbated in certain individuals. For instance, neuroimaging studies in adolescents with BPD have identified structural and functional abnormalities in the default mode network and limbic–cortical circuits that are associated with ED; such circuit abnormalities represent neural correlates of emotion dysregulation and may contribute to its pathological process.³³ Studies on BPD, characterized by severe ED, indicate that early reductions in amygdala volume and disruptions in oxytocin signaling are correlated with later symptoms and interpersonal dysfunction.¹⁵ Moreover, a combination of high heart rate and low heart rate variability, reflecting reduced parasympathetic (vagal) tone, has been linked to depression and may represent a physiological basis for diminished emotion regulation capacity.³⁴

4.2. Psychological factors

Certain temperamental traits, such as high negative emotionality and low effortful control, observed in early childhood, serve as strong indicators of future challenges in emotion regulation.^{35,36} Cognitive factors are pivotal in the manifestation of ED among adolescents. Cognitive distortions, deficits in higher-order cognitive processes, and maladaptive cognitive patterns are prevalent in this population. Adolescents with ED often display a negative interpretation bias towards ambiguous situations, a propensity for catastrophizing, and challenges in problem-solving when experiencing emotional distress.^{37,38} Deficiencies in mentalization and metacognitive abilities hinder the development of a coherent understanding of emotional states, leading to confusion and maladaptive coping strategies.^{27,39} Additionally, impaired cognitive flexibility, which refers to the capacity to adjust thinking and behavior according to changing situational demands, is closely linked to ED.⁴⁰

4.3. Social–environmental factors

The social environment is where individuals acquire and hone their emotion regulation abilities. Negative encounters within this sphere significantly increase the likelihood of developing ED.

The family environment plays a foundational role in emotional socialization. Meta-analytic evidence confirms a strong association between parenting styles and ED in youth.⁴¹ Negative parenting practices, such as psychological control and low care, are linked to both internalizing and externalizing problems, with ED acting as a key mediator in this relationship.⁴² Supportive emotional socialization, characterized by parental acceptance, empathy, and constructive guidance in response to children's negative emotions, facilitates the development of adaptive regulation skills. Conversely, unsupportive responses (e.g., dismissing or punishing emotional expression) hinder this acquisition, increasing vulnerability to ED.⁴³ Furthermore, adverse attachment experiences can impair the development of mentalization, the capacity to understand one's own and others' mental states. Deficits in mentalization make it difficult for adolescents to accurately identify and interpret emotions, thereby increasing ED in interpersonal contexts.^{44,45}

As peer relationships gain prominence during adolescence, they become critical for emotional well-being. Peer victimization, rejection, and conflict are significant sources of emotional distress and can exacerbate an individual's vulnerability to ED.⁴⁶ Neuroimaging studies indicate that early interpersonal stressors, such as childhood maltreatment and peer victimization, are associated with common neural alterations in circuits responsible for processing negative emotions.^{47,48} Longitudinal evidence suggests a bidirectional relationship: experiencing peer victimization predicts later ED, which may, in turn, increase susceptibility to further victimization due to impaired social skills.⁴⁹ From a cognitive perspective, victims of peer aggression often develop maladaptive implicit processing patterns, such as a reinforced "victim self-schema" and a vigilance-avoidance pattern toward social threats, which perpetuate emotional dysregulation.^{50,51}

Exposure to childhood trauma, encompassing physical abuse, emotional abuse, or neglect, is a significant risk factor for ED.⁵² Childhood maltreatment broadly impacts children and adolescents' emotional regulation functions, prompting them to adopt more maladaptive stress coping strategies.⁵³ Meta-analytic neuroimaging findings show that individuals with a history of childhood maltreatment exhibit heightened amygdala reactivity to threatening stimuli, reflecting a sustained hypervigilant response.⁵⁴

Puetz *et al.*⁵⁵ investigated how different forms of childhood maltreatment influence threat-related neural responses. They found that the maltreated group exhibited heightened activation in the left ventral amygdala to angry faces, while the neglected group showed increased activation in the bilateral dorsal amygdala and distributed frontoparietal networks. The above findings indicate that such trauma severely disrupts the development of neural circuits involved in stress responses and emotional regulation, thereby impairing emotional reactivity regulation and behavioral control abilities. Furthermore, the association between childhood abuse and subsequent maladaptive behaviors including NSSI is often influenced by both ED and trauma symptoms.⁵⁶

5. Interventions for adolescent emotion dysregulation

Effective interventions for ED necessitate a comprehensive strategy that targets biological vulnerabilities and psychosocial factors. Common intervention methods include pharmacological, neuromodulatory, psychosocial, and digital interventions.

5.1. Pharmacological interventions

Pharmacological approaches are commonly used for adolescents with ED accompanied by other mental disorders, and their application must strictly follow the diagnosis of the underlying disease. A longitudinal study found that pharmacological treatment (e.g., methylphenidate and atomoxetine) had a positive effect on improving ED symptoms in adolescents with attention-deficit/hyperactivity disorder (ADHD).⁵⁷ A recent meta-analysis confirmed that central stimulants, including methylphenidate and lisdexamfetamine, significantly improve ED symptoms in individuals with ADHD, with an overall moderate pooled effect size (Hedges' $g = 0.51$).⁵⁸

For individuals with autism spectrum disorder, a recent meta-analysis of 45 randomized controlled trials (RCTs) confirmed that antipsychotics significantly improve emotional dysregulation and irritability in autism spectrum disorder compared to placebo (standardized mean difference [SMD] = 1.028), with aripiprazole (SMD = 1.179) and risperidone (SMD = 1.074) showing the largest effects.⁵⁹ However, their use in adolescents is substantially limited by significant adverse effects, including weight gain, metabolic changes, and extrapyramidal symptoms, particularly in the absence of severe behavioral disturbance.⁶⁰

5.2. Neuromodulation techniques

In recent years, neuromodulation techniques have

emerged as promising tools for directly modulating neural activity in psychiatric disorders. Two commonly used non-invasive brain stimulation techniques, repetitive transcranial magnetic stimulation (rTMS) and transcranial direct current stimulation (tDCS), can modulate cortical excitability and have been investigated for their potential to improve emotional regulation.⁶¹ Current evidence regarding the efficacy of neuromodulation in adolescents with ED remains limited. One open-label trial of adjunctive deep TMS in adolescents with externalizing behavior disorders reported preliminary improvements in ED following 15 sessions.⁶² A recent double-blind, sham-controlled RCT in 60 adolescents with major depressive disorder and NSSI found that high-intensity transcranial alternating current stimulation targeting the prefrontal–limbic circuit led to significant improvements in emotion regulation difficulties (Cohen's $d = 0.89$).⁶³

For BPD and ADHD, conditions characterized by severe ED, systematic reviews of interventions using rTMS and tDCS have reported promising effects on ED, impulsivity, and suicidal ideation.^{61,64} However, it is critical to note that the vast majority of these studies were conducted in adult populations; direct evidence for the efficacy and safety of neuromodulation techniques in adolescents with ED remains extremely limited.

5.3. Psychological interventions

Various psychological intervention methods have been proven effective in improving ED in adolescents. CBT helps adolescents identify and change negative thought patterns and learn adaptive coping strategies to improve emotional regulation skills. Research indicates that CBT yields small to moderate effects ($g = 0.20$ to 0.29) in improving adolescent ED,⁶⁵ as evidenced by improved scores on the Emotion Regulation Checklist, reflecting enhanced emotion regulation abilities and reduced impulsive emotional responses. In contrast, dialectical behavior therapy (DBT) and mindfulness interventions focus more on cultivating emotional acceptance, distress tolerance, and present-moment awareness, promoting emotional regulation by reducing impulsive or avoidant behaviors. For adolescents with BPD or self-harming behaviors, DBT has demonstrated significant effectiveness in improving ED and reducing impulsive behaviors.⁶⁶ Research studies indicate that DBT outperformed conventional treatment in improving irritability and behavioral outbursts among children with disruptive mood dysregulation disorder, with effects remaining stable at the 3-month follow-up.⁶⁷ Another study comparing DBT, acceptance and commitment therapy, and their combined intervention showed significant improvements in BPD symptoms, ED, and interpersonal functioning across all

three groups, with no significant differences between them.⁶⁸ Regarding mindfulness interventions, Sharma *et al.*'s⁶⁹ systematic review indicated that mindfulness-based interventions effectively improve adolescent ED and co-occurring issues such as stress, anxiety, and depression, with effects supported by RCTs. Although some studies suggest efficacy lasting 3–6 months, long-term effects require further validation.

Given the substantial impact of family dynamics on ED, the inclusion of family members in treatment is crucial. Emotion-focused parent management training improves parents' emotional awareness and regulation abilities, optimizes parents' responses to and communication with adolescents' emotions, and indirectly improves children's emotional functioning and reduces adolescent emotional and behavioral problems.⁷⁰ Overall, various psychosocial interventions effectively enhance emotional regulation abilities in adolescents with ED. However, existing research lacks direct comparisons between different psychological interventions and exhibits heterogeneity in sample characteristics and assessment tools.⁷¹ Consequently, it remains inconclusive whether any single approach holds an overall advantage.

5.4. Digital interventions

In recent years, advances in digital technology have opened new avenues for personalized interventions in the field of emotional well-being. Mobile applications use real-time feedback and skill training to help adolescents practice emotional regulation strategies in their daily lives.⁷² For example, the BRITE app, designed for adolescents with suicidal tendencies, provides real-time prompts for users to assess the severity of their emotional distress and delivers emotion regulation strategies based on CBT and DBT principles. It also includes a "safety plan" module with features such as one-click emergency contact calls and a positive memory bank to help adolescents cope with emotional crises.⁷³ Virtual reality technology simulates emotion-inducing scenarios to provide a safe practice environment, enhancing cognitive reappraisal and stress coping abilities.⁷⁴ A pilot study of Emo-regulator, a system integrating virtual reality with electroencephalography-based neurofeedback, demonstrated that eight participants who used the system for two weeks showed improved ability to down-regulate negative emotions and increased frequency of cognitive reappraisal use, supporting the feasibility of digital interventions that combine virtual reality with real-time neurofeedback for emotion regulation training.⁷⁵ However, the evidence base for digital interventions remains nascent, with most studies characterized by small sample sizes and short follow-up periods. Large-scale, well-controlled trials are needed

before such approaches can be recommended as standalone interventions for adolescent ED.

6. Conclusion and future directions

Adolescent ED, influenced by neurodevelopmental processes, psychological factors, and environmental influences, is a significant public health concern. It acts as a transdiagnostic predictor for various mental health issues, such as self-harm and suicidality. Existing evidence indicates that cognitive-behavioral and mindfulness-based psychological interventions can effectively improve emotional regulation functions, while neuromodulation technologies offer novel targeted treatment pathways for ED. Future research should focus on developing a multi-level, integrated personalized intervention framework. By identifying distinct subtypes defined by neurocognitive and social characteristics, intervention strategies can be precisely tailored. Advancing mechanism-driven, stratified treatments and accessible real-time interventions represents a critical direction for reducing the burden of ED and promoting adolescent mental health.

Adolescent ED is shaped by neurodevelopmental, psychological, and environmental factors, and acts as a transdiagnostic predictor of various mental health issues, including self-harm and suicidality. Existing evidence indicates that CBT, DBT, and mindfulness-based interventions can effectively improve emotion regulation, while neuromodulation techniques offer novel but still experimental treatment pathways. However, current theoretical models, though valuable, lack a unified framework capable of integrating biological, psychological, and social factors; similarly, most interventions remain focused on a single level of regulation. Comprehensive, multi-level strategies, such as those inspired by the integrative "ball-in-bowl" model, require further empirical validation.

To address these gaps, future research should focus on the following directions. First, longitudinal, multimodal designs should be employed to map the co-development of neural circuits and emotion regulation strategies, thereby elucidating distinct pathways to ED. Second, ED should be classified into neurocognitive subtypes (e.g., based on emotional sensitivity versus cognitive control deficits) to guide mechanism-informed personalization of interventions. Third, given the theoretical promise of neuromodulation for adolescent ED, rigorous large-scale, sham-controlled trials are urgently needed to establish its efficacy and long-term safety in adolescent populations. Fourth, smart, real-time intervention systems should be developed that integrate ecological momentary assessment with mobile technology, enabling dynamic delivery

of personalized regulation strategies during high-risk emotional situations in adolescents' daily lives.

Acknowledgments

None.

Funding

This work was supported by the Key Research Plan of Jining City (grant number 2024YXNS206).

Conflict of interest

Kun Li is a Youth Editorial Board Member of this journal, but was not in any way involved in the editorial and peer-review process conducted for this paper, directly or indirectly. The authors declare that they have no conflicts of interest.

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Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Availability of data

Not applicable.

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