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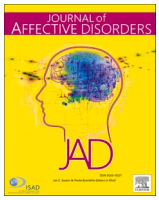



Neurobiological mechanisms of anhedonia in adolescents with mood disorders: A scoping review

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ABSTRACT

Background: Anhedonia—a core symptom of mood disorders—reflects impairments in both reward anticipation and reward consumption. While its neurobiological underpinnings have been extensively studied in adults, considerably less is known about adolescent populations, despite critical neurodevelopmental changes during this period. This scoping review aims to map current evidence onto neurobiological correlates of anhedonia in adolescents with mood disorders.

Methods: A systematic search was conducted in PubMed and PsycInfo to identify studies investigating anhedonia in adolescents with mood disorders using neuroimaging techniques. Due to heterogeneity in study design and outcomes, findings were synthesized narratively.

Results: Twenty-one articles met the inclusion criteria, reporting on 1278 patients. The analysis revealed that anhedonia involves (1) increased frontal and decreased striatal activity during reward/incentive processing, and activation of regions of the limbic system during loss-related error processing; (2) striatal and corticolimbic activity during emotional processing; (3) reduced medial prefrontal cortex activity during cognitive processing; (4) involvement of corticostriatal and corticolimbic functional connectivity networks; (5) an inverse association between cerebral blood flow in corticolimbic and frontal regions in cerebral blood flow analysis; (6) altered alpha and theta activity and reduced cortical reactivity, and that; (7) GABA involvement in anhedonia remains inconclusive.

Conclusions: The neurobiological signatures of anhedonia in adolescents appear to involve altered activation and connectivity in reward-related circuits, particularly during anticipatory and error-processing phases. These findings underscore the need for greater methodological consistency, standardized phenotyping of anhedonia, and longitudinal studies to better understand developmental trajectories and inform early interventions.

1. Introduction

Anhedonia, broadly defined as a diminished ability to anticipate or experience pleasure, is a hallmark symptom of mood disorders, including major depressive disorder (MDD) and bipolar disorder (BD) (Auerbach et al., 2019; Klein, 1973). While well-documented in adult populations, the expression and neurobiological underpinnings of anhedonia in adolescents* remain underexplored. This knowledge gap is particularly concerning given the rising prevalence of mood disorders in youth and the unique neurodevelopmental trajectories that characterize adolescence (Sacco et al., 2024).

Adolescence constitutes a critical period in neurodevelopment, characterized by extensive biological, emotional, and cognitive reorganization. This developmental window is marked by protracted maturation of prefrontal control regions, heightened limbic responsivity, and ongoing development of reward-related circuitry, resulting in a functional imbalance between affective drive and cognitive regulation. These neurodevelopmental divergences contribute to heightened sensitivity to reward and increased exploratory behavior, largely driven by dynamic maturation of the mesocorticolimbic circuitry (Whitton and Pizzagalli, 2022; Liang et al., 2023). Perturbations in reward processing during this sensitive phase may exert enduring effects, shaping trajectories of social functioning, academic achievement, vocational aspirations, and overall adaptive capacity. Empirical evidence links adolescent

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anhedonia to impaired peer relationships, disengagement from school, and elevated risk for substance use (Chen et al., 2024; Fletcher, 2010; Lewinsohn et al., 1998). Crucially, early-onset anhedonia is a robust predictor of persistent mood dysregulation and adverse long-term outcomes, highlighting the imperative for early detection and developmentally tailored interventions (Barch et al., 2019).

Neurodevelopment during adolescence is further shaped by pubertal neuroendocrine changes, including fluctuations in gonadal hormones that modulate reward sensitivity, threat processing, and emotional reactivity. These biological shifts coincide with profound changes in social roles and expectations, such as increasing[1] peer salience, autonomy from caregivers, and heightened sensitivity to social evaluation. Within this context, anhedonia in adolescents often manifests behaviorally as reduced social engagement, diminished exploratory and goal-directed behavior, motivational withdrawal, and blunted responsiveness to typically rewarding experiences. These behavioral expressions may differ from adult presentations and are strongly embedded in the adolescent's evolving social and developmental milieu, complicating clinical recognition and assessment (Gupta et al., 2024).

Despite its clinical significance, anhedonia in adolescents remains challenging to assess and treat effectively. Current diagnostic frameworks often conceptualize anhedonia narrowly, emphasizing loss of pleasure while neglecting its multidimensional nature—particularly impairments in reward anticipation, effort valuation, and learning (Su and Si, 2022; Shin et al., 2024). Clinically, anhedonia in adolescents is most commonly assessed using self-report measures such as the Snaith–Hamilton Pleasure Scale (SHAPS), which primarily captures consummatory pleasure, and the Temporal Experience of Pleasure Scale (TEPS), which differentiates anticipatory and consummatory components. Behavioral reward-learning paradigms are also used to index motivational and reward responsiveness. However, these tools vary in their sensitivity to distinct anhedonia dimensions and often fail to account for neurodevelopmental differences in reward circuit maturation between adolescents and adults (Liu et al., 2022; Thapar et al., 2012), increasing the risk of underrecognition or misinterpretation in youth." Neuroimaging modalities—including functional near-infrared spectroscopy (fNIRS), electroencephalography (EEG), and particularly functional magnetic resonance imaging (fMRI)—have significantly advanced our understanding of the neural substrates underlying anhedonia. In adult populations, consistent evidence implicates dysregulation within key reward-related structures, including the striatum, ventral tegmental area (VTA), and orbitofrontal cortex (OFC), with distinct neural profiles observed across major depressive disorder (MDD) and bipolar disorder (BD) (Whitton and Pizzagalli, 2022; Rizvi et al., 2018). Emerging studies utilizing fNIRS and EEG further underscore the neurobiological complexity of anhedonia. fNIRS findings reveal aberrant activation in prefrontal and orbitofrontal regions (Fan et al., 2024; Feng et al., 2021; Chase et al., 2025), while EEG research highlights alterations in event-related potentials (ERPs) associated with both reward anticipation and reward consumption (Sun et al., 2023). However, these findings cannot be readily generalized to adolescent populations, whose reward circuitry is still undergoing maturational changes and may be uniquely susceptible to mood-related disruptions. Furthermore, although adult studies have begun to differentiate between anticipatory and consummatory dimensions of reward processing, such distinctions remain largely underexplored in adolescent research (Pornpattananangkul et al., 2019; Kang et al., 2023).

Anhedonia also reflects dysfunction in the mesocorticolimbic pathway, comprising of the VTA, nucleus accumbens (NAc), and medial prefrontal cortex (mPFC), critical for reward processing and motivational behaviours (Nakamura et al., 2023). Glutamatergic and GABAergic imbalance in this circuit is correlated with anhedonic symptoms and reward-processing deficits (Wang et al., 2021). Neuroimaging studies in depressed adults likewise show reduced activation of this circuitry in the NAc and anterior cingulate cortex (ACC) (Heshmati and Russo, 2015; Pizzagalli et al., 2009). However, a recent meta-analysis comparing adults and adolescents with MDD found non-overlapping reward-related clusters in depressed adolescents, indicating developmental differences within reward circuits between adolescents and adults with MDD (Bore et al., 2024). These findings underscore the need for age-specific investigations of anhedonia.

To address this critical knowledge gap, the present scoping review systematically synthesizes findings from neuroimaging and neurotransmission studies examining anhedonia in adolescents diagnosed with mood disorders. By focusing on functional brain correlates elucidated through these complementary lines of inquiry, the review aims to offer a comprehensive overview of the current evidence base and to identify key limitations and directions for future research. Advancing our understanding of the neurobiological underpinnings of anhedonia during adolescence holds promise for informing more developmentally sensitive diagnostic frameworks and guiding targeted, mechanism-based interventions for mood disorders.

2. Methods

2.1. Search strategy

A comprehensive systematic search was conducted across PubMed and PsycInfo without date restrictions, aiming to capture all relevant empirical literature on anhedonia in adolescents with mood disorders. Search terms included combinations of keywords related to major depressive disorder, bipolar disorder, anhedonia, and neuroimaging modalities (e.g., fMRI, fNIRS, PET, EEG). The full search syntax for each database is detailed in *Supplementary Table 1*. The search was conducted on July 24, 2025. This review adhered to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses extension for Scoping Reviews (PRISMA-ScR) framework (Tricco et al., 2018).

2.2. Eligibility criteria

Two independent reviewers (AK & FM) screened all titles, abstracts, and subsequently full texts based on predefined eligibility criteria using Rayyan (Ouzzani et al., 2016). To minimize bias, reviewers were blinded during the screening process. Blinding was lifted after reviewers completed screening. Discrepancies were resolved through discussion. Studies were included if they met the following criteria: 1) article concerns an original study published in a peer-reviewed journal and written in English; 2) participants must be aged between 10 and 24; reflecting an extended conceptualization of adolescence that accounts for ongoing neurobiological and psychosocial development into early adulthood. 3) at least 50% of participants were diagnosed with mood disorders based on the Diagnostic and Statistical Manual of Mental Disorders (DSM) or the International Classification of Diseases (ICD); 4) studies used neuroimaging modalities; 5) articles must contain empirical data; 6) anhedonia must be measured using questionnaires; and 7) full-text must be available. Studies were excluded if they met the following criteria: 1) article concerns a letter, commentary, view, review or meta-analysis; 2) all or a portion of the participants suffered from comorbid schizophrenia; and 3) article included animal subjects.

2.3. Data extraction

A standardized data extraction form was developed a priori. The following information was collected from each eligible study: (1) study title, first author, publication year, and journal; (2) mood disorder

[1] *Although the World Health Organization defines adolescence as ages 10–19, contemporary developmental neuroscience increasingly conceptualizes ages 10–24 as a period of extended adolescence due to continued brain maturation, sociodevelopmental transitions, and identity formation. This framework was adopted in the present review.

diagnosis and assessment method; (3) method of anhedonia assessment; (4) sample characteristics, including size, sex distribution, age range, and medication status; (5) neuroimaging modality, task type, and paradigm; and (6) key neurobiological findings, including specific brain regions and circuits implicated. When available, additional contextual data were extracted, including comorbidities, methodological strengths or limitations, and comparison group information.

2.4. Data synthesis

Given the heterogeneity in neuroimaging modalities, experimental paradigms, and outcome measures, results were synthesized using a narrative approach. Neurobiological findings were systematically categorized based on neuroimaging modalities and further subcategorized according to functional neural circuits (e.g. corticolimbic, corticostriatal) and specific phases of reward processing (anticipation, error processing, consummation). The synthesis was structured to address the following guiding questions: 1. Which neurobiological patterns have been reported in association with anhedonia in adolescents with mood disorders?; and 2. how have neuroimaging modalities been used to explore the potential relevance of these patterns for clinical characterization, without aiming to establish diagnostic or differential-diagnostic validity.

Consistent with scoping review methodology, the goal was to map and synthesize emerging evidence rather than to draw definitive conclusions.

3. Results

3.1. Study selection and characteristics

The database search (completed on July 24, 2025) identified 345 records. After screening, a total of 21 articles were considered eligible for inclusion. The study selection process is summarized in Fig. 1. Included studies were grouped based on the neurofunctional processes investigated, including: (1) reward/incentive processing (n = 4), (2) emotional processing (n = 3), (3) cognitive processing (n = 1), (4) functional connectivity (n = 4), (5) cerebral blood flow (n = 1), (6) electrical activity (n = 5), and (7) neurotransmission (n = 3). Key study characteristics and outcomes are summarized in Table 1.

3.2. Reward (incentive) processing

Four studies examined neural responses during various phases of reward processing—anticipation, consumption, and prediction error—using incentive tasks such as the Monetary Incentive Delay (MID) task and the Reward Flanker Task (RFT) (Rizvi et al., 2016; Bradley et al., 2017).

Bradley et al. (2017), using the RFT in MDD adolescents (n = 22) and HC (n = 6), found a positive correlation between right angular gyrus activity during reward anticipation and anhedonia severity. Additionally, left precuneus activity was positively associated with positive prediction error processing and anhedonia in the MDD adolescents.

Auerbach et al. (2022) conducted an incentive processing task to assess neural responses to monetary rewards. They reported reduced NAc activity following reward outcome in MDD adolescents (n = 129) compared with healthy controls (HC) (n = 64), although this was not significantly associated with anhedonia severity.

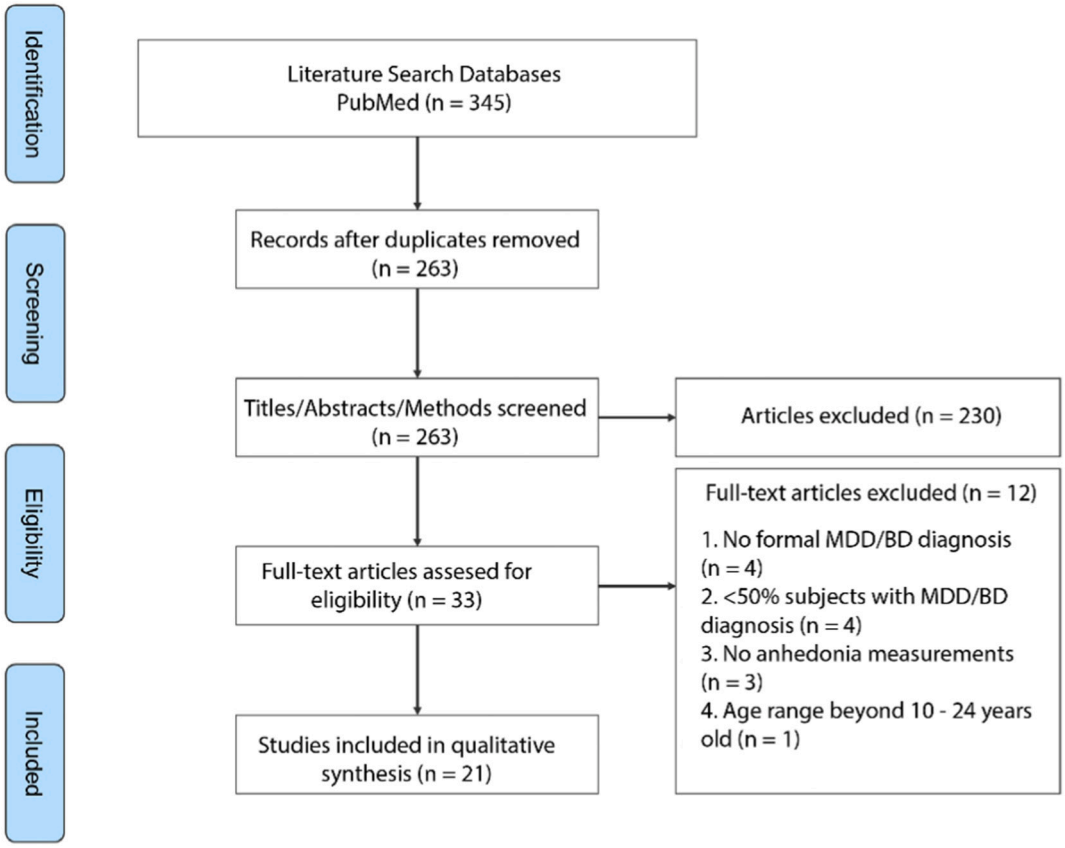

Fig. 1. PRISMA flowchart for study selection.
Note. Adapted from Page et al. (2021). For more information, visit: http://www.prisma-statement.org/.

Table 1
Summary of key study characteristics and outcomes of included articles (n = 21).

Lead author (year)	Subjects (characteristics)		Measurement of mood disorder and anhedonia			Imaging technique	Brain regions	Study findings
			Mood disorder		Anhedonia			
	Target group	Control group	MDD	BD				
Bradley et al. (2017)	n = 22 (45.5% female, 12–20 years old)	n = 6 (Sex not specified, 12–20 years old)	K-SADS-PL; CDRS-R		SHAPS	fMRI	Right angular gyrus Left precuneus	Anticipatory stage: Right angular gyrus activation was associated with greater anhedonia severity. Reward attainment: Similar activity patterns as in reward anticipation. However, no correlations with anhedonia severity. Prediction error: Left precuneus activation was associated with greater anhedonia severity.
Auerbach et al. (2022)	n = 129 (69% female, 14–17 years old)	n = 64 (55% female, 14–17 years old)	MFQ RCADS		SHAPS	fMRI (Incentive processing task)		Blunted activation in the right nucleus accumbens following reward receipt. – However, not associated with baseline or follow-up symptoms or trait measures
Willinger et al. (2022)	n = 30 (67% female, 11–19 years old)	n = 33 (70% female, 11–19 years old)	K-SADS-PL or MINI-KID		CDI	fMRI (Monetary incentive delay)	Posterior insula Medial thalamus habenula PCC postcentral gyrus fusiform gyrus	Anhedonia scores negatively correlated magnitude-modulated loss PE signalling in medial thalamus/habenula, PCC, postcentral gyrus and fusiform gyrus of the MDD group. Anhedonia scores negatively correlated with loss-related outcome error signal in posterior insula of the MDD group.
Liu et al. (2020)	n = 64 (44.7% female, 12–20 years old)	N/A	CDRS-R, BDI, KSADS-PL		SHAPS TEPS	fMRI	bilateral ACC striatum nucleus accumbens putamen	No associations between anhedonia severity and blood CRP levels in the whole sample or in the mood and anxiety subgroup alone when we excluded adolescents with only externalized behavior symptoms Reward anticipation not correlated with CRP levels in whole brain Reward attainment was significant correlated (spec. in parts of VN and DAN) Neural activation in bilateral dACC was negatively correlated with CRP levels CRP levels positively correlated with bilateral accumbens nuclei (ROI analysis) and left lobule VI of cerebellum (whole brain analysis) during positive prediction error
Zhang et al. (2020)	n = 35 (68.8% female, 14–18 years old)	n = 37 (67.6% female, 14–18 years old)	BDI-II		TEPS SHAPS	fMRI	Frontal sites	Anticipatory stage: LPC largest for positive images at frontal sites, and largest for negative images at central sites in MDD group. However, no correlation with anhedonia severity. Gain stage: Smaller gain FN in MDD group compared to HCs, independent of emotional cues. Lower FN amplitudes correlated with higher scores on the consummatory subscale of the TEPS. Loss stage: Greater FN amplitudes for positive and neutral images in the MDD group compared to HCs. Greater FN loss amplitudes correlated with higher anhedonia severity on the SHAPS
Henderson et al. (2014)	n = 19 (68% female, 12–20 years old)	*n* = 18 (56% female, 12–20 years old)	K-SADS-PL CDRS-R BDI-II		CDRS-R BDI-II	fMRI (Face task)	Anterior insula MFG / dACC Cerebellum	Neural responses to happy faces were negatively correlated with anhedonia severity in the anterior insula, MFG/dACC and the cerebellum.
Thai et al. (2020)	n = 11 (27.3% female, 14–18 years old)	N/A	K-SADS-PL CDRS-R MADRS		TEPS	fMRI (Word-Face stroop task)	Right accumbens Subcallosal cingulate cortex	Decrease in activation of the right accumbens and subcallosal cingulate cortex were associated with increased pleasure after ketamine injection across all stimuli.
Liang et al. (2023)	n = 57 (MDD-mel, 67% female, 12–23 years old) n = 44 (MDD-nMel, 68% female, 12–23 years old)	n = 58 (85% female, 12–23 years old)	HAMD		SHAPS	fNIRS (VFT)	mPFC Frontal cortex Temporal cortex	MDD group (MDD-mel and MDD-nMel) showed lower brain activation in the frontal and temporal cortex compared to healthy controls MDD-mel showed lower activation in the mPFC compared to MDD-nMel MDD-mel showed higher cerebral hemodynamic changes compared to MDD-nMel Higher anhedonia scores were observed in the MDD-mel group.
Rzepa and McCabe (2018)	n = 44 (77% female, 13–21 years old)	n = 42 (76% female,	BDI MFQ		SHAPS TEPS FCPS	fMRI (RSFC)	dmPFC x ACC dmPFC x precuneus	Negative correlation right dmPFC x ACC with TEPS-A Positive correlation right dmPFC x left precuneus with TEPS-A

(*continued on next page*)

Table 1 (*continued*)

Lead author (year)	Subjects (characteristics)		Measurement of mood disorder and anhedonia			Imaging technique	Brain regions	Study findings
			Mood disorder		Anhedonia			
	Target group	Control group	MDD	BD				
		13–21 years old)						
Gabbay et al. (2013)	n = 21 (57% female, 12–19 years old)	n = 21 (57% female, 13–19 years old)	K-SADS-PL		CDRS-R	fMRI (RSFC)	Ventral and dorsal caudate x SMA, MFG,	Anhedonia scores were positively correlated with iFC strength between ventral dorsal caudate with SMA, MFG, supramarginal gyrus, precuneus and pgACC
			BDI-II		BDI-II		Supramarginal gyrus, Precuneus, pgACC	Anhedonia scores were negatively correlated with iFC strength between left NAc with sgACC and left caudate.
			CDRS-R				Right dorsal rostral putamen x Supramarginal gyrus Left NAc x sgACC, Left caudate Right NAc x Occipital fusiform cortex	Anhedonia scores were negatively correlated with iFC strength between right NAc with occipital fusiform.
Pan et al. (2022)	n = 303 (48.2% female, 14–18 years old)	N/A	DAWBA		DAWBA	fMRI	Ventral striatum	Left VS connectivityrw was associated with anhedonia at baseline Anhedonia at 2-year follow-up was associated with left VS connectivityrw, and anhedonia at 4-year follow-up was associated with right VS connectivityrw.
Liang et al. (2025)	n = 92 (anDE, 89.1% female, 10–17 years old) *n* = 58 (non-anDE, 77.6% female, 10–17 years old)	n = 51 (86.7% female, 10–17 years old)	N/A		CDI	fMRI (RSFC)	Left DCP x right SFG	Anhedonia scores were positively correlated with transformed hsCRP in anDE compared to non-anDE. Anhedonia severity was significantly correlated with left DCP and right SFG connectivity. Anhedonia severity was negatively predicted by left DCP and right SFG connectivity. Transformed hs-CRP did not yield this predictive effect.
Dimick et al. (2022)	n = 81 (BD, 63% female, 13–20 years old)	n = 75 (52% female, 13–20 years old)		K-SADS-PL	K-SADS-PL	fMRI (CBF)	ACC Amygdala MFG	ACC and MFG CBF negatively correlated with anhedonia severity in the BD group. Amygdala CBF increased in the BD group.
McVoy et al. (2019)	n = 25 (80.0% female, 14–17 years old)	n = 14 (73.3% female, 14–17 years old)	MDD		SHAPS	resting-state qEEG	Average alpha coherence Frontal alpha coherence Frontal beta coherence Frontal theta coherence	Greater anhedonia severity was found in depressed individuals with lower average alpha coherence and lower frontal alpha, beta and theta coherence. All these node pairs showed significantly lower coherence in the MDD group than the HCs.
Umemoto et al., 2021	n = 38 (100% female, 13–18 years old)	n = 43 (100% female, 13–18 years old)	BDI-II K-SADS		SHAPS	EEG	Posterior Alpha asymmetry	depressed adolescents exhibited significantly larger posterior alpha (i.e., lower brain activity) over the right versus left hemisphere, whereas healthy adolescents showed no hemispheric differences. posterior alpha power asymmetry is negatively correlated anhedonia severity. But the associations with alpha asymmetry did not remain significant when restricted to adolescents with MDD.
Zandbagleh et al. (2024)	*n* = 74 (52.7% female, 18–25 years old)	*n* = 279 (48.2% female, 18–25 years old)	BDI mass		BDI_Anh	EEG	Parietal Theta	periodic parietal theta correlates negatively with the scores on the anhedonia BDI subscale

(*continued on next page*)

Table 1 (*continued*)

Lead author (year)	Subjects (characteristics)		Measurement of mood disorder and anhedonia			Imaging technique	Brain regions	Study findings
			Mood disorder		Anhedonia			
	Target group	Control group	MDD	BD				
Wen et al. (2023)	n = 188 (71.3% female, 12–23 years old)	*n* = 94 (75.5% female, 12–23 years old)	DSM-V HAMD HAMA NSSI		SHAPS	EEG EEG microstate analysis fMRI	Beta, theta coherence and frontal Alpha	negative correlations between anhedonia scores (SHAPS) and alpha, theta, and beta coherence
Dhami et al. (2020)	n = 45 (60.0% female, 16–24 years old)	n = 20 (55.0% female, 16–24 years old)	MDD		DARS	TMS-EEG	dlPFC	Lesser P200 amplitude in both the left and right dlPFC was associated with greater anhedonia severity.
Gabbay et al. (2012)	n = 20 (12.60% female; aged 12–19)	n = 21 (15.71% female; aged 12–19)	CDSR-R BDI		CDSR-R Anh BDI Anh	Structural MRI 1-H MRS	ACC	Mean ACC GABA was decreased in anhedonic adolescents with MDD compared with HC group and with depressed non-anhedonic adolescents with MDD Negative association with GABA and anhedonia scores in anhedonic MDD subgroup No significant correlation between GABA and anhedonia scores in non-anhedonic group
Gabbay et al. (2017)	n = 44 (23.52% female; aged 12–21)	*n* = 36 (23.64% female; aged 12–21)	KSADS-PL, CDRS-R		BDI Anh CDRS-R Anh	Structural MRI 1-H MRS	ACC	In combined sample, GABA levels were different between anhedonic and non-anhedonic subgroups and HC; follow-up pairwise comparisons showed lower GABA levels in anhedonic youth with MDD compared with HC In the newly recruited group, there was no association b/w GABA and clinical variables, but in the combined sample, GABA was negatively correlated with anhedonia in the MDD group alone In the combined sample, it was seen that anhedonia significantly predicted GABA in MDD. It predicted 13.5% of the variance in GABA
Bradley et al., 2018	n = 20 (8.40% female; aged 12–21 years)	n = 16 (8.50% female; aged 12–21 years)	KSAD-PL		BDI Anh CDRS-R Anh	Striatal GABA MRS structural MRI	ACC Striatum	Within the MDD group, striatal GABA was not correlated with anhedonia, even when controlling for depression severity There were no significant correlations between Glx and anhedonia, either when controlling for or not controlling for depression severity

Willinger et al. (2022) observed reduced OFC activation during loss related reward outcome errors using the MID in MDD adolescents (n = 30) compared with HC (n = 33). Anhedonia scores were negatively correlated with loss-related activity in the posterior insula, habenula, and posterior cingulate cortex.

Liu et al. (2020) investigated inflammatory markers and neural reward processing. C-reactive protein levels (CRP) levels were negatively correlated with dorsal ACC (dACC) activity during reward processing, positively correlated with bilateral NAc activation during positive prediction error and positively correlated with regions of the visual and dorsal attention networks during reward attainment in adolescents with mood disorders (n = 42). However, no direct associations were found between CRP and anhedonia severity.

In summary, MDD adolescents exhibit increased frontal and decreased striatal activation during reward processing. Furthermore, prediction error signals were linked to posterior regions, with loss-related activity most consistently associated with anhedonia severity.

3.3. Emotional processing

Three studies examined emotional processing in the context of anhedonia. Zhang et al. (2020) compared event related potentials in adolescents with MDD (n = 35) with HC (n = 37) during the MID task combined with emotionally cued images. Late positive component (LPC) amplitudes varied by emotional valence and location but were not directly associated with anhedonia severity. Feedback negativity (FN) during gain processing was reduced in MDD compared with HC and correlated with consummatory anhedonia subscale scores.

Henderson et al. (2014) compared anhedonic adolescents with MDD (n = 19) with HC (n = 18) using a face task paradigm while subjects were presented with emotional faces. They found that neural responses to happy faces were negatively associated with anhedonia scores in the anterior insula, medial frontal gyrus (MFG), dACC, and cerebellum. Additionally, anhedonia severity was positively correlated with limbic activation in response to sad facial expressions; however, these associations did not remain significant after adjusting for overall depressive symptom severity.

Thai et al. (2020) investigated how ketamine infusions affected anhedonic symptoms using a paradigm that presented affective words superimposed on either congruent or incongruent emotional faces in MDD participants (n = 11). They observed that ketamine administration led to reduced activation in the NAc and subcallosal cingulate cortex, which was associated with increased pleasure scores on the anhedonia questionnaire.

Taken together, emotional processing of social stimuli in MDD adolescents involves altered corticolimbic and striatal activity, with some regions showing associations with anhedonia severity, particularly following intervention.

3.4. Cognitive processing

One study (Liang et al., 2023) analyzed differences in cognitive processing between a MDD group (n = 101) and HC (n = 58) using fNIRS and a verbal fluency task. They found decreased frontal and temporal activation in MDD adolescents (n = 101) compared with HC (n = 58). The melancholic MDD subgroup (MDD-Mel, n = 57) exhibited higher anhedonia scores and reduced mPFC activation compared with the non-melancholic MDD subgroup (MDD-nMel, n = 44). A negative correlation between mPFC activity and anhedonia severity was found only in the MDD-Mel subgroup.

3.5. Functional connectivity

In addition to task-based fMRI studies, functional connectivity studies provide valuable insights into network organisations and interactions of brain regions (Stevens, 2016).

Rzepa and McCabe (2018) investigated how resting-state functional connectivity (RSFC) relates to anhedonia severity by comparing participants with MDD (n = 44) with HC (n = 42). They identified that lower dmPFC–ACC functional connectivity was associated with greater anticipatory anhedonia., and increased dmPFC–precuneus connectivity was positively correlated with anticipatory anhedonia in MDD group compared with HC.

Gabbay et al. (2013) found anhedonia severity positively correlated with connectivity involving ventral caudate, dorsal caudate, supplemental motor area (SMA), MFG, ACC, right dorsal rostral putamen and supramarginal gyrus (n = 21). Whereas NAc–subgenual ACC and occipital cortex connectivity showed negative correlations with anhedonia severity.

Pan et al. (2022) tracked ventral striatum (VS) connectivity over four years (n = 303). Left VS connectivity was associated with baseline anhedonia and 2-year follow-up; right VS connectivity was associated with anhedonia at 4-year follow-up, suggesting developmental shifts.

Liang et al. (2025) compared the associations between high-sensitivity CRP (hs-CRP) and functional connectivity in depressed participants with anhedonia (anDE, n = 92), depressed participants without anhedonia (non-anDE, n = 58) and HC (n = 51). They found significant differences in functional connectivity between anDE and non-anDE between the right ventral caudate and left cerebellum VI, and between the left dorsal caudal putamen (DCP) and right superior frontal gyrus (SFG). Overall, they found positive correlations between transformed hs-CRP and anhedonia subscores in anDE compared with non-anDE. Additionally, they found that the connectivity between the left DCP and right superior frontal gyrus negatively predicted anhedonia severity, whereas transformed hs-CRP did not have a predictive effect for anhedonia severity. However, significant associations were found between the left DCP and right SFG and anhedonia severity in anDE.

In summary, aberrant connectivity in corticostriatal and corticolimbic networks is associated with anhedonia severity, particularly involving the ACC and VS.

3.6. Cerebral blood flow

CBF was assessed using arterial spin labeling (ASL) MRI in Dimick et al. (2022). They observed that ACC and MFG cerebral blood flow (CBF) were inversely correlated with anhedonia severity in BD patients (n = 81) compared with HC (n = 75). None of these results remained significant after correcting for multiple comparisons.

3.7. Electrical activity (EEG)

McVoy et al. (2019) found negative correlations between anhedonia scores and theta, alpha and beta coherence in quantitative EEG in MDD adolescents (n = 25). Additionally, average and frontal coherence in the theta band was lower in MDD adolescents compared with HC (n = 14). No associations were found with delta coherence.

Umemoto et al. (2021) reported alpha asymmetry (reduced left posterior power) in MDD adolescents (n = 38), while HC did not show this alpha asymmetry (n = 43). Alpha asymmetry correlated with anhedonia severity across all participants, though not within the MDD group alone.

Zandbagleh et al. (2024) investigated the EEG power spectral density (PSD) of 40 adolescents with MDD and 74 HC, and separated the PSD in periodic (rhythmic) and aperiodic (arrhythmic) components. They found significant negative correlations between BDI scores and parietal theta and frontal alpha power in the entire group of HC and patients with MDD. These correlations were higher in the anhedonia subscale of the BDI.

Wen et al. (2023) linked microstate C → D transitions to higher anhedonia scores and lower attention in MDD adolescents (n = 188). Microstate C is often associated with the default mode network, while microstate D corresponds to dorsal attention network. Additionally,

attention was negatively correlated with anhedonia and positively correlated with dorsal attention network activity. fMRI and EEG convergence suggested dorsal attention network variability mediated the anhedonia–attention relationship.

Dhami et al. (2020) combined treatment with transcranial magnetic stimulation with EEG in 45 adolescents with MDD. Greater anhedonia severity was associated with lower P200 amplitude following dorsolateral PFC (dlPFC) stimulation in adolescents with MDD ($n = 45$).

To summarize, several studies find altered alpha and theta coherence as well as power were associated with greater anhedonia severity. However, the studies differ in the specific patterns. Furthermore, most studies only find these associations in combined samples of HC and MDD, making it unsure whether these patterns are associated with anhedonia severity or suffering from MDD. In addition, one study showed that higher anhedonia scores are associated with a higher N200 amplitude after TMS of the dlPFC. This could mean that anhedonia is a predictor for treatment efficacy, although it remains to be investigated whether this association also translates into therapeutic response.

3.8. Neurotransmission

Gabbay et al. (2012, 2017) assessed neurotransmission correlates using magnetic resonance spectroscopy (MRS) and demonstrated reduced ACC GABA levels in anhedonic MDD adolescents ($n = 20$) compared with non-anhedonic MDD adolescents ($n = 20$) and HC ($n = 21$), with a negative correlation between GABA and anhedonia severity. These findings were replicated in independent samples.

No associations were found between glutamate/glutamine (Glx) and anhedonia severity (Gabbay et al., 2017; Bradley et al., 2018). Bradley et al. (2018) also reported greater striatal vs. ACC GABA in MDD adolescents (n = 20) compared to HC ($n = 16$). Additionally, striatal and ACC GABA were negatively correlated in the MDD group. However, striatal and ACC GABA were not linked to anhedonia.

In conclusion, the first studies show that reduced GABAergic activity, particularly in the ACC, might be a neurochemical correlate of anhedonia in adolescents with MDD.

4. Discussion

4.1. Summary of findings

This scoping review synthesized functional neuroimaging evidence on the neurobiological correlates of anhedonia in adolescents with mood disorders, specifically major depressive disorder (MDD) and bipolar disorder (BD). The 21 included studies were thematically grouped into seven neurofunctional domains: (1) **Reward (incentive) processing**, the most consistently implicated domain, with anhedonia linked primarily to deficits during reward anticipation and error monitoring. Frontal hyperactivation (e.g., dorsolateral prefrontal cortex [dlPFC]) and striatal hypoactivation (e.g., caudate, nucleus accumbens) were frequently observed; (2) **Functional connectivity**, with alterations identified in frontal, corticolimbic, and corticostriatal networks. Notably, diminished dorsomedial prefrontal cortex–anterior cingulate cortex (dmPFC–ACC) connectivity correlated with the severity of anticipatory anhedonia; (3) **Emotional processing**, in which anhedonia severity was negatively associated with corticolimbic activation and positively with corticostriatal responses to negative affective stimuli (e.g., sad or angry facial expressions) and social rejection–related cues. Evidence regarding responses to positive stimuli (e.g., happy faces or reward cues) was limited and less consistent across studies.; (4) **Cognitive processing**, where reduced oxygenated hemoglobin (Oxy-Hb) levels in the medial prefrontal cortex (mPFC), particularly in melancholic MDD subtypes, were observed during executive-function tasks requiring sustained attention and cognitive control, such as verbal fluency and working memory–related paradigms. These reductions were linked to increased anhedonia severity; (5) **Cerebral blood flow (CBF)**, in which one single available study showed no significant associations with anhedonia following correction for multiple comparisons; (6) **Electrical activity**, in which several studies find associations between altered alpha and theta coherence as well as power with greater anhedonia severity and increased dlPFC-evoked potentials in patients with greater anhedonia severity; and (7) **Neurotransmission**, which yielded mixed results—some studies reported decreased gamma-aminobutyric acid (GABA) concentrations in the ACC correlating with anhedonia severity in MDD, while others found no significant associations.

4.2. Emerging patterns and contradictions

Although relatively sparse, current evidence suggests that adolescent anhedonia is characterized by overactivation of frontal cortical regions and underactivation of striatal areas, particularly during reward anticipation. However, contradictory findings emerged, including increased NAc activity in some MDD cohorts and reduced mPFC Oxy-Hb in a fNIRS study, possibly reflecting developmental heterogeneity related to pubertal neuroendocrine status, symptom subtype (e.g., melancholic vs. non-melancholic MDD), and variability in prefrontal–striatal maturation. Notably, anhedonia severity in adolescents with MDD appears to be predominantly associated with the **anticipatory and error-processing phases** of reward processing, rather than consummatory reward experience. These processes may be mediated by disrupted cost-benefit computations—overestimating effort and underestimating reward—previously demonstrated in both human and animal studies (Treadway et al., 2012; Xia et al., 2024).

At the behavioral level, these neural alterations may manifest as reduced exploratory behavior, diminished initiation of goal-directed activity, and motivational withdrawal—hallmarks of adolescent anhedonia that often precede overt reports of reduced pleasure.

Moreover, GABA may be a mediating factor in this circuitry as it is implicated in modulation of dopamine in reward processing regions such as the ACC and mPFC (Der-Avakian and Markou, 2012). Supporting this, transient blockage of GABA receptors in the rat, mPFC showed reduction in willingness to pursue high-effort or risky rewards (Piantadosi et al., 2016). In adolescents, ongoing maturation of inhibitory control systems—interacting with pubertal hormonal fluctuations—may further amplify vulnerability to such motivational impairments.

Additionally, attention-related neural activity was implicated in anhedonia. Reduced alpha and theta coherence and decreased P200 amplitudes in response to attention-related tasks were consistently linked to greater anhedonia severity (McVoy et al., 2019; Wen et al., 2023). Given the increasing academic, social, and self-regulatory demands of adolescence, such attentional inefficiencies may compound motivational disengagement from both social and achievement-related reward contexts. Previous studies have also reported on the involvement of P200 amplitudes in attention-related tasks (Conley et al., 1999; Sokhadze et al., 2017). Taken together, these findings suggest that beyond reward dysfunction, selective attention deficits may also underlie anhedonic symptoms in adolescents with mood disorders.

Importantly, the neuroimaging findings synthesized in this review are broadly consistent with behavioral experimental studies in adolescents with mood disorders. Behavioral work has demonstrated impaired reward learning, reduced approach behavior, diminished exploratory drive, and blunted responsiveness to incentives in anhedonic youth. The observed alterations in anticipatory reward processing, frontostriatal connectivity, and attentional control mechanisms provide a plausible neural substrate for these behavioral phenotypes, linking circuit-level dysfunction to real-world motivational and affective impairments.

Although this review included both major depressive disorder and bipolar disorder, the evidence base was overwhelmingly weighted toward MDD. Only one study specifically examined adolescents with bipolar disorder, precluding meaningful comparison between diagnostic

groups. As a result, the neural mechanisms of anhedonia in bipolar disorder during adolescence remain largely unexplored. Given known differences in reward processing and affective regulation between MDD and BD in adults, it is likely that the neurobiological substrates of anhedonia differ between these conditions. Future studies with adequately powered adolescent BD samples are needed to delineate disorder-specific versus transdiagnostic mechanisms.

4.3. Methodological considerations and limitations

4.3.1. Incentive type and reward phase

The predominance of monetary reward paradigms may limit ecological validity, particularly in adolescents, for whom **social rewards** (e.g., peer acceptance) are developmentally more salient (Forbes and Dahl, 2012). Adolescence is marked by profound changes in social role expectations, increasing peer salience, and heightened sensitivity to social evaluation, which may engage partially distinct reward-processing mechanisms compared to monetary incentives. Emotional salience also varies by task. For example, Henderson et al. (2014) employed explicit emotional face recognition tasks requiring conscious appraisal and labeling of affect, whereas Thai et al. (2020) used implicit socio-affective paradigms that relied more heavily on automatic emotional processing. These differences in cognitive load and processing level likely modulate neural activation patterns, particularly within corticolimbic networks, and may contribute to inconsistencies across emotional processing findings.

4.3.2. Psychometric heterogeneity

A major limitation is the use of diverse anhedonia measures across studies. Tools such as the SHAPS and CDI focus primarily on consummatory pleasure, whereas the TEPS differentiates between anticipatory and consummatory domains. This divergence may lead to inconsistent identification of brain regions associated with anhedonia, particularly as anticipatory anhedonia is more strongly correlated with symptom severity (Ely et al., 2021; Stringaris et al., 2015; Redlich et al., 2015).

4.3.3. Neuroimaging modalities

Only one study specifically employed functional near-infrared spectroscopy (fNIRS) to investigate anhedonia in adolescents, despite its advantages as a youth-friendly and motion-tolerant neuroimaging modality. Liang et al. (2023) reported reduced oxygenated hemoglobin (Oxy-Hb) concentrations in the medial prefrontal cortex (mPFC) among adolescents with melancholic MDD— an observation that contrasts with adult fMRI findings and may reflect developmental differences in prefrontal recruitment during reward-related cognition. Additional fNIRS studies examining broader MDD and BD samples also documented reduced frontal cortical activation in adolescents relative to healthy controls (Liu et al., 2024; Li et al., 2024; Usami et al., 2014; Shin et al., 2024; Ono et al., 2017). Given its superior temporal resolution, cost-effectiveness, and ecological validity, fNIRS represents a promising tool for translational research and clinical implementation in adolescent psychiatry.

4.3.4. Neurochemical imaging

Additionally, methodological limitations are found in neurotransmitter studies as well. Magnetic Resonance Spectroscopy (MRS) studies measuring neurotransmitter concentrations like GABA typically use editing techniques such as J-editing; however, these can be limited as the isolated neurotransmitter signals can contain significant contamination from co-edited macromolecules, making up to 50% of the measured GABA signals complicating interpretations (Bradley et al., 2018; Gabbay et al. (2012, 2017); Gu et al., 2018). These limitations, alongside the small sample sizes in the studies, could potentially explain the contradictory findings between Bradley et al. (2018) and Gabbay et al. (2012, 2017).

4.3.5. Translational insights from animal models

Anhedonia, defined by diminished motivation to pursue, evaluate, and learn from rewarding stimuli, is a core symptom of major depressive disorder (MDD). Despite its clinical significance, the neural mechanisms through which anhedonia influences behavior remain incompletely understood. Emerging evidence from preclinical models offers important insights. For instance, Xia et al. (2025) demonstrated that stress-susceptible mice, relative to stress-resilient counterparts, exhibit a distinct basolateral amygdala (BLA) activation pattern during reward encoding, suggestive of a "rumination-like" neural signature. Such findings provide a useful framework for examining how stress exposure, reward learning, and motivational persistence may interact differently during adolescence—a period characterized by heightened stress sensitivity and evolving social hierarchies.

5. Conclusion

This review focused exclusively on adolescents with clinically diagnosed mood disorders in order to isolate neural correlates specifically associated with established psychopathology. Studies of subthreshold symptoms or high-risk populations (e.g., familial risk) were excluded to minimize confounding effects related to prodromal states or vulnerability markers. Nevertheless, inclusion of high-risk and subthreshold samples represents an important direction for future reviews, as such populations may help identify early neural markers of anhedonia prior to disorder onset. Our scoping review underscores the neurobiological complexity of anhedonia in adolescents with mood disorders, highlighting the central involvement of disrupted anticipatory reward processing and attentional control mechanisms. Integrating pubertal neuroendocrine influences, changing social roles, and behavioral manifestations of anhedonia provides critical developmental context for interpreting heterogeneity in neural findings. Convergent evidence across neuroimaging modalities reveals consistent dysfunction within frontal, corticolimbic, and corticostriatal networks. However, substantial heterogeneity in psychometric instruments, task paradigms, and sample characteristics constrains the generalizability and interpretability of current findings.

To advance the field, future research should prioritize experimental paradigms targeting anticipatory reward phases and socially salient incentives; employ developmentally appropriate, multimodal neuroimaging techniques such as fNIRS; adopt standardized, multidimensional assessments of anhedonia (e.g., TEPS, DARS); expand inclusion criteria to encompass bipolar disorder and transdiagnostic populations; and leverage translational animal models to delineate circuit-level mechanisms.

Conceptualizing adolescent anhedonia as a dynamic and developmentally embedded neurobiological construct is critical for improving early identification and informing targeted interventions capable of disrupting its progression into chronic affective pathology.

CRediT authorship contribution statement

A. Lok: Writing – review & editing, Supervision, Conceptualization. **A.N. Kemble:** Writing – review & editing, Writing – original draft, Validation, Methodology, Investigation, Conceptualization. **F. Mol:** Writing – review & editing, Validation, Project administration, Investigation, Conceptualization. **H. Hoxha:** Writing – review & editing, Validation. **I.O. Bergfeld:** Writing – review & editing, Supervision, Methodology, Conceptualization.

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Declaration of competing interest

The authors have no Conflict of Interest to declare.

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Appendix A. Questionnaire abbreviations

- K-SADS-PL = Kiddie Schedule for Affective Disorders and Schizophrenia Present and Lifetime version
- MFQ = Mood and Feelings Questionnaire
- BDI = Beck Depression Inventory
- MINI-KID = Mini International Neuropsychiatric Interview for Children and Adolescents
- RCADS = Revised Child Anxiety and Depression Scale
- CDRS-R = Children's Depression Rating Scale-Revised
- MADRS = Montgomery Asberg Depression Rating Scale
- SCID = Structured Clinical Interview for DSM-5
- HAMD = Hamilton Rating Scale for Depression
- SHAPS = Snaith-Hamilton Pleasure Scale
- TEPS-(A/C) = Temporal Experience of Pleasure Scale-(anticipatory/consummatory)
- FCPS = Fawcett-Clark Pleasure Scale
- CDI = Children's Depression Inventory

Appendix B. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jad.2026.121376.

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