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Review

Cannabis and Anhedonia in Psychiatric Disorders: A Systematic Review

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Abstract

Background: Cannabis use is common among individuals with psychiatric disorders, but its relationship with anhedonia and reward-processing deficits remains unclear. Anhedonia is multidimensional, encompassing anticipatory and consummatory pleasure, motivation, reward learning, and neurobiological signatures such as blunted striatal dopamine release and reduced reward-related activation in mesocorticolimbic regions. THC, the primary psychoactive constituent of cannabis, acts directly on CB1 receptors within this circuitry. However, findings on how THC-specific cannabis use affects anhedonia and reward-related outcomes across psychiatric populations are inconsistent, varying by dose, frequency, and use pattern. This systematic review synthesizes current literature on THC-specific cannabis use and anhedonia or reward-related outcomes across psychiatric disorders. **Methods:** Following PRISMA 2020 guidelines, Medline, PsycINFO, and Embase were searched through March 2026. Eligible studies included experimental, quasi-experimental, and observational designs assessing cannabis exposure in populations with clinical or subclinical psychiatric disorders or symptoms, reporting at least one subjective, behavioral, or neurobiological measure of anhedonia or reward processing (e.g., anticipatory/consummatory pleasure, effort-based motivation, reward responsiveness, or neural reward activity). **Results:** Twenty studies met inclusion criteria, spanning psychosis-spectrum disorders (13 studies, N = 53,043), MDD/depressive symptoms (8 studies, N = 4,657), bipolar disorder (1 study, N = 103), and anxiety symptoms (1 study, N = 153). In schizophrenia and first-episode psychosis, longitudinal studies consistently linked cannabis use to reduced intrinsic motivation and blunted neural reward sensitivity, while cross-sectional findings were mixed. Among clinical high-risk youth, cannabis use was inversely associated with baseline social anhedonia but did not predict subsequent change. In MDD, cannabis use disorder—rather than general use—predicted greater odds of anhedonia onset over 3–6-year follow-up; five of seven moderate-to-high-quality studies found significant associations. Abstinence/treatment findings were mixed: a 28-day abstinence paradigm reduced self-reported anhedonia, while reduced cannabis use during CBT/MET was linked to reduced ventral striatal reward activation despite depressive symptom improvement. The bipolar study found no association with physical anhedonia; the anxiety study found greater cannabis severity linked to anticipatory but not consummatory anhedonia. **Conclusions:** Evidence most consistently implicates CUD in worsening motivational and anticipatory reward deficits in schizophrenia and MDD. Findings for bipolar and anxiety disorders are limited. Methodological heterogeneity underscores the need for longitudinal, mechanistic studies clarifying causal pathways and disorder-specific vulnerabilities.

Keywords: cannabis; anhedonia; motivation; reward processing; psychiatric disorders; psychosis; depression

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Introduction

Cannabis is the third most frequently used controlled substance globally, after alcohol and tobacco (1). The prevalence of problematic cannabis use in the general population is roughly 3% (2), however the prevalence of cannabis use and cannabis use disorder (CUD) amongst individuals with psychiatric disorders is typically two- to three-fold higher (3). Evidence suggests that cannabis use, particularly products containing high concentrations of the psychoactive component Δ9-tetrahydrocannabinol (THC), may play a role in the initiation and persistence of psychotic disorders (4). Similarly, recent studies have reported significant associations between cannabis use frequency and greater depressive symptom severity (5–7). Despite these associations, the mechanisms underlying the relationship between cannabis use and psychiatric symptom expression remain unclear.

Cannabis acts on the endocannabinoid system, which plays a central role in reward processing, and repeated cannabis exposure may increase susceptibility to anhedonia and reduce sensitivity to rewarding stimuli (8–10). Beyond acute receptor-level effects, chronic THC exposure – particularly during adolescence, when the endocannabinoid system plays a critical role in synaptic pruning circuit maturation – has been linked to structural changes in reward-related brain regions (11). This includes reduced hippocampal volume with regular use, and remodeling of dendritic architecture in the prefrontal neurons following adolescent exposure in preclinical models (11,12). This raises the possibility of a neurotoxic or neurodevelopmental pathway, distinct from acute pharmacological modulation of reward signaling, through which early or sustained cannabis exposure could produce lasting deficits in motivation and hedonic capacity.

Anhedonia, defined in the DSM-5 as a diminished ability to experience pleasure from typically enjoyable activities, is a core feature of several psychiatric disorders, including depressive disorders and psychotic disorders such as schizophrenia (SZ) (19, 20). Historically conceptualized as a unitary construct reflecting an inability or diminished capacity to experience pleasure, it is now increasingly understood as a multifaceted disruption in reward processing. This encompasses distinct, yet related domains, such as anticipatory and consummatory pleasure, effort-based motivation, reward learning, sustained goal-directed behavior, and social and physical reward experiences (15,16). Contemporary models emphasize that these processes unfold across the reward cycle, from initial desire and anticipation, through motivation and effort initiation, to consummatory pleasure, and together define anhedonia as a multidimensional, transdiagnostic construct implicated across multiple psychiatric conditions (14–16).

Historically, research on anhedonia has relied heavily on self-report measures that primarily assessed consummatory pleasure, and do not differentiate between different subdomains of reward processing (17). Newer measures, such as the Dimensional Anhedonia Rating Scale (DARS), aim to capture multiple components of anhedonia, including interest, motivation, effort, and pleasure. In parallel, experimental paradigms such as the Effort Expenditure for Rewards Task (EEfRT), the Joystick-Operated Runway Test (JORT), and the Sweet Taste Test have been developed to evaluate objective aspects of reward sensitivity and effort-based decision-making (18).

Because reward-processing abnormalities, including anhedonia, are a major feature of several psychiatric disorders, namely major depressive disorder (MDD) and SZ, cannabis-related alterations in these systems may have particularly important implications (19). Reward dysfunction has also been reported across other psychiatric conditions, including bipolar disorder (BP) and anxiety disorders, however the extent to which cannabis interacts with reward-related processes in BP and anxiety remains less well characterized (20,21). In such populations, cannabis exposure may interact with underlying reward dysfunction to exacerbate pre-existing deficits across these domains, particularly motivation and apathy, with emerging evidence suggesting that adolescent cannabis use may drive some of these neurobiological and behavioral changes (22,23).

Studies of long-term, regular cannabis users have demonstrated heightened mesocorticolimbic activation (22). In cue-reactivity paradigms, long-term daily cannabis users demonstrate greater activation to cannabis cues than to cues for natural rewards (e.g. palatable food), a pattern which is

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not observed in non-users (23). Such findings are consistent with incentive-sensitization models of addiction, which propose that with repeated drug exposure, drug-related stimuli acquire disproportionate motivational salience relative to natural rewards. Preclinical and human evidence further indicates that sustained THC exposure may result in CB1 receptor downregulation in reward-related regions, a chronic neuroadaptation that could contribute to lasting alterations in hedonic processing and motivational drive (24). Consistent with this, meta-analytic findings report functional alterations in neural networks associated with reward processing, cognitive control, and enhanced reward seeking in cannabis users (25). Although findings remain inconsistent regarding whether cannabis use blunts responses to non-drug rewards, the broader literature on repeated or heavy use generally supports the conclusion that sustained cannabis exposure is associated with measurable alterations in brain activation within reward-related circuitry, particularly the ventral striatum, nucleus accumbens, and prefrontal regions (26–28).

Prior research has examined the relationship between cannabis use and anhedonia within psychiatric populations (26, 27). One observational study on patients with SZ and schizoaffective disorder evaluating anhedonia using a subjective measure found that cannabis users presented a decrease in anticipatory pleasure (31). Another study involving clinical high risk for psychosis (CHR-P) youth found that cannabis users had significantly lower social anhedonia than non-users at baseline and 12 months follow up. Social anhedonia decreased over time in both cannabis users and non-users; no between-group differences in change were observed (32). Psychosis-related conditions span a continuum from clinical high risk states to established schizophrenia spectrum disorders, and cannabis exposure may influence reward-related processes differentially across these stages of illness, as reflected in diverging patterns of associated evident in various studies (33). A longitudinal study in participants with MDD and cannabis use disorder found that cannabis abstinence for 28 days may significantly improve depressive and anhedonia symptoms (34); in contrast, an observational study using ecological momentary assessment found the opposite pattern on a weekly timescale: within individuals, weeks with more cannabis use days and greater average use were associated with lower anhedonia than that same individual's lower-use weeks, through this association was not observed when examined day-to-day(35). This suggests that cannabis may meaningfully interact with reward circuitry, through it remains unclear whether this reflects short-term mood enhancement from cannabis, or people using more when they're already less anhedonic, or another mechanism.

A recent systematic review of 21 studies examined the relationship between cannabis use and anhedonia across depression, psychosis, and schizophrenia populations, as well as non-clinical and animal samples. The authors found evidence for a reciprocal rather than unidirectional relationship: baseline anhedonia appeared to predispose individuals to cannabis use, while subsequent use intensified existing anhedonia, with these effects most pronounced among adolescents and individuals with psychiatric disorders (36). However, this review's scope extended broadly across both THC- and CBD-related effects, and its human evidence synthesis was organized primarily around directionality rather than around specific reward-processing domains. The study did not systematically differentiate anticipatory, consummatory, motivational, and neurobiological components of reward function as distinct, comparable outcomes. Despite calls from the field to adopt a broader, multidimensional conceptualization of anhedonia 21), no synthesis—including the aforementioned—has yet comprehensively examined the full reward-processing cycle across the range of psychiatric diagnoses and illness stages, from subclinical to clinical populations, using a THC-specific lens. It remains unclear whether cannabis use is associated with anticipatory deficits, consummatory impairment, motivational dysfunction, or altered neural reward signaling, and whether these associations differ across psychiatric diagnoses and stages of illness. Accordingly, the present systematic review will examine the association between THC-specific cannabis use and anhedonia or reward-related outcomes by integrating subjective, behavioral, and neurobiological measures of reward-related functioning across a broader range of psychiatric disorders. Specifically, we seek to clarify these domain-specific associations and to explore how cannabis-related disruptions in reward processing may differentially manifest across clinical and subclinical populations.

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Methods

This systematic review was conducted and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines. The review protocol was prospectively registered with the International Prospective Register of Systematic Reviews (PROSPERO; registration number: CRD420261483080)

Search Strategy

A comprehensive search was conducted using Medline, PsycINFO, and Embase databases from inception to March 2026. The search strategy included terms related to 1) cannabis use, 2) anhedonia, motivation and reward-processing and 3) psychiatric disorders. The full search strategy is available in the Supplementary Material. The articles retrieved in our search were uploaded into Covidence to complete title/abstract screening and full text screening. Screening was completed by two independent reviewers (SS, PG), and any discrepancies were resolved through discussion with TPG.

Eligibility Criteria

Inclusion criteria were as follows: 1) Studies employing human participants 2) Peer-reviewed original research including experimental, quasi-experimental, and observational designs; 3) Studies only examining THC-specific cannabis use; 4) Studies assessing for clinical symptoms at a sub-threshold (e.g. CHR-P, depressive and mood symptoms) and threshold level (e.g. MDD, SZ) for all psychiatric disorders and symptoms ; 5) Studies evaluating anhedonia (i.e., impairments in one or more domains of reward processing, including anticipatory pleasure, consummatory pleasure, reward responsiveness, reward learning, effort expenditure for reward, and intrinsic motivation). Relevant assessments included validated self-report measures of hedonic capacity or motivation, clinician-administered instruments with discrete anhedonia components, and established behavioral or neurobiological reward-processing paradigms.

Studies were excluded if they met the following criteria: 1) Studies examining general cannabis use/cannabis use disorder without co-occurring disorders or subthreshold symptoms; 2) Reviews or meta-analyses, case series, case reports, commentaries, opinion papers, unpublished studies, conference posters/published abstracts, dissertations; 3) Published in a language other than English; 4) Studies examining solely CBD; 5) Studies that solely investigated global depressive symptom scores without a clearly defined anhedonia, motivation or reward processing-specific subscale; 6) Studies examining patient motivation to quit; 7) Studies that involved animal models.

Data Extraction & Quality Assessment

Data extraction was completed independently by two reviewers (SS, PG). Extracted variables included: author, year, of publication, number of participants, age of participants, percentage of females in the sample, study design, sample designation (clinical versus non-clinical), clinical diagnostic measures used, psychiatric diagnosis, anhedonia measure and designation (objective or subjective), cannabis use measure, and key findings.

Methodological quality was assessed using design-appropriate tools. The Newcastle-Ottawa Scale (NOS) was used to evaluate methodological quality for observational studies. Studies were designated as high quality if they scored ≥ 7, and low quality if they scored ≤ 4 (37). For randomized controlled trials (RCTs), methodological quality was evaluated using the Cochrane Collaboration Risk of Bias Tool 2.0 (RoB 2) (38). Studies were designated as either low risk, some concerns, or high risk. For quasi-experimental studies, the Risk Of Bias In Non-randomized Studies – of Interventions, Version 2 (ROBINS-I V2) was used. Studies were designated as low risk of bias (except for concerns about uncontrolled confounding), moderate risk of bias, serious risk of bias, or critical risk of bias. The quality ratings were used to inform the interpretation of findings (Tables 1 and 2).

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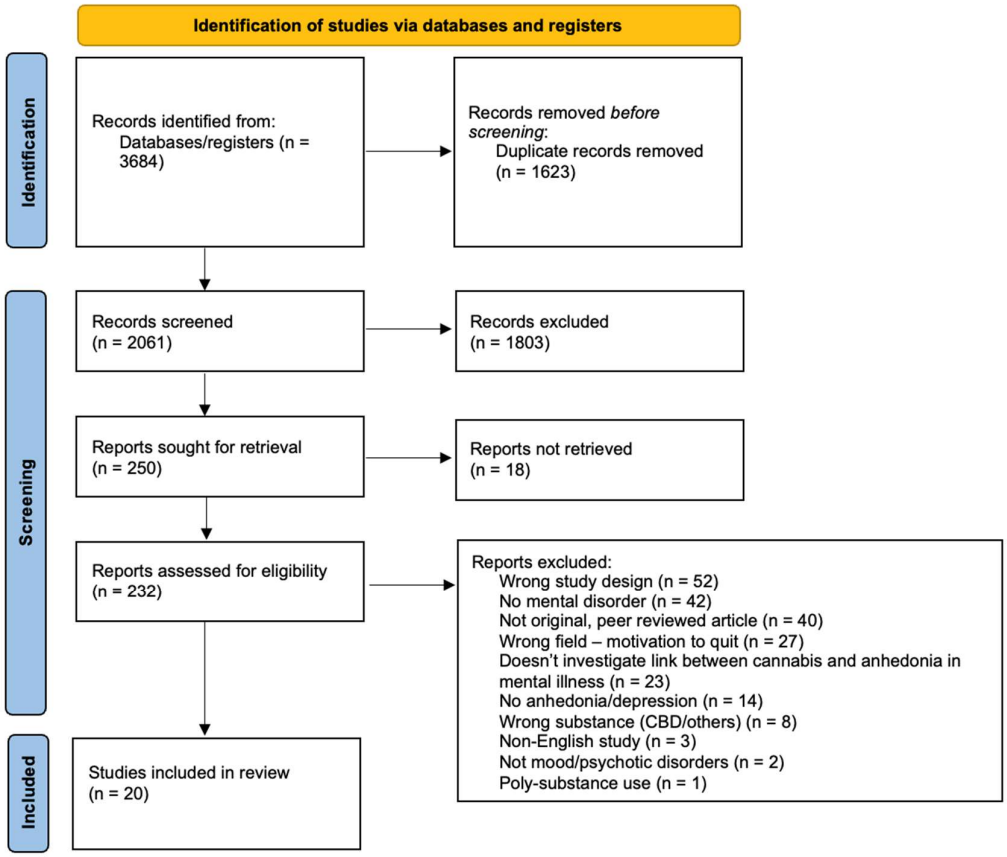

Figure 1. PRISMA Flow DiagramResults.

Study Characteristics

Our search produced 3,684 articles, and after removal of duplicates and screening, 20 studies met our inclusion criteria (see PRISMA diagram, Figure 1). Included studies were published between 2000 and 2025 and varied in study design; seven studies were longitudinal and observational, nine studies were cross-sectional and observational, two were longitudinal and quasi-experimental, one was cross-sectional and experimental, and one was an RCT. Sixteen studies comprised a clinical sample, whereas four were population studies. Of the 20 studies, 13 investigated psychosis-spectrum conditions (N = 53,043 participants), including schizophrenia spectrum disorders (SSD) and CHR-P states, while such eight focused on MDD or depressive symptoms (N = 4,657 participants). Of these studies, one also explored bipolar disorder, and another explored anxiety. One of these studies focused on both MDD and psychotic disorders (39). No study measured a true pharmacological dose (e.g., mg of THC), and only two, Cornelius et al. and Pope et al. specified both quantity and method of use (i.e. blunts/day and joints/day of use, respectively) (40,41). The remaining studies relied on categorical diagnoses or ordinal frequency scales. Thirteen studies involved subjective measures of anhedonia, and three studies involved objective measures, with two studies involving both subjective and objective measures. Of the objective measures employed, only one study employed a behavioral objective measure, the remainder were neurobiological measures (35,42–45). In terms of the study quality ratings, for observational studies, 3 studies were rated to be low quality (35,41,46), 7 studies were rated to of a moderate quality (31,39,45,47–50), and 7 were rated to be high quality (32,42–44,51–53). For the experimental studies, the RCT was rated to have a high risk of bias (40), and for the two quasi-experimental studies, one was rated to have a serious risk of bias (54), and another was rated to have a critical risk of bias (34).

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Psychosis Spectrum Conditions and Cannabis

Thirteen studies examined psychosis spectrum conditions in the context of comorbid cannabis use. Six studies examined CHR-P or early psychosis presentations (32,41,42,46,53,54). Eight studies focused on SSD, namely schizoaffective, schizophreniform, brief psychotic disorder; etc (31,39,41,43,47,48,53,54). Most studies employed clinician-administered diagnostic measures,., although several studies assessed psychosis-related symptoms dimensionally using continuous severity scales and one study employed a self-reported diagnostic measure (41–43,46,50). Study designs included experimental (n = 2), cross-sectional experimental (n = 1), longitudinal quasi-experimental (n = 1), longitudinal observational (n = 5), and cross-sectional observational (n = 6) approaches. Twelve studies involved clinical cohorts, while one utilized a large population-based sample (31,32,39,41–43,45–48,50,53,54).

Longitudinal studies examining cannabis use and anhedonia in psychosis-spectrum conditions were heterogeneous, with patterns differing by diagnostic group. In adults with SZ, one study found that lower baseline intrinsic motivation was associated with baseline cannabis use. Patients using cannabis showed greater deficits in goal-directed behavior and interest, measured by the Intrapsychic Foundations subscale of the Heinrichs-Carpenter Quality of Life Scale, which reflects anticipatory and motivational aspects of reward. At 6-month follow-up, cannabis users showed smaller improvements in intrinsic motivation, and higher baseline motivation predicted reductions in cannabis use (43). Another longitudinal study using a two-choice decision task with monetary incentive cues and fMRI reported that chronic cannabis use in SZ was associated with reduced behavioral and neural sensitivity to anticipated rewards, whereas non-cannabis-using patients and healthy cannabis users demonstrated higher sensitivity to anticipated rewards (48). A quasi-experimental study in antipsychotic-naïve SSD participants found that a lifetime history of CUD moderated the relationship between striatal connectivity, a neurobiological index of reward processing, and treatment response, with little predictive value in those with a CUD history (54).

In contrast, longitudinal studies in youth at (CHR-P) showed null or positive relationships between cannabis use and hedonic capacity. Cannabis users exhibited significantly lower social anhedonia than non-users at both baseline and 12-month follow-up; however, the longitudinal change in social anhedonia did not differ between groups (32). Similarly, in a nationally representative CHR-P cohort, greater social anhedonia at baseline was associated with lower cannabis use frequency and severity at baseline and one-year follow-up (42).

Cross-sectional studies showed mixed results. Several studies in SZ and first-episode psychosis indicated cannabis-related deficits in anticipatory pleasure and reward processing. Adults with SZ or schizoaffective disorder who were regular lifetime cannabis users scored lower than non-users for anticipatory but not consummatory pleasure (31). In first-episode psychosis, persistent abstinence was associated with higher anticipatory pleasure relative to continued use, and greater intensity of cannabis use was linked to decreased anticipatory pleasure, consummatory pleasure, and BAS reward response in patients but not in community controls (45). An experimental study in male SSD patients found that cannabis-using controls showed greater response to pleasant stimuli than to cannabis stimuli, whereas cannabis-using SSD patients showed little bias toward pleasant stimuli. Reduced LPP predicted increased subsequent cannabis use, while SANS anhedonia scores did not differ between patient users and non-users (53). Two studies found no significant differences in physical anhedonia (PAS scores) between SZ males with versus without lifetime CUD (47) and between participants with and without a lifetime history of cannabis abuse/dependence (39).

In first-episode psychosis patients, past three-month cannabis users had lower Anhedonia-Asociality scores than non-users, although cannabis dose was not associated with anhedonia (41). Among CHR-P patients, cannabis use for mood enhancement showed a trend-level association with reduced anhedonia (46). In a non-clinical, population-based genetic study, social anhedonia was negatively correlated with cannabis use frequency, while no significant genetic correlations were observed for physical anhedonia or CUD (50).

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Table 1. Summary of studies examining psychosis spectrum disorders (13 studies, *N* = 53,043).

Author, Year	Study Design	Sample	Diagnostic Measure	Psychiatric Diagnoses	Anhedonia Measure	Subjective/ Objective Measure	Cannabis Use Measure	Key Findings	RoB
Amir et al., 2023	Prospective longitudinal, observational (12 month follow-up)	1,288 clinical high-risk youth, M age = 18.7, 55.7% male	SIPS, SCID	Psychosis (CHR-P)	A single item on the negative subscale of the SIPS	Subjective - clinician	CUD from the SCID, AUS/DUS	In participants at CHR-P, greater social anhedonia at baseline was associated with significantly lower cannabis use frequency and severity at baseline and at one-year follow-up.	NOS, 7
Bahorik et al., 2017	Prospective longitudinal, observational (6 month follow-up)	576 substance-using adults with schizophrenia, M age = 38.3, 80.7% male	SCID	Schizophrenia	Intrinsic motivation evaluated using the sum of 3 items from the Intrapsychic Foundations subscale of the Heinrichs-Carpenter QLS	Subjective - clinician	Self-report (use within past 90 days)	Those with lower baseline motivation levels were more likely to report baseline cannabis use. Cannabis use significantly predicted greater intrinsic motivation deficits at baseline.	NOS, 7
Cassidy et al., 2014	Cross-sectional, experimental	35 patients with schizophrenia-spectrum disorders and 35 nonpsychotic controls, M age = 25.0, 100% male	SCID, SANS, SAPS	Schizophrenia/ schizoaffective disorder	SANS, LPP response	Subjective - clinician, objective (LPP response)	Self-report, CAST	Patients with SSD showed blunted late positive potential (LPP) response to pleasant stimuli as compared to controls. Across measures, cannabis-using controls showed greater response to pleasant stimuli than to cannabis stimuli whereas cannabis-using patients showed little bias toward pleasant stimuli. Reduced LPP response to pleasant stimuli was predictive of more frequent subsequent cannabis use. The LPP captures a reward-processing deficit in patients with SSD. There was no significant difference in SANS anhedonia scores between patient users and non-users.	NOS, 9
Cassidy et al., 2012	Cross-sectional, observational	91 patients with first-episode psychosis and 91 community controls, M age = 25.0, 75% male	SAPS, SANS, SCID	Psychosis patients (affective and non-affective)	TEPS, BAS	Subjective - clinician	Structured interview assessing cannabis use intensity (frequency of use and time since last use); SCID for CUD diagnosis.	Psychosis patients who were persistently abstinent from cannabis reported higher anticipatory pleasure compared to those with continuing use. Greater intensity of cannabis use was strongly associated with decreased anticipatory pleasure, consummatory pleasure and BAS reward response in psychosis patients, but not in controls.	NOS, 5
Dervaux et al., 2010	Cross-sectional, observational	109 male inpatients with schizophrenia, M age = 30, 100% male	DIGS-3.0	Schizophrenia	Chapman Physical Anhedonia Scale	Subjective	DIGS	There were no significant differences between the two groups (schizophrenia patients with versus without lifetime CUDs) on Physical Anhedonia Scale mean scores	NOS, 6
Fish et al., 2021	Longitudinal, observational	34 schizophrenia patients and 49 healthy controls, M age = 27.3, % male = 78.2%	Psychiatrist diagnosed using ICD-10 criteria	Schizophrenia, psychosis, brief psychotic disorder	Reward sensitivity measured using reaction time and fMRI BOLD responses during a	Objective	Self-report (cannabis use history)	Reward sensitivity was increased in control cannabis users and schiozphrenia patient non-users compared to healthy controls who did not use cannabis and schizphrenia patient cannabis users,	NOS, 5

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Author, Year	Study Design	Sample	Diagnostic Measure	Psychiatric Diagnoses	Anhedonia Measure	Subjective/ Objective Measure	Cannabis Use Measure	Key Findings	RoB
					monetary incentive delay task			supporting the hypothesis of reward hypersensitivity in chronic cannabis use. Non-user schizophrenia patients showed increased reward sensitivity compared to non-user controls. Chronic cannabis user patients showed a decrease instead of the expected increase in reward-related sensitivity compared to non-user patients.	
Gill et al., 2015	Cross-sectional, observational	102 clinical high-risk adolescents/young adults, M age = 21.1, 75.5% male.	SIPS/SOPS	Psychosis (CHR-P)	Chapman Physical Anhedonia Scale	Subjective	Self-report (use within past 30 days), Reasons for Use Scale	In cannabis using CHR-P patients, the motive for 'enhancement' had a moderate association with anhedonia at a trend level of significance. Anhedonia means differed in CHR users vs non-users descriptively, but no inferential test was conducted.	NOS, 2
Liraud & Verdoux, 2000	Cross-sectional, observational	103 inpatients, M age = 36, 39.8% male	MINI	MDD, schizophrenia, non-affective psychotic disorders (schizoaffective disorder, delusional disorder, psychotic disorder not otherwise specified)	Chapman Physical Anhedonia Scale	Subjective	Lifetime history of CUD using the MINI	Physical anhedonia scale scores did not differ between participants with and without a lifetime history of cannabis abuse/dependence.	NOS, 6
Pope et al., 2021	Cross-sectional, observational	231 first-episode psychosis patients, M age = 23.9 years, 74.0% male	SCID, SANS, SAPS	Psychosis (first-episode), Schizophrenia-spectrum disorders	Anhedonia-asociality subscale of SANS	Subjective - clinician	LSUR-12, self-report (calendar based assessment of use of previous 12 weeks)	The Anhedonia-Asociality score was significantly lower among those using cannabis in the past three months than among those not using cannabis. Among participants who used cannabis in the past three months, cannabis dose was not associated with anhedonia.	NOS, 4
Santesteban-Echarri et al., 2022	Prospective longitudinal, observational (2 year follow-up)	699 clinical high-risk youth (plus 96 healthy controls), M age = 18.2, 54.4% male	SIPS, SOPS	Psychosis (CHR-P)	SOPS	Subjective - clinician	SCID for CUD; AUS/DUS for cannabis use frequency; also self-reported age at first use & # of times used in past 6 months	Clinical high risk cannabis users had significantly lower social anhedonia than non-users at baseline and 12 months follow up. Social anhedonia decreased over time in both cannabis users and non-users; no between-group differences in change were observed.	NOS, 7
Schnakenberg Martin & Lysaker, 2020	Cross-sectional, observational	71 middle-aged adults with schizophrenia/schizoaffective disorder, M age = 49.6, 84.5% male	PANSS	Schizophrenia/schizoaffective disorder	TEPS	Subjective	ASI, regular cannabis use as indicated by # of months they've consumed cannabis greater or equal to 3 times per week in their lifetime - approximately the middle third of subjects were	Regular lifetime users scored significantly lower than non-users on anticipatory pleasure, but not consummatory pleasure. Anticipatory pleasure was positively associated with prosocial functioning in participants without regular cannabis use, but this association	NOS, 5

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Author, Year	Study Design	Sample	Diagnostic Measure	Psychiatric Diagnoses	Anhedonia Measure	Subjective/ Objective Measure	Cannabis Use Measure	Key Findings	RoB
							omitted from the analysis based on reported cannabis consumption to create two distinct groups of little to no (zero months) and moderate (greater or equal to 3 years) regular lifetime cannabis use	was not present in participants with three or more years of regular cannabis use. Cannabis use did not moderate the relationship between emotional expressivity and anticipatory pleasure.	
Thies et al., 2020	Prospective longitudinal, quasi-experimental (3 month follow-up)	48 antipsychotic-naïve patients with schizophrenia spectrum disorders, M age = 20.7, 70.8% male	SCID, CGI, BPRS	Schizophrenia spectrum disorders (schizophrenia, schizophreniform disorder, schizoaffective disorder, or psychotic disorder not otherwise specified)	Functional magnetic resonance imaging (fMRI) used to investigate striatal connectivity index (SCI), which is involved in reward processing	Objective	CUD using SCID	A lifetime history of CUD moderated the relationship between striatal connectivity index (reward processing regions) and treatment response such that it had little predictive value in schizophrenia spectrum disorder (SSD) patients with a CUD history.	ROBINS-I V2, Serious risk
Vaissiere et al., 2020	Cross-sectional, observational	UK Biobank and international GWAS participants 51,372 CUD users and 8013 adults with anhedonia	Self-report schizotypy items (UKB) and Chapman schizotypy scales - Revised Physical Anhedonia Scale, the Revised Social Anhedonia Scale, the Perceptual Aberration Scale and the Hypomanic Personality Scale (NFBC 1966)	Schizotypy/subclinical psychosis	Chapman Physical Anhedonia Scale	Subjective	Self-reported 'number of times used cannabis' and 'maximum frequency of cannabis use', CUD using ICD-10 criteria	One significant genetic correlation was observed between schizotypy and cannabis use, a negative correlation between social anhedonia and number of times used cannabis. None of the genetic correlations between cannabis phenotypes and physical anhedonia were significant, and social anhedonia showed no significant genetic correlations with cannabis use disorder or maximum frequency of cannabis use.	NOS, 6

Note. SIPS = Structured Interview for Prodromal Syndromes; SCID = Structured Clinical Interview for DSM Disorders; CHR-P = Clinical High Risk for Psychosis; AUS = Alcohol Use Scale; DUS = Drug Use Scale; QLS = Heinrichs-Carpenter Quality of Life Scale; SANS = Scale for the Assessment of Negative Symptoms; SAPS = Scale for the Assessment of Positive Symptoms; CAST = Cannabis Abuse Screening Test; TEPS = Temporal Experience of Pleasure Scale; BAS = Behavioural Approach System Reward Response subscale; DIGS = Diagnostic Interview for Genetic Studies; PANSS = Positive and Negative Syndrome Scale; LSUR-12 = Longitudinal Substance Use Recall (12-year version); ASI = Addiction Severity Index; BPRS = Brief Psychiatric Rating Scale; CGI = Clinical Global Impressions Scale; ICD-10 = International Classification of Diseases, 10th Revision; MINI = Mini International Neuropsychiatric Interview; SOPS = Scale of Prodromal Symptoms; LPP = Late Positive Potential.

Depression and Cannabis

Eight studies examined the relationship between cannabis use, depression and anhedonia (34,35,39,40,44,49,51,52). Study designs included three cross-sectional observational studies, three longitudinal observational studies, one single-arm trial and one RCT. Diagnostic approaches varied, with most studies using structured clinical interviews to assess MDD, while others examined depressive symptoms dimensionally. Anhedonia was assessed using both subjective self-report

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measures and an objective neurobiological indices, specifically fMRI BOLD activation during a card-guessing monetary reward task and during passive music listening (40,52). Cannabis exposure was operationalized heterogeneously, including frequency of use, quantity of use, and diagnoses of CUD.

Three cross-sectional studies examined the relationship between cannabis and anhedonia in depression and reported heterogeneous findings. Among youth with MDD, self-reported anhedonia did not differ between frequent cannabis users and non-users (55), and in a diagnostically heterogeneous inpatient sample that included MDD, physical anhedonia did not vary by lifetime CUD history (39). However, more granular analyses suggested domain- and sex-specific effects. In adolescents with elevated affective symptoms, cannabis use was associated with greater anticipatory but not consummatory anhedonia with effects observed only in females (49). Moreover, despite no self-report differences in youth with MDD, neuroimaging revealed altered reward-related activation, with cannabis-using participants demonstrating greater BOLD activation to preferred versus neutral music in frontal, cingulate, and striatal regions, and medial frontal activation positively correlated with cannabis use in the prior 28 days (52).

Five longitudinal studies assessed temporal relationships between cannabis use and anhedonia in depression. In two observational community-based samples, cannabis use was associated with subsequent anhedonia. In adults with past-year MDD, meeting criteria for CUD was associated with greater odds of reporting anhedonia over a three-year follow-up period, whereas cannabis use without CUD did not (51). Similarly, in a community-based cohort without baseline depressive symptoms, the incidence of anhedonia was four times greater in individuals with CUD than in individuals without CUD at follow-up 4-6 years later (44). One observational ecological momentary assessment study reported within-person associations between anhedonia and cannabis use. In weekly models, lower anhedonia scores were associated with more cannabis use days and greater average use in the same and following week, with significant effects primarily observed among regular users. However, in daily models, anhedonia was not associated with use quantity and was only variably associated with the likelihood of any cannabis use (35). Two studies employed abstinence-based designs to document changes in anhedonia and reward processing over time. In a quasi-experimental abstinence paradigm, adults with comorbid MDD and CUD demonstrated significant reductions in anhedonia (SHAPS) over a 28-day period of biochemically confirmed cannabis abstinence, with changes in cannabis exposure significantly associated with changes in anhedonia (34). In an experimental fMRI study, reductions in cannabis use during Cognitive Behavioral Therapy/Motivational Enhancement Therapy were associated with reduced reward-related activation in the ventral striatum and related regions, contrary to the authors' hypothesis (40).

Table 2. Summary of studies examining MDD and depressive symptoms with comorbid cannabis use (8 studies, N = 4, 657).

Author, Year	Study Design	Sample	Diagnostic Measure	Psychiatric Diagnoses	Anhedonia Measure	Subjective/ Objective Measure	Cannabis Use Measure	Key Findings	RoB
Bovasso, 2001	Prospective longitudinal, observational (12-14 year follow-up)	1,920 community-dwelling adults; mean age not reported, gender not reported	DIS	Depression/depressive symptoms	DIS	Subjective - clinician	Meeting criteria for CUD using DIS	In participants with no baseline depressive symptoms, those with a diagnosis of CUD at baseline were four times more likely to have depressive symptoms at the follow-up assessment, after adjusting for covariates. Post-hoc analyses found that anhedonia and suicidal ideation were the specific depressive symptoms predicted by baseline CUD; the incidence of anhedonia was approximately four times greater in cannabis abusers than in nonabusers.	NOS, 9
Collins et al., 2024	Prospective 90-day longitudinal, observational	55 adults with MDD and reported cannabis use, modal age = 25–34, 18.2% male	PHQ-9, SCID	MDD	Anhedonia item of the PHQ-9	Subjective	Self-report (TLFB)	Lower anehdonia is associated with more days of cannabis use in the same week, but it is not associated with distinguishing between any	NOS, 3

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Author, Year	Study Design	Sample	Diagnostic Measure	Psychiatric Diagnoses	Anhedonia Measure	Subjective/ Objective Measure	Cannabis Use Measure	Key Findings	RoB
								and no cannabis use in the same week. Lower levels of average anhedonia predicted more cannabis use than usual in the next week. Daily anhedonia is associated with same day and next day cannabis use in regular users but not low cannabis consumers, but daily anhedonia is associated with using at least some cannabis during the next day in all consumers.	
Cornelius et al., 2013	Prospective longitudinal, randomized controlled trial (3 month follow-up)	6 youth with comorbid cannabis dependence and major depressive disorder, M age = 21.7 years, 83.3% male	SCID, K-SADS	MDD, CUD	Reward sensitivity measured by fMRI BOLD activation in the ventral striaturm during a card-guessing monetary reward task	Objective	SCID for CUD at baseline, self-report (frequency of use, # of blunts/week)	Higher baseline cannabis use was associated with lower ventral striatal reward-related activation. Over treatment, overall reward-related neural activation decreased; however the 5 individuals who decreased their cannabis use (mean decrease of 63.6% blunts per week) demonstrated an increase in their level of ventral striatal reactivity while the one subject that demonstrated an increase in their level of cannabis use (a 79% increase), demonstrated a decrease in their level of ventral striatal reactivity.	RoB 2, High risk
Feingold et al., 2017	Prospective longitudinal, observational (3 year follow-up)	2,348 adults with past-year MDD, modal age = 30–44, 31.4% male	AUDADIS-IV	MDD	Anhedonia item from the AUDADIS-IV	Subjective	AUDADIS-IV to determine if they met CUD criteria, Self-report (cannabis use frequency)	Exploring specific MDD symptoms, after controlling for confounding factors, individuals with a CUD had significantly greater odds to report anhedonia. Levels of cannabis use (no use, users without a CUD and those with a CUD) were associated with increased number of depressive symptoms at follow-up, particularly differences in prevalence of anhedonia at follow-up compared to nonusers. Individuals with CUD demonstrate more anhedonia at follow up , although cannabis users without CUD did not.	NOS, 8
Ford et al., 2014	Cross-sectional, observational	61 late adolescents/young adults, M age = 19.8, 45.9% male	BDI-II, SCID, HDRS	MDD	SHAPS, reward processing as measured by fMRI BOLD activation during music listening, and subjective environment of music listening was assessed by asking participants to rate each of the music stimuli, using the same standardized scale (1) how much did you like the neutral/preferred music you just heard on a scale from 0 to 100; (2)	Subjective (SHAPS, enjoyment of music listening using 3 items), objective (objective reward processing as measured by fMRI BOLD activation during music listening)	Use in past 28 days was measured by TLFB, self-report (frequency, recent use status), frequent use confirmed by urine screen	During passive music listening, youth with combined MDD and frequent cannabis use showed greater BOLD activation to preferred vs neutral music in frontal, cingulate, and striatal regions; this effect was not observed in MDD-only, cannabis-only, or healthy control groups. Activation in medial frontal regions was positively correlated with cannabis use in the past 28 days. MDD cannabis users did not differ significantly from MDD-only participants. Participants with MDD and frequent cannabis use reported significantly higher SHAPS scores (i.e., greater anhedonia) compared with marijuana users without MDD.	NOS, 7

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Author, Year	Study Design	Sample	Diagnostic Measure	Psychiatric Diagnoses	Anhedonia Measure	Subjective/ Objective Measure	Cannabis Use Measure	Key Findings	RoB
					how much would you like to hear this neutral/preferred music again on a scale from 0 to 100; and (3) to rate the neutral/preferred music you just heard on a scale from −100 to +100				
Liraud & Verdoux, 2000	Cross-sectional, observational	103 inpatients, M age = 36, 39.8% male	MINI	MDD, schizophrenia, non-affective psychotic disorders (schizoaffective disorder, delusional disorder, psychotic disorder not otherwise specified)	Chapman Physical Anhedonia Scale	Subjective	Lifetime history of CUD using the MINI	Physical anhedonia scale scores did not differ between participants with and without a lifetime history of cannabis abuse/dependence.	NOS, 6
Lucatch et al., 2020	Prospective longitudinal, quasi-experimental (1 month follow-up)	11 adults with comorbid major depressive disorder and cannabis use disorder, M age = 30.0, 35.7% male	SCID, HDRS	MDD, CUD	SHAPS	Subjective	TLFB, CUDIT-R, MWC, Narcocheck THC-COOH Assay	During a 28-day biochemically confirmed cannabis abstinence , anhedonia significantly improved, with reductions observed on the SHAPS total score over time, an 88.7% decrease from baseline, and significant associations between THC reduction and SHAPS improvement; The HDRS-17 item indexing anhedonia, specifically consummatory pleasure, motivation and interest (work and interests) also showed significant improvement.	ROBINS-I V2, Critical risk
Nguyen et al., 2025	Cross-sectional, observational	153 adolescents, M age = 15.9, 35.9% female	BDI-II, MASC, K-SADS	MDD, CUD	TEPS	Subjective	K-SADS, CUPIT, self report (daily sessions, frequency, age of onset, quantity), urine toxicology	Analyses showed that relative to those who never used or had only tried cannabis once, adolescents who used cannabis endorsed worse anticipatory anhedonia, but not consummatory anhedonia.	NOS, 6

Note. DIS = Diagnostic Interview Schedule; PHQ-9 = Patient Health Questionnaire-9; SCID = Structured Clinical Interview for DSM Disorders; TLFB = Timeline Followback; K-SADS = Kiddie Schedule for Affective Disorders and Schizophrenia; AUDADIS-IV = Alcohol Use Disorder and Associated Disabilities Interview Schedule (Fourth Edition); BDI-II = Beck Depression Inventory-II; HDRS = Hamilton Depression Rating Scale; SHAPS = Snaith-Hamilton Pleasure Scale; MINI = Mini International Neuropsychiatric Interview; CUDIT-R = Cannabis Use Disorder Identification Test–Revised; MWC = Marijuana Withdrawal Checklist; TEPS = Temporal Experience of Pleasure Scale; CUPIT = Cannabis Use Problems Identification Test.

Other Disorders and Cannabis

Two cross-sectional observational studies in BP and anxiety were included (39,49). The first study investigating BP was conducted in an urban inpatient unit at the University of Bordeaux psychiatric hospital, and found no association between cannabis use and physical anhedonia scores,

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even after adjustment for a diagnosis (39). The second study recruited youth with anxiety symptoms in the New York Metropolitan area found that across the sample, more severe cannabis use correlated with worse anticipatory anhedonia, with no significant difference for consummatory anhedonia levels (49).

These findings demonstrate that there may be a relationship between psychiatric illness and cannabis use for anxiety, as cannabis use was associated with worse anticipatory anhedonia, however this finding is not reflected for consummatory anhedonia, and no significant relationship was apparent for BP and physical anhedonia (39,49).

Table 3. Summary of studies examining miscellaneous symptoms and psychiatric disorders with comorbid cannabis use (2 studies, N = 266).

Author, Year	Study Design	Sample	Diagnostic Measure	Psychiatric Diagnoses	Anhedonia Measure	Subjective/ Objective Measure	Cannabis Use Measure	Key Findings	RoB
Liraud & Verdoux, 2000	Cross-sectional, observational	103 inpatients, M age = 36, 39.8% male	MINI	Bipolar disorder	Chapman Physical Anhedonia Scale	Subjective	Lifetime history of CUD using the MINI	Physical anhedonia scale scores did not differ between participants with and without a lifetime history of cannabis abuse/dependence.	NOS, 6
Nguyen et al., 2025	Cross-sectional, observational	153 adolescents, M age = 15.9, 35.9% female	BDI-II, MASC, K-SADS	Anxiety symptoms	TEPS	Subjective	K-SADS, CUPIT, self report (daily sessions, frequency, age of onset, quantity), urine toxicology	Adolescents who used cannabis endorsed worse anticipatory anhedonia relative to those who never used or had only used cannabis once.	NOS, 6

Note. MINI = Mini International Neuropsychiatric Interview; BDI-II = Beck Depression Inventory–II; MASC = Multidimensional Anxiety Scale for Children; K-SADS = Kiddie Schedule for Affective Disorders and Schizophrenia; TEPS = Temporal Experience of Pleasure Scale; CUPIT = Cannabis Use Problems Identification Test; NOS = Newcastle–Ottawa Scale.

Discussion

This systematic review integrated findings related to anhedonia and motivation outcomes for cannabis users across psychiatric diagnoses. Although our search targeted a broad range of psychiatric disorders, relevant published studies were identified only for psychotic disorders, major depressive disorder (MDD), bipolar disorder, and anxiety disorders. Findings were largely heterogeneous, particularly for psychotic disorders, with a majority of studies (6 of 10) reporting deleterious effects of cannabis use on anhedonia, though a substantial minority (4 of 10) found no effect; direction of effect depended on baseline symptoms and the type of anhedonia assessed. Similarly, in MDD, most studies (5 of 7) linked cannabis use—specifically CUD—to higher anhedonia, with reduced cannabis use demonstrating improvements in reward-related outcomes. Evidence for bipolar disorder and anxiety was limited to a single study each, with the bipolar study finding no effect of cannabis and the anxiety study finding an adverse effect.

Clinical domain	Number of studies supporting an adverse effect of cannabis	Number of studies supporting no effect of cannabis	Number of studies supporting a therapeutic effect of cannabis	General findings
MDD Symptom or Diagnosis Development	2	0	0	Cannabis use disorder (CUD) appears to be associated with increased risk of developing anhedonia and depressive symptoms over time (100% of studies).

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Clinical domain	Number of studies supporting an adverse effect of cannabis	Number of studies supporting no effect of cannabis	Number of studies supporting a therapeutic effect of cannabis	General findings
BD Symptom or Diagnosis Development	0	1	0	Limited evidence suggests no clear association between cannabis use and anhedonia or symptom development in BD (100% of studies).
Schizophrenia Spectrum Disorder Symptom or Diagnosis Development	4	4	2	Evidence in SSD populations is mixed; cannabis use is often associated with impairments in anticipatory pleasure and reward processing, though several studies report null or potentially protective associations (approximately 40% adverse).
Prognosis of MDD	3	1	1	Cannabis use, particularly CUD, is generally associated with worse reward-related outcomes in MDD, though reductions in use or abstinence may improve anhedonia symptoms (60% adverse).
Prognosis of BD	0	1	0	Evidence is extremely limited and does not indicate a clear relationship between cannabis use and reward-related outcomes in BD.
GAD Symptom or Diagnosis Development	1	0	0	Limited evidence suggests cannabis use may be associated with worse anticipatory anhedonia among individuals with anxiety symptoms (100% of studies).
Prognosis of Schizophrenia / SSD	3	2	1	In established psychotic disorders, cannabis use is frequently associated with poorer reward sensitivity and intrinsic motivation, although some studies report null or potentially protective associations depending on the domain assessed (~50% adverse).

Psychotic Disorders

The relationship between cannabis use and anhedonia in psychosis spectrum populations appears to vary depending on diagnosis, clinical status, and study design. Longitudinal studies in SZ consistently indicate that cannabis use is associated with greater deficits in intrinsic motivation and reduced reward sensitivity, suggesting that cannabis may exacerbate underlying motivational impairments in this population (43,48). In contrast, CHR-P youth demonstrated a different pattern, where social anhedonia is inversely related to cannabis use at baseline and predicts lower use over time, while cannabis itself does not appear to alter anhedonia longitudinally (42). Consistent with this, a cross-sectional study of CHR-P participants reported that cannabis use was most commonly motivated by mood enhancement, followed by social factors, with symptom relief rarely cited; mood enhancement showed only trend-level associations with anhedonia, and social motives were linked to anxiety (46). Importantly, this study was of low methodological quality and descriptive in nature, underscoring the need to interpret these patterns cautiously. Together, these findings suggest that cannabis use in CHR-P youth may be socially and emotionally motivated rather than reflecting intrinsic reward deficits (32,56).

Cross-sectional and experimental studies in SZ and first-episode psychosis provide a more mixed picture. Some report that cannabis use is associated with reduced anticipatory and consummatory pleasure, blunted reward responsiveness, and altered neurophysiological markers of reward processing, including reduced late positive potential responses (31,45,53). Notably, the experimental study carried a high risk of bias, while the quasi-experimental studies were at serious or critical risk of bias, which should be considered when interpreting findings. Other cross-sectional studies report no significant differences in behavioral or genetic measures of anhedonia, particularly for physical anhedonia or self-reported scores (39,41,47,50,53), indicating that potential effects of cannabis may be selective to certain domains of reward processing or may depend on the measure used. It is important to note that the study conducted by Pope and colleagues was also of a low methodological quality. However, these results overall suggest a differential impact of cannabis on reward and motivational processes across psychotic-spectrum populations. In SZ and first-episode

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psychosis, cannabis use appears to exacerbate intrinsic deficits in reward and motivation, whereas in CHR-P youth, use may be influenced by mood and social factors rather than direct effects on anhedonia. This highlights the importance of considering diagnostic status, social context, and methodological quality when assessing cannabis-related motivational outcomes.

Depressive Disorders

When our analysis was constricted to moderate-to-high quality studies, cannabis use was generally associated with anhedonia in MDD samples: 5 of 7 studies found significant associations, while two self-report cross-sectional studies were negative. One study found domain-specific effects in adolescents, with cannabis use linked to anticipatory anhedonia only in females (49), comparable to trends observed in young adult patients with psychosis and suggesting that adolescent cannabis use may impact motivation rather than experiential pleasure (45,49). Prospective studies indicate that CUD, rather than cannabis use, may increase risk for certain depressive symptoms such as anhedonia and suicidal ideation, with baseline depressive symptoms not predicting later cannabis use, supporting a unidirectional pathway from cannabis to symptom emergence (44,51). Observational designs limit causal inference, but these findings suggest CUD may selectively exacerbate certain depressive domains rather than overall severity. Abstinence and treatment studies provide additional mechanistic evidence. In the open-label study by Lucatch et al., 28-day biochemically-verified cannabis abstinence was associated with robust reductions in anhedonia (SHAPS), as well as improvements in motivation and interest on the HDRS-17, indicating potential reversibility of reward deficits. Dose–response associations between THC reduction and anhedonia further support a potential substance-related effect (34). In contrast, Cornelius et al. found that decreases in cannabis use during CBT/MET corresponded with reduced ventral striatal reactivity to non-drug reward, despite improvements in depressive symptoms, suggesting that cannabis and depression exert opposing effects on striatal reward circuits (40). These findings highlight the need to distinguish between subjective anhedonia and neural reward responses. Together, the evidence supports a role for cannabis-related reward modulation in anhedonia amongst individuals with comorbid MDD and CUD, underscoring the need for high-quality longitudinal studies to clarify causal mechanisms and the direction of neural change.

Other Disorders

The null findings regarding cannabis use and physical anhedonia in patients with BP may reflect that anhedonia is largely state-dependent in bipolar disorder, despite evidence in the literature that some individuals exhibit residual trait-like anhedonia between mood episodes (57). Longitudinal studies are needed to clarify this distinction and to explore any potential causal relationship between trait versus state anhedonia and cannabis use (39). For the study examining anxiety disorders, the differential outcome for anticipatory versus consummatory anhedonia demonstrates that adolescent cannabis use may specifically impair reward anticipation and motivation rather than the capacity to feel pleasure (49). This may be due to a propensity in adolescents with low motivation and reward deficits to use cannabis to compensate for these deficits (58), along with neurobiological changes resulting from cannabis use targeting specifically the corticostriatal circuits involved in reward motivation, leaving regions related to reward experience intact (59).

Strengths and Limitations

It is important to consider the findings from this review in the context of limitations present in the literature and methodology. First, half of the studies integrated into this review were cross-sectional, limiting the ability to examine prospective effects to evaluate causality. Furthermore, variability in study design, including the inclusion of quasi-experimental studies, contributed to elevated risk of bias ratings. Due to their non-randomized nature, these studies remain vulnerable to residual or unmeasured confounders. Accordingly, all findings should be interpreted considering

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these quality ratings and the associated limitations in internal validity. Several studies also possessed sample biases or uneven demographic representation. Two studies included exclusively male samples, and 12 studies included majority male samples (over 50% of participants) (47,53).

The search strategy for this review also yielded a relatively small pool of 20 eligible studies. Analyses for BP and anxiety disorders also were not stratified by diagnosis, limiting conclusions about disorder-specific effects. Other diagnoses were also identified in these studies, but were not integrated into analyses, necessitating future work integrating more key psychiatric populations. Furthermore, all studies with objective anhedonia measures were neurobiological (e.g. fMRI, LPP response) measures except one, demonstrating a further need for use of objective behavioral measures of anhedonia. Additionally, in nearly half of included studies (9 of 20), anhedonia or reward-related function was not the primary outcome of interest but rather a secondary or exploratory measure embedded within a broader symptom or biomarker battery. Furthermore, no study measured a true pharmacological dose, though two specified both a quantity measure and method of use, not accounting for THC potency or cannabis quantity per unit (40,41). Two studies with biochemical verification used urine THC-COOH levels primarily to confirm recent use or abstinence rather than to quantify exposure, and the Lucatch et al. study quantifying a biochemical measure did not specify method of use (34,49). The remaining 17 studies relied on categorical diagnoses, ordinal frequency scales, or binary use variables, limiting exposure precision and precluding dose-response analyses.

However, this review also possesses several strengths. First, data extraction and quality assessment for this review were conducted independently by two reviewers (SS and PG) to ensure consistency and reliability. To minimize the potential for bias influencing the findings, observational studies of low methodological quality were identified and addressed in the synthesis. This review also fills a gap in the understanding and conceptualization of anhedonia deficits in cannabis users with co-occurring psychiatric diagnoses and concerns. The examination of a wide range of diagnoses and clinical presentations across psychotic disorders and depression, including anxiety disorders, SSD, CHR-P, MDD, and BP, may provide a comprehensive analysis of how substance use relates to varying severities of co-occurring psychiatric conditions.

Conclusions and Clinical Implications

Clinically, these findings highlight the need for increased attentiveness towards substance using populations across psychiatric spectrum-conditions, as cannabis use may exacerbate existing motivational impairments in SZ and first-episode psychosis (43,45,48,53). Integrated interventions that utilize both psychiatric treatment and substance use management may be more effective for those with dual diagnoses (60). For individuals with CUD in particular, reductions in cannabis may yield measurable improvements in anhedonia and motivation, as observed in abstinence-based studies (34,40). Preventive strategies targeting high-risk adolescents and young adults are critical, given the evidence that early or frequent use may precipitate or amplify reward-processing deficits (45,49).

These findings also underscore the importance of reconceptualizing anhedonia in clinical practice. Rather than treating anhedonia as a unitary symptom, clinicians should account for its multidimensional nature, including differentiating anticipatory versus consummatory pleasure, motivation, and effort-based components (15,16,18) as well as multifactorial contributors to anhedonia including inherent aspects of the psychiatric disorders and cannabis use. Comprehensive assessment using both subjective and objective measures may guide more tailored patient interventions, potentially improving clinical monitoring of reward deficits.

Preventive education and early intervention programs should be prioritized to reduce harm, especially for individuals with genetic vulnerability or exposure to social and environmental risk factors such as childhood adversity and urban upbringing (61,62). Routine screening for cannabis use, combined with motivational and cognitive-behavioral strategies in standard psychiatric care, may help mitigate its adverse effects on anhedonia and broader reward-processing deficits (60).

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However, further research is needed to clarify the relationship between cannabis use and motivational deficits in psychiatric conditions, including other psychiatric disorders such as posttraumatic stress disorder (PTSD), obsessive-compulsive disorder, and personality disorders.

Supplementary Materials: The following supporting information can be downloaded at the website of this paper posted on Preprints.org.

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