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Anhedonia: A Comprehensive Narrative Review

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Abstract

Anhedonia is a transdiagnostic symptom defined as the lack of pleasure due to disruptions in reward processing pathways, specifically in the prefrontal cortex and striatum. This paper aims to investigate the complex nature of anhedonia as observed in many psychiatric disorders, as well as analyze its neurobiological mechanisms, computational models, and treatment options. To do this, we completed a thorough review of recent literature, including developments in the field. The literature shows that current interventions aimed at treating anhedonia are also often inadequate, which leads to a greater need for novel treatments like neurostimulation and personalized therapeutic models. Many studies suggest that using genetic and behavioral data could also improve treatment efficacy. Further research is necessary to develop individualized models and interventions for anhedonia. Future studies in the field should be aimed at exploring the social context of anhedonia and integrating computational models with neurobiological components to improve treatment options.

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Introduction

Théodule Ribot first defined anhedonia in 1897 as the inability to enjoy usually pleasurable activities or experiences.[1] More recently, the DSM-5-TR defined anhedonia as a loss of pleasure or interest in response to stimuli previously perceived as rewarding during a pre-morbid state.[2] The clinical manifestations of anhedonia include difficulty feeling pleasure, engaging in goal-directed activities, and adapting to changing circumstances. According to the DSM-5-TR, anhedonia, along with depressed mood, is one of the core symptoms for the diagnosis of a Major Depressive Episode and the "with melancholic features" specifier for Major Depressive Disorder (MDD).[2] It is also reported in other disorders such as schizophrenia, anxiety disorders, and post-traumatic stress disorder (PTSD). According to Snaith[3] and Treadway and Zald,[4] anhedonia has long been neglected in psychopathology, despite its pervasive impact on mood, motivation, and social and cognitive performance. Its significance has grown considerably in recent years as more researchers have been interested in investigating its transdiagnostic relevance in psychiatry.

The multidimensional nature of anhedonia, which includes a decrease in pleasure from physical or sensory experiences as well as a decreased desire

to engage in pleasurable activities, has important implications for understanding its role in psychiatric disorders. According to Pelizza and Ferrari,[5] anhedonia could be a state marker that fluctuates with mood and symptom severity, or a trait marker that reveals underlying vulnerabilities in persons predisposed to depression and schizophrenia. A state marker is a measure of a patient's current symptoms or condition and a trait marker is a biological factor that shows their natural risk for developing major mental disorders and plays a role in causing these conditions.[6] Fortunati *et al*.[7] highlight the subjective nature of anhedonia, where the individuals' inability to experience pleasure notably impacts their quality of life, leading to social withdrawal and diminished functioning.

Anhedonia is becoming an increasingly significant focus for study and clinical intervention because of its widespread influence across multiple life domains. Understanding the underlying causes of anhedonia may lead to more effective treatments, particularly as current research has started investigating its neurological basis and function in reward circuits.[8] As such, this paper aims to examine the underpinnings of anhedonia, its relationship with stress, how it manifests in depression and other related disorders, and current therapeutic techniques through an in-depth literature review of current research and developments in the field.

Methods

We performed a narrative review of the literature to consolidate evidence regarding the manifestation of anhedonia in various psychiatric disorders, including depression, schizophrenia, anxiety disorders, and PTSD, as well as the neurobiological underpinnings of anhedonia and its treatments. We performed systematic keyword searches across three primary databases: PubMed, PsycINFO, and Google Scholar. The databases were selected due to their comprehensive coverage of psychological, biomedical, and interdisciplinary research.

We searched for different combinations of the word "anhedonia" with related disorders and ideas to demonstrate that the review is transdiagnostic. The search terms encompassed examples such as: anhedonia, anhedonia AND depression, anhedonia AND schizophrenia, anhedonia AND anxiety, anhedonia AND PTSD, anhedonia AND treatments. These were chosen according to the topics discussed in the review and exemplify methodologies employed in prior literature searches involving anhedonia. Moreover, secondary searches integrated "anhedonia" with reward processing, neurobiology, or computational models to locate mechanistic studies.

We primarily searched for papers from 2000 to 2023 (the most recent studies at the time of writing), but we also included some important historical works (like Ribot's original definition and Snaith's early conceptual paper) to provide context. The search was not strictly limited by date, but we focused on research from the last 15 to 20 years to obtain the most up-to-date findings and developments.

The criteria for inclusion were as follows: Peer-reviewed journal articles written in English (including empirical studies, meta-analyses, systematic reviews, and conceptual/theoretical papers) that specifically focused on anhedonia or its underlying mechanisms. Empirical studies encompassed randomized controlled trials, observational and correlational studies, neuroimaging or neurophysiological investigations, and computational or modeling papers that measured or studied anhedonia. Review articles and book chapters that included evidence related to anhedonia were also incorporated. Studies needed to focus on human subjects (clinical or healthy populations) where anhedonia was evaluated or examined. Animal studies and other preclinical research were predominantly omitted unless they offered significant mechanistic insights. We did not include non-peer-reviewed works (like commentaries, editorials, dissertations, meeting abstracts) or works that were not written in English. We also excluded articles that did not focus on anhedonia, like general depression or anxiety papers that didn't explicitly measure or talk about pleasure or motivation deficits. These criteria (English language, human focus, peer-reviewed sources, and direct relevance to anhedonia) conform to established practices in narrative review methodology.

After our initial searches of the databases, we looked at the reference lists of all the relevant arti-

cles to find any other studies ("snowballing"). This iterative method helped us find important papers and new work that we might not have encountered with keyword searches alone. The final compilation of sources included in the corpus shows this wide, transdiagnostic search strategy, which is in line with best practices for narrative literature reviews.

Literature Review

Anhedonia in Different Psychiatric Disorders

Anhedonia is a phenomenon that manifests across various psychiatric disorders, each with a distinct symptomatology. This section analyzes the presentation of anhedonia in depression, schizophrenia, anxiety, and PTSD.

Anhedonia and Depression

Anhedonia, a core symptom of MDD, impairs the ability to feel pleasure and the motivation to participate in such activities. People affected by depression exhibit diminished activity in reward-processing brain regions, which leads to reduced interest in activities pleasurable previously.[4,8] Rizvi *et al.*[9] classified anhedonia in depression as either consummatory anhedonia, the inability to enjoy rewards in the moment, or anticipatory anhedonia, the inability to look forward to or expect pleasure from future events. Depressive anhedonia is also closely associated with stress-related mechanisms. A study shows that chronic stress and its neuroinflammatory responses can worsen anhedonia by further impairing dopamine transmission in the mesolimbic pathway.[10] The combined effects of dopamine dysregulation and stress-induced neuroinflammation highlight the complex neurobiological pathways that cause anhedonia in depression.

Anhedonia and Schizophrenia

Anhedonia is a key negative symptom of schizophrenia that can significantly affect life quality and social functioning. Individuals affected by schizophrenia often report difficulty deriving pleasure from social interactions and other previously enjoyable activities. A study suggests that unlike in depression, patients with schizophrenia may have the ability to feel pleasure during enjoyable experiences but struggle with the motivation to seek out these experiences.[11] This distinction is important for understanding the presentation of anhedonia in schizophrenia, which may be more closely linked to deficits in motivation rather than the feeling of pleasure itself.

According to Fortunati *et al.*,[7] subjective experiences in schizophrenia differ greatly, with some patients reporting extreme emotional blunting and others reporting fleeting moments of pleasure that are hard to sustain. Fortunati *et al.*[7] also classified social anhedonia, or the inability to feel pleasure in social settings, as a distinct type of anhedonia in schizophrenia. Additionally, another study found that anhedonia in schizophrenia is further worsened by impairments in social cognition and functioning because individuals with this disorder may have difficulty recognizing or interpreting rewarding social cues, leading to diminished interaction with other people.[12] Thus, understanding social anhedonia is important to develop interventions addressing both neurobiological and social deficits.

Anhedonia and Anxiety

While anhedonia is most commonly observed in depression and schizophrenia, it also manifests in anxiety disorders and PTSD. Taylor *et al.*[13] found that anhedonia often overlaps with feelings of worry and hypervigilance in anxiety disorders, reducing the ability to experience positive emotions. This can be further worsened by the state of persistent anxiety, leading to diminished pleasure in everyday activities and social withdrawal. Winer *et al.*[14] posited that the reduced capacity for pleasure is detrimental because it reduces the ability to engage in positive, mood-enhancing experiences that can buffer against anxiety symptoms, thereby potentially contributing to the worsening of symptoms. Additionally, Taylor *et al.*[13] found that anhedonia could also play a role in anxiety onset and maintenance, perpetuating avoidance behaviors, increasing threat reactivity, reducing tolerance for distress, and impairing learning. This suggests that anhedonia not only worsens anxiety but also limits opportunities for recovery and resilience.

Anhedonia may worsen anxiety in depression by disrupting sensory processing and environmental

perception.[15] Grillo[15] posits that intense pleasures can help restore these experiences, reducing both anxiety and depression temporarily. This suggests that targeting sensory and experiential deficits could be a key therapeutic strategy for alleviating co-occurring anxiety and depression. Thus, better understanding and targeting anhedonia in anxiety disorders could help alleviate persistent symptoms and improve overall treatment outcomes.

Anhedonia and PTSD

Anhedonia is a common symptom in PTSD because trauma-exposed individuals experience heightened sensitivity to stress. One study found that the neural circuit involved in reward processing is often disrupted in PTSD, with decreased activity in the brain's reward centers contributing to emotional numbing and avoidance-like symptoms.[16] Additionally, Kirshenbaum *et al*.[17] found that neural sensitivity to stress can predict the development of anhedonia in trauma survivors, with long-term disruptions in reward processing persisting for years after the traumatic event. This suggests that understanding and addressing reward processing disruptions in PTSD could be crucial for preventing and treating anhedonia in trauma survivors.

Anhedonia in PTSD is believed to result from inhibited emotional expression, heightened sensitivity to negative stimuli, and disruptions in the brain's reward system, with some suggesting it could also be a pre-existing risk factor due to early-life adversity.[18] Frewen *et al*.[19] explored anhedonia in women with PTSD, distinguishing between hedonic deficits (reduced positive affect) and negative affective interference (negative responses to positive stimuli). The study linked anhedonia to PTSD severity, depressive symptoms, emotional responses- particularly in social contexts- and observed differing neural responses between social and non-social stimuli, with trauma exposure and early-life abuse identified as key factors.[19]

Neurobiological and Computational Models

Neurobiological Models

The neurobiological mechanisms of anhedonia have been extensively studied, with most research focusing on the reward circuitry of the brain. The main component of this system is the mesolimbic dopamine pathway since it is important for reward and pleasure processing.[8] Research has found dopamine dysregulation in this circuit to be involved in the development of anhedonia, especially in cases of MDD.[8] A study indicated that a decrease in dopamine transmission in certain brain regions, the ventral tegmental area (VTA) and nucleus accumbens, can lead to reduced motivation and pleasure-seeking behavior, which are distinctive features of anhedonia.[20]

In their 2018 study, Cooper *et al*.[20] also reported that reward processing deficits are the primary biological mechanisms of anhedonia in depression, mainly because of their association with dopamine transmission and reinforcement learning. They hypothesized that anhedonia is linked to reduced dopamine transmission, which affects the experience of pleasure and the effectiveness of reinforcement learning. Typically, anhedonic symptoms in depression are often thought to be due to failures in reinforcement systems. However, this study's findings suggest that while depressed individuals may have a diminished ability to learn by utilizing rewards, the primary deficit may lie in reward sensitivity rather than learning, meaning that they may be less motivated by or responsive to rewards.[20]

Other than dopamine, neurotransmitters such as serotonin and glutamate are also involved in the manifestation of anhedonia. Research has linked serotonin to mood regulation and emotional processing, showing that disruptions in serotonergic signaling may lead to anhedonia in depression.[21] Another study suggests that glutamate dysregulation may alter synaptic plasticity and increase anhedonic symptoms, especially in brain regions related to reward processing and emotion.[22]

Stress-related mechanisms also play an important role in the onset and continued experience of anhedonia. A study showed that chronic stress impairs neuroplasticity and diminishes the ability of the brain to adjust to positive stimuli, contributing to the persistence of anhedonia.[23] Recent research also shows that stress-induced inflammation further disrupts the brain's reward

circuitry, especially in individuals with high neural sensitivity to stress.[10] This neuroinflammatory response worsens anhedonia by increasing dopamine dysregulation and impeding the operation of the reward system.

Neuroimaging studies have also provided insight into the structural and functional abnormalities in the brains of those with anhedonia. A study by Treadway and Zald[4] consistently observed reduced activity in the prefrontal cortex and ventral striatum, suggesting that anhedonia may arise from impaired top-down regulation of reward processing. These irregularities in brain function may indicate broader changes in neural circuitry, leading to a decreased ability to feel pleasure and motivation to partake in rewarding activities.

Understanding the neurobiological basis of anhedonia provides various opportunities for developing targeted interventions. In one study, pharmacological treatments targeting the mesolimbic pathway to enhance dopamine function showed potential in treating anhedonic symptoms in depression and schizophrenia.[22] Another study suggested anti-inflammatory treatments, a novel approach, to treat the stress-related components of anhedonia by reducing neuroinflammation and restoring reward processing.[16] As research in this area progresses, a better understanding of the neurobiology of anhedonia may lead to more promising interventions for individuals with various psychiatric disorders suffering from this symptom.

Computational Models

Computational models are becoming increasingly important in understanding the complex causes of anhedonia, providing insights that go beyond the traditional approaches. These models simulate brain processes involved in reward processing, decision-making, and motivation to help identify key disruptions linked with anhedonia. They offer a framework to link observable symptoms of anhedonia with underlying neural mechanisms by incorporating findings from neuroimaging studies and behavioral data.[20]

The reinforcement learning (RL) framework is the most commonly used computational model for studying anhedonia. It examines how individuals learn to associate actions with rewards and alter behavior based on feedback. In one study, Treadway and Zald[4] found that this process is disrupted in anhedonia, which results in decreased reward responsiveness and diminished motivation to seek pleasurable experiences. According to another study using the RL model, individuals with anhedonia have deficits in both reward sensitivity and the ability to learn from positive reinforcement, suggesting that the brain's reward prediction error signals, which measure the difference between expected and actual rewards, may be impaired.[20] This means that individuals with anhedonia fail to assess rewards and thus may not make optimal choices. These deficits can be simulated within RL models, allowing researchers to test different hypotheses about the underlying causes of anhedonia. Through this, future research can address the motivational deficits associated with anhedonia and lead to the development of targeted interventions that enhance reward sensitivity and improve the ability to learn from positive reinforcement.

Value-based decision-making models have also been used to study how anhedonia affects decision-making for delayed and immediate rewards. Pizzagalli[21] and Olson *et al.*[24] reported that individuals with anhedonia tend to favor smaller immediate incentives over larger delayed rewards, displaying a disruption in the brain's ability to anticipate future pleasure. Computational models can quantify these changes in the judgment of reward value and identify the neural circuits involved in the decision-making impairments seen in anhedonia by simulating decision-making processes. Even though computational models have significantly advanced our knowledge of anhedonia, there is a need for more individualized models, which serves as a big challenge. Current models often fail to record the heterogeneity of anhedonia across psychiatric disorders. To offer a more comprehensive view of the manifestation of anhedonia, future models should integrate personalized data, such as genetic, neuroimaging, and behavioral data.[17] Further, incorporating longitudinal data may improve the prediction of treatment outcomes and disease progression.[16] Moreover, computational models should study the role of inflammation and immune dysregulation in anhedonia. A recent study suggests that inflamma-

tion disrupts reward-related circuits, and incorporating inflammatory biomarkers into models could lead to new treatment avenues.[10]

A promising approach by Klein *et al*.[22] combined computational models with neurostimulation techniques. These models could lead to the development of targeted interventions, like deep brain stimulation (DBS) or transcranial magnetic stimulation (TMS), and optimize treatment parameters by identifying disrupted neural circuits. Additionally, Rizvi *et al*.[9] reported that machine learning also shows the potential to analyze large datasets to identify patterns and improve diagnostic accuracy and treatment plans. As computational models evolve, they can provide a better understanding of anhedonia, paving the way for individualized treatments and improved outcomes.

Treatment Approaches for Anhedonia

Given the complex neurobiological and psychological causes behind anhedonia, effective treatment approaches often require interventions that target both biological and cognitive factors. Traditional approaches to treating anhedonia focus on pharmacological interventions, while recent research explores psychotherapy and lifestyle-based interventions to treat this symptom.

Pharmacological interventions for anhedonia primarily focus on modulating the brain's dopamine and serotonin systems, which are critical for reward processing and motivation. Patients with anxiety and depression are frequently prescribed serotonin-norepinephrine reuptake inhibitors (SNRIs) and selective serotonin reuptake inhibitors (SSRIs).[22] However, Pizzagalli[21] found that while SSRIs and SNRIs are effective in alleviating general depressive symptoms, their efficacy in specifically treating anhedonia has been limited. This may be due to their indirect effect on the dopamine system, as they primarily target serotonin and norepinephrine pathways rather than directly enhancing dopamine transmission.

New pharmacological treatments target the dopamine system more directly. Treadway and Zald[4] reported that drugs such as Bupropion, which increases dopamine activity, have shown promise in treating anhedonic symptoms by improving motivation and reward sensitivity. Novel treatments such as ketamine, an NMDA receptor antagonist, have also shown immediate and lasting effects on anhedonia, potentially due to its effect on glutamatergic signaling and neural plasticity.[22] However, further study is required to determine the long-term safety and effectiveness of these treatments for anhedonia.

Psychological treatments for anhedonia aim to improve emotional and cognitive regulation while re-engaging patients with pleasurable activities. One such approach is Positive Affect Treatment (PAT), which encourages positive emotional experiences and focuses on reforming cognitive patterns that reduce motivation to directly boost positive affect and reduce anhedonic symptoms.[25] PAT combines behavioral activation with cognitive-behavioral therapy (CBT) techniques to encourage patients who are feeling unmotivated to resume enjoyable activities or indifferent initially.[9]

Behavioral activation alone has also been proven effective in treating anhedonia, especially when combined with mindfulness and relaxation techniques. These techniques help patients identify and pursue activities that match their previous interests, reinforcing reward-seeking behavior.[26] Structured psychotherapy, such as CBT and dialectical behavior therapy (DBT), have also been used to treat anhedonia by helping patients recognize harmful thought patterns and substituting them with more adaptive cognitive strategies.[27]

Additionally, mindfulness-based therapies have become increasingly popular as adjunctive treatments for anhedonia. Examples of these are mindfulness-based cognitive therapy (MBCT) and mindfulness-based stress reduction (MBSR). These therapies encourage present-moment awareness and acceptance, helping individuals disengage from ruminative thinking and re-engage with pleasurable experiences.[25]

Neurostimulation methods such as deep brain stimulation (DBS) and transcranial magnetic stimulation (TMS) have become increasingly popular in recent years as prospective treatments for anhedonia. TMS increases neural activity and enhances mood regulation by non-invasively stimulating particular brain areas, such as the dorsolateral prefrontal

cortex. In a 2022 study, Klein *et al.*[22] reported that TMS can improve reward responsiveness and reduce anhedonia in treatment-resistant depression. On the other hand, DBS is more invasive and targets deeper brain structures involved in reward processing, such as the nucleus accumbens. Preliminary studies show that DBS may treat anhedonia in individuals with severe treatment-resistant psychiatric disorders, but more research is needed to study its long-term efficacy and safety.[22]

Lifestyle interventions like exercise and diet modifications have also shown potential in reducing anhedonic symptoms. Regular physical activity has been shown to improve mood and increase dopamine signaling in the brain's reward pathways, to decrease anhedonia.[9] Dietary interventions targeting inflammation and neuroplasticity, like increasing omega-3 fatty acid intake, have also been suggested as complementary treatments for anhedonia, but more clinical trials are needed to confirm their efficacy.[10]

Discussion

Our findings emphasize the complex nature and role of anhedonia across various psychiatric disorders. In addition to being a hallmark of depression, anhedonia is also among the symptoms of schizophrenia, anxiety disorders, and PTSD. The presentation of anhedonia in these disorders exhibits its transdiagnostic nature, implying that shared underlying neurobiological mechanisms could be targeted for treatment. The findings from a multitude of studies have shed light onto the brain regions and circuits, such as the prefrontal cortex, striatum, and reward-related circuits, involved in anhedonia.[4,20,23] These neurological disruptions appear consistent across disorders, while the specific manifestations of anhedonia may differ depending on the diagnosis.

Anhedonia is still difficult to treat, especially because it is resistant to conventional antidepressants. Current pharmaceutical and psychotherapy treatments frequently fail to address the motivational and reward-related deficits associated with anhedonia.[27] Novel therapies, such as positive affect treatment, which focuses on improving positive emotions and increasing participation in rewarding activities, show promise but require more research.[25] Neurostimulation approaches, including TMS and DBS, have emerged as possible treatments because they directly target the brain circuits involved in reward processing.[22] These techniques may provide hope for patients suffering from treatment-resistant forms of anhedonia, but further research is needed to improve their efficacy and assess long-term effects.

The use of computational models has provided a more structured framework for understanding the changes in reward processing caused by anhedonia. These models have helped us understand how anhedonia shows itself in various psychiatric populations by mimicking the impacts of decreased reward sensitivity, motivation, and decision-making.[20] Computational models also enable the discovery of possible biomarkers and neurological targets for treatment, facilitating the development of tailored therapies. However, current models are restricted in their ability to represent the diversity of anhedonia, particularly among mental illnesses. To gain a better understanding of anhedonia, future research should focus on constructing individualized computer models that include genetic, neuroimaging, and behavioral data.[17]

Conclusion

While considerable progress has been made in understanding the neurobiological and psychological mechanisms of anhedonia, much is left to be discovered, particularly in terms of incorporating individualized treatments into clinical practice. Future research should focus on bridging the gap between neurobiological insights and clinical applications, eventually leading to the development of more effective interventions for those suffering from this debilitating symptom.

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