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Anhedonia in Bipolar Depression and Major Depression

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Abstract:

Anhedonia is a hallmark feature of depression and is highly prevalent among individuals with mood disorders. The history and neurobiology of anhedonia has been most extensively studied in the context of unipolar Major Depressive Disorder (MDD), with converging lines of evidence indicating that marked anhedonia heralds a more chronic and treatment-refractory illness course. Furthermore, findings from neuroimaging studies suggest that anhedonia in MDD is associated with aberrant reward-related activation in key brain reward regions, particularly blunted reward anticipation-related activation in the ventral striatum. However, the ongoing clinical challenge of treating anhedonia in the context of Bipolar Disorder (BD) also highlights important gaps in our understanding of anhedonia's prevalence, severity, and pathophysiology along the entire mood disorder spectrum. In addition, although current theoretical models posit a key role for reward hyposensitivity in BD depression, unlike studies in MDD, studies in BD do not clearly show evidence for reduced reward-related activation in striatal or other brain regions. Although further research is needed, the evidence to date hints at a divergent pathophysiology for anhedonia in unipolar and bipolar mood disorders, which, if better understood, could lead to significant improvements in the diagnosis and treatment of MDD and BD.

Keywords: Anhedonia, Bipolar Depression, Major Depression.

Introduction:

Anhedonia, the inability to experience pleasure, constitutes a core symptom of both bipolar depression (BD) and major depressive disorder (MDD). Recent research suggests intriguing differences in its presentation and potential underlying mechanisms between these two conditions [1].

Severity: Studies suggest BD, particularly bipolar (BD), might exhibit more severe anhedonia compared to MDD. This could be linked to the broader symptom profile of BD, involving additional impairments like anxiety and poorer functioning [2].

Reward Processing: Both BD and MDD show abnormalities in reward processing brain regions like the ventral striatum. However, research points towards qualitative differences. BD might involve altered reward anticipation and motivation, while MDD could be linked to deficits in experiencing pleasure itself [3, 4].

Neurotransmitters: Dopamine and glutamate dysregulation are implicated in both disorders. However, BD might involve additional neurotransmitter imbalances, like serotonin and norepinephrine, potentially contributing to its distinct anhedonic profile [2].

Subtypes: Recent studies explore anhedonia within subtypes of BD and MDD. For example, atypical BD, with prominent negative symptoms, might show similar anhedonia patterns to MDD [5].

Course and Fluctuations: Anhedonia in BD may display greater fluctuations due to manic episodes, leading to temporary relief followed by deeper lows during depression MDD, on the other hand, presents a more constant anhedonic state. [1].

Treatment Implications: Understanding these differences holds therapeutic potential. Tailoring treatments to address the specific anhedonic profile in each disorder could improve outcomes. For example, BD might benefit from interventions targeting reward anticipation, while MDD might respond better to strategies directly enhancing pleasure experience [4].

Future Directions: Ongoing researches explore biomarkers to differentiate anhedonia across disorders. Additionally, investigating environmental factors and their interplay with genetic predispositions could provide valuable insights for prevention and personalized treatment approaches [4].

Psychology

Different concepts of anhedonia have been described, for example, Meehl's low hedonic capacity as a predisposing factor for both schizophrenia and depression, and Klein's impairment of appetitive and consummatory pleasure as a hallmark of endogenomorphic depression. More recent and popular concepts pertain to the pleasure cycle and positive affectivity. Both concepts partially overlap and offer a link between psychological categories (e.g., pleasure or anhedonia) and their neurobiological substrates. [6]

However, they differ in their origin and perspective. The pleasure cycle concept is grounded in the affective neurosciences and narrowly focused on reward. Part of its success has been the scientific strategy of dividing the concept of pleasure into the affective reaction (objective behavioral, physiological, and neural reactions) and its subjective affective experience. [7]

The positive affectivity concept is grounded in a more general theory of affective psychology. Given their popular use (in which clinical anhedonia is often equated with deficits in reward processing or positive affectivity), a careful description of these concepts seems justified.[8]

1. **The pleasure cycle.** The study of reward processing has found substantial evidence for distinct phases within a pleasure cycle, each phase having its own brain circuitry: an appetitive phase (dominated by wanting), a consummatory phase (dominated by liking), and a satiety phase (dominated by learning). Wanting is defined as the motivation for or the incentive salience of a reward, liking as the actual pleasure or hedonic impact of a reward, learning as associations, representations, and predictions about future rewards based on past experience. Wanting, liking, and learning constantly interact, wax and wane during the different phases of the pleasure cycle. [9]

They all have both conscious and unconscious components. Anhedonia is then defined as "the impaired ability to pursue, experience, and/or learn about pleasure, which is often, but not always accessible to conscious awareness". This model describe the reward process as initially building a stimulus reward association, which then leads to interest/desire (wanting a reward), anticipation (state of readiness for a reward), motivation (initial energy expenditure to attain a reward), effort (sustained energy expenditure to attain reward), hedonic response (e.g., enjoyment of reward), and feedback integration (updating reward presence and values). In this model rewards can be primary/fundamental (e.g., food, sex, social interaction) or secondary/higher order (e.g., monetary, artistic, altruistic, and transcendental); their involved brain mechanisms are supposed to generally overlap and to be distinct from the mediation of other features of the same event (e.g., sensory, cognitive). [10]

The actual experience of pleasure is different from a mere sensation and/or a thought, it is an additional "hedonic gloss" generated by the pleasure networks of the human brain. Although historically the liking part of anhedonia in depression has been emphasized, recent findings point at a greater importance of both wanting and learning: that is, a reduction in reward anticipation, the willingness to exert effort in order to get a reward, and the ability to modify behaviour as a function of reward. [11]

2. ***Positive affectivity***. In addition to anhedonia as a specific dysfunction of the pleasure cycle, anhedonia may also be described within more general, dimensional models of affect. Based on studies of the intercorrelations among emotional experiences (subjective reports of words, faces, and experiences) these models organize our affective space in 2 (or more) higher order dimensions (the circumplex models of affect): for example, valence and arousal, positive and negative affect (PA and NA). Affective states are then construed as linear combinations of these 2 dimensions. [12]

In these models a distinction can be made between "core affect" (object-free, flee-floating neuro-physiological state – consciously accessible as a simple, non-reflective feeling) and emotional episodes. These latter are changes in core affect elicited by and attributed to an object. Emotional episodes are accompanied by instrumental action, physiological, and expressive changes (as part of the changes in core affect and as part of the instrumental action), perceptual-cognitive appraisal of the object (for example, expectations for the future, relevance for one's goals, causal antecedents), and other conscious, subjective experiences. The subjective experience of a specific emotion (or emotional meta-experience; e.g., a person perceiving himself as being happy) finally results from a cognitive interpretation/categorisation of the neurophysiological changes that are elicited by the object: these changes being organised in relation to their object, behavioural responses, past experiences, and semantic knowledge. [13]

The modelling of the affective space into PA and NA seems of particular interest for anhedonia in depression. PA and NA pertain to the general and evolutionary adaptive systems of approach and withdrawal. PA represents the extent to which a person avows a zest for life, and NA the extent to which a person reports feeling upset or unpleasantly aroused. PA and NA are not therefore oppo-sites, but uncorrelated and independent dimensions. [14]

In particular, PA feelings (feelings of energy, vigour, enthusiasm, confidence) serve as a motivating source and affective reward of goal-directed behaviour. Anhedonia may then be defined as low PA. Defined as such, anhedonia appears to be a relatively specific feature of depression: more specific than high NA.[15]

Psychopathology

Anhedonia may be deconstructed according to different principles.

Inherent in its current use is the difference between consummatory (lack of actual pleasure, experiential enjoying, liking) and appetitive (lack of interest, looking forward and wanting) anhedonia. Thereby, the form (in a Jaspersian tradition) of anhedonia is complex: phenomena pertaining to both feeling and affective states, and urge, drive, and will.[11]

A difference has to be made between anhedonic symptoms (patients' subjective complaints; for example, loss of interest and enjoyment) and signs (behaviour deemed pathological by a clinician; e.g., lack of positive emotions or mood observed/elicited during a psychiatric interview). Theoretically, anhedonic symptoms should be differentiated from an impaired ability to express (in words or behaviour) the feeling of pleasure, that is, a kind of "anhedonic alexithymia."[16]

Anhedonia may be pervasive or selective. Like pleasure, anhedonia may then be divided according to its object. Pleasures like food, sex, and social interaction are said to be fundamental (or primary, instinctual) – the reward is inherent. They contrast with higher order (or secondary, non-instinctual) pleasures (like music, art, money, intellectual and altruistic activities) – the reward is not inherent and has to be learned. These rewards may belong to the past (remembered), present (actually experienced), or future (anticipated).[17]

Although anhedonia suggests a categorical all-or-nothing phenomenon, anhedonia (or hedonic functioning) has to be seen within a dimensional perspective.[18]

Finally, anhedonia may exist on its own or may be part of a more general flattening of emotions and affect (e.g., general paralysis of feeling, blunted affect, derealisation).[19]

From a psychopathological point of view some problems/shortcomings of the concept" anhedonia as the impaired ability to pursue (wanting), experience (liking), and/or learn about pleasure" should be mentioned. First, “anhedonia” clearly cuts across the classic tripartite model of the mind. Anhedonic symptoms pertain to volition (e.g., apathy or diminished motivation; avolition or diminished ability to initiate and maintain goal directed behaviour; anergia or diminished perceived energy), fatigue (weariness or diminished ability following mental or physical activity), affect (lack of pleasure and enjoyment) and cognition, without clear boundaries. [9]

Second, the pleasure cycle (like a hedonic treadmill driven by ever changing concrete primary or secondary rewards) seems more suitable for understanding and describing (the lack of) positive emotions, and less suitable for describing (the lack of) positive mood or affect. For this latter purpose, the concept of positive affect seems more helpful with anhedonia being defined as low positive affect (including emotion, mood, and affective trait). [11]

Hedonic symptoms within the pleasure cycle are part of an emotional episode: a sudden change in affect, elicited by an object and characterized by diminished interest, energy, and pleasure. When present (or absent), these symptoms are at the foreground of our awareness but difficult to assess due to their short duration and limited pervasiveness in consciousness. [15]

Anhedonic mood is longer lasting, more object-free, characterized by an overall diminished zest for life and feeling of energy, having more influence on other physiological and psychological processes. Although more at the background of our awareness, it is more easily assessed due to its longer duration and pervasiveness in consciousness. [20]

When interviewing a depressed patient, anhedonic signs will be looked for. For anhedonic symptoms the physician will have to rely on the verbal account of the patient; this will be less suitable for positive emotions (for which limited memory by nature), and more applicable for mood and trait.[21]

The reviewing of anhedonia underscores the complex and hybrid nature of mental symptoms. Anhedonic symptoms are a primary unit of analysis in the diagnosis and treatment of depression, but are clearly not natural kinds. They result from a biological signal, a primordial (pre-conceptual, prelinguistic) awareness of this signal and a formatting of this awareness through different configurators (cultural, social, familial, personal); finally, symptoms are expressed and constructed within a clinical and dialogical encounter. Concepts like the pleasure cycle and the affective circumplex may shed different lights on this black box of anhedonic symptom formation.[22]

Phenomenologically, severe and pervasive anhedonia in depression (as described in the aforementioned models) is more than a symptom or constellation of symptoms. It rather could be the primal manifestation or essential characteristic, an existential change in the sense of reality, of which anhedonic symptoms are representative. [23]

Assessment of anhedonia as such could then lead to a better understanding of the depressed patient and (in the end) better outcome. This better understanding in general could be as (or even more) important than the different phases of the pleasure cycle as specific targets for biological or psychotherapeutic interventions.[24]

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