Chapter 1
The Different Facets of Anhedonia and Their Associations with Different Psychopathologies

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Abstract Over the last several decades, there has been increasing interest in the role that anhedonia plays in various psychopathologies, ranging from mood disorders, to eating disorders, to psychotic disorders. The term 'anhedonia' (which simply means, *without pleasure*) has been used to describe a wide range of constructs, affective experiences, and events. Given the breadth of the term, it is likely that different aspects of anhedonia may be related to different psychopathologies in various ways. This review discusses how the literature has parsed anhedonia and how the various components and facets of anhedonia may relate to various psychopathological constructs. In addition, this review takes concepts and theories from the broad affective science literature and identifies additional components of anhedonia that may be critical to the field's understanding of the construct. Given the importance that anhedonia plays in a multitude of psychopathological constructs, a careful analysis of the various components and facets of anhedonia may provide a conceptual framework for research in this area.

Keywords Anhedonia • Psychopathology • Affective experience • Anticipatory and consummatory positive affect

Abbreviations

AN	Anorexia nervosa
APA	Anticipatory positive affect
BED	Binge eating disorder
BN	Bulimia nervosa

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CPA	Consummatory positive affect
DSM-5	*Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition*
EEG	Electroencephalogram
HIV	Human immunodeficiency virus
HSDD	Hypoactive sexual desire disorder
MDD	Major depressive disorder
PTSD	Post-traumatic stress disorder
RDoC	Research Domain Criteria
SUD	Substance use disorder

1.1 Introduction

Anhedonia, which can broadly be defined as a diminished capacity to experience pleasure, is a construct associated with various psychiatric disorders. The importance of anhedonia in psychopathology (and hedonic capacity more generally) is further supported by its inclusion as a key domain in the Research Domain Criteria (RDoC), a new conceptual framework for psychopathology research recently launched by the United States' National Institute of Mental Health that focuses on transdiagnostic domains, rather than disorders.

Despite its importance, the scientific and clinical literature uses the term *anhedonia* in different ways and within different contexts to describe a broad range of emotional experiences. Given the breadth of the term, it is likely that various aspects of anhedonia relate to different psychopathologies differently.

The thesis of this review is therefore that the construct of anhedonia is more nuanced than what is often described in the literature, and divisible facets of it are often obscured. First, we will review different ways that anhedonia has been (or could be) parsed. Second, we will discuss how these separable facets relate to different psychopathologies. Of note is that in order to focus this review and to not overlap with other chapters in this volume, the present review will discuss hedonic experience and expression, rather than the cognitive aspects of hedonic capacity (e.g., evaluation of reward value, affective forecasting, reward learning, and reward prediction error).

1.2 A Review of the Various Facets of Anhedonia

1.2.1 Role of Anhedonia in the Time Course of Reward Processing

1.2.1.1 Anticipatory vs. Consummatory Positive Affect

Some researchers use *anhedonia* to refer to deficits in the affective response to a rewarding or pleasurable stimulus. However, there are two components to the

positive affect experienced in rewarding situations – anticipatory positive affect (APA) and consummatory positive affect (CPA). The difference between APA and CPA is temporal. APA is the excitement felt as the animal waits for the receipt of a reward, and CPA is the enjoyment felt after receipt of a reward. APA and CPA are also linked to different behavioral and motivational outcomes – with the former linked to motivation and goal-directed behavior and CPA linked to satiation at the attainment of a goal [1]. Berridge and Robinson [2] describe these constructs as 'wanting' and 'liking', respectively.

Although APA precedes CPA for a single rewarding stimulus, there is actually a bidirectional relationship between the two. On the one hand, animals experience APA as they await a reward and CPA after they receive it. On the other hand, animals only experience APA once they have experienced CPA in response to a novel stimulus and learned to associate positive affect with that stimulus [3–5]. That is, most of the time, an animal can only *want* something that it has previously *liked*. Additionally, studies have shown that the more one looks forward to a reward, the more one enjoys it when they get it [6, 7].

Neurobiological studies also support the distinction between anticipatory and consummatory positive affect. Animal models of reward processing have demonstrated that there are separable neural pathways associated with CPA and APA [2, 8]. Functional neuroimaging studies of humans have also supported this distinction. For example, Knutson et al. [9] observed activation in the nucleus accumbens while individuals anticipated reward, but this activation subsided during the delivery of rewards. On the other hand, the ventromedial frontal cortex appeared to be more involved during the experience of consummatory affect (see [10] for review).

Given the distinction between APA and CPA, it is important to consider that anhedonia may represent a deficit in anticipatory positive affect, consummatory positive affect, or both. That is, individuals with anhedonia may not anticipate that rewards will be pleasurable, not react with joy and/or satisfaction when they receive them, or both.

1.2.1.2 Distal vs. Proximal Reward

While deficits in APA and CPA are critical facets of the broad construct of anhedonia, there are other temporal demarcations that could be made within APA and CPA as well. First, it is possible that anhedonia may manifest as a deficit in anticipatory pleasure for more distal rewards (e.g., a reward occurring in several months), while anticipatory pleasure for proximal rewards (e.g., a reward occurring in several minutes) is relatively intact (see [11] for related work). For example, an individual may not look forward to receiving a university degree or work-related promotion, but still look forward to a movie that is about to start.

Second, after the reward is received (i.e., within the CPA phase), anhedonia may manifest as a more rapid decrease in consummatory pleasure. That is, the hedonic impact of a stimulus, and the amount of consummatory positive affect that it generates, might fade more quickly in certain individuals or in certain psychopathologies.

This deficit in the time course of the hedonic response may or may not also relate to the "peak" affective response experienced by the individual [12].

Thus, anhedonia can reflect the length of time a stimulus generates anticipatory processes, consummatory processes, or both. From this discussion, we see that it is also possible to describe anhedonia as a 'narrowing of the time window' during which APA and CPA are experienced surrounding the receipt of reward.

1.2.2 Loss of Interest vs. Loss of Pleasure

Anhedonia, along with persistent depressed mood, is one of the cardinal symptoms of major depressive disorder according to the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (DSM-5) [13]. However, even within the context of one disorder, there are two definitions of anhedonia. According to the DSM-5, an individual is experiencing anhedonia if he/she reports deriving less pleasure from daily activities than usual *or* if he/she reports feeling less interested in those daily activities. While "loss of interest" and "loss of pleasure" are considered anhedonia, it is not clear whether they are equivalent.

Animal models illustrate a behavioral distinction between loss of interest and loss of pleasure. In rats, 'loss of interest' is often operationalized as reduced exploratory behavior in novel environments such as the classic open field tests [14, 15]. This pattern of behavior is often conceptualized as a loss of incentive motivation [16]. On the other hand, 'loss of pleasure' is operationalized as a reduction in responsiveness to previously rewarding stimuli (e.g., decreased preference for and consumption of sucrose [17]).

In animal models of depression, both of these behaviors are conceptualized as anhedonia and, indeed, both are consequences to exposure to chronic stress [18] and improve with the administration of antidepressant medications [19]. However, a factor analysis of these putative animal indices of depression indicates that exploratory behavior and sucrose consumption load onto separate factors [20]. Given that separable behaviors are observed in animal models of depression, it is unclear whether the construct of "anhedonia" should apply to both loss of interest and loss of pleasure.

In humans, self-reports of loss of interest and loss of pleasure have been shown to be correlated but separable constructs [21]. This distinction is supported by epidemiological, neuroimaging and psychopharmacological data as well. For example, although the symptoms of loss of pleasure and loss of interest tend to cluster together in clinical populations, not every patient with major depression experiences both symptoms, and they correlate differently with other symptoms of depression [22]. Dysfunction in the mesocorticolimbic dopaminergic pathway has been linked to both symptoms, although different structures within the pathway have been implicated in loss of pleasure (nucleus accumbens; see [23] for review) and loss of interest (prefrontal cortex; [24]). Finally, pharmacological studies examining the effects of non-serotonergic antidepressants on treatment-resistant symptoms (among them loss of interest and

loss of pleasure) have found that different classes of noradrenergic and dopaminergic drugs are more effective at treating loss of interest and loss of pleasure [25].

1.2.3 Anhedonia as Flat Affect

Anhedonia has also been used to describe flattened affect across multiple emotions and valences. That is, rather than experiencing a diminished capacity to experience pleasure, some scientists and clinicians use the term anhedonia to mean reduced affect across numerous emotional dimensions (e.g., reduced happiness, reduced sadness, reduced anger, etc.) This phenomenon, also known as 'restricted range of affect' or 'emotional numbing', is associated with psychopathologies such as post-traumatic stress disorder (PTSD).

Supporting this definition, factor analytic research in traumatized populations has shown that loss of interest/pleasure and emotional numbing load onto the same latent factor [26–28]. As discussed below, it is possible that this association between symptoms might contribute to the high rates of comorbidity between post-traumatic stress disorder and major depressive disorder [29] – if the two symptoms are in fact part of the same construct rather than two separate ones.

1.2.4 Role of the Stimulus in Anhedonia

A final consideration in our discussion of anhedonia concerns the quality (or category) of the stimulus that elicits the anhedonia. Anhedonia can refer to decreased ability to experience pleasure to all (or at least multiple) positive stimuli, or it can refer to a specific stimulus. Thus, individuals can experience less pleasure in response to a particular stimulus, while responses to other stimuli remain intact. In the present review, we consider three types of stimuli – social stimuli, sensory stimuli, and drug/substances.

1.2.4.1 Social Anhedonia

Most individuals derive pleasure from their social interactions with others, such as conversing, sharing experiences, doing activities together, expressing their feelings, loving, and even competing with other people. Social anhedonia, therefore, involves deriving significantly less or even no pleasure from these social situations.

1.2.4.2 Sensory Anhedonia

Individuals also derive pleasure from various sensory or physical stimuli (i.e., stimuli affecting any of the five senses). Those who experience this type of anhedonia

(often labeled "physical anhedonia") [30] derive less pleasure from physical sensations than those who do not experience physical anhedonia. Although physical anhedonia refers to decreased pleasure for all sensory stimuli, such as smells, sounds, touch, movement, and temperature, two of the physical stimuli most commonly studied in the context of are sexual and gustatory stimuli. Sexual anhedonia, though often associated with major depression, has also been examined in the context of sexual dysfunction. As mentioned earlier, gustatory anhedonia is often used in animal models of anhedonia.

1.2.4.3 Anhedonia and Drug Use

Anhedonia can be associated with several aspects of drug use. Several hedonic theories of addiction posit that drugs act on the mesocorticolimbic dopamine systems, which mediate the intense feelings of pleasure when addictive drugs are administered and the anhedonia during withdrawal [31, 32]. Additionally, it is possible that physiological and psychological tolerance to addictive drugs are related to anhedonia – that is, when taken regularly, the same amount of a particular addictive drug induces less pleasure than it did when the individual first began taking the drug [33].

1.2.5 Summary

In the previous section, we outlined several instances in which the term anhedonia is used to describe a constellation of clinical symptoms including: deficits in the temporal experience of reward, restricted range of affect, loss of interest, loss of pleasure, and anhedonia across a wide range of pleasurable stimuli. This array of deficits may play different roles in various psychopathologies. In the next section, we review the role that these different facets of anhedonia may play in various psychopathological constructs. Of note is that many of the disorders reviewed in this chapter are discussed in more detail in other chapters in this volume. The present chapter therefore focuses on how the different components of and definitions of anhedonia relate to each disorder rather than being an exhaustive review of the role of anhedonia in those disorders.

1.3 Role of Anhedonia in Various Psychopathologies

1.3.1 Anhedonia and Major Depression

Although anhedonia is a feature of many clinical syndromes, major depression is perhaps its archetypal disorder. As mentioned earlier, loss of interest or pleasure is one of

the two symptoms required by the *DSM-5* for a diagnosis of MDD, and approximately 70 % of individuals with the disorder experience anhedonia [13, 34].

1.3.1.1 MDD and Deficits in Reward Processing

A large literature indicates that disturbed reward processing is a prominent, perhaps central, feature of depression [35]. Theoretical models dating back to Donald Klein's work have posited that deficits in *anticipatory* reward processing are most relevant to depression [1, 36] and behavioral, neurobiological, and physiological evidence generally supports this thesis. For instance, Sherdell, Waugh, and Gotlib [37] found that reduced anticipatory, but not consummatory, positive affect predicted lower motivation to expend effort to obtain reward among individuals with MDD. Many studies have implicated the mesolimbic dopamine system in depression (see [38] for a review), which is primarily involved in prediction of and motivation for future rewards. Several studies have also found that frontal electroencephalogram (EEG) asymmetry, a psychophysiological index of reward processing, is altered among depressed individuals during anticipation of monetary reward [39, 40], but not during reward consumption [41].

However, some depressed individuals – for instance, those with melancholic depression – may experience altered consummatory as well as anticipatory reward processing. Klein wrote that these individuals "enjoy nothing and their mood cannot be improved by positive rewards" [36, p. 4], and a DSM-5 criterion for melancholic depression is lack of mood reactivity to positive stimuli [13]. Consistent with this conceptualization, melancholic depressed individuals show an abnormal posterior EEG asymmetry during consummatory reward processing, whereas their non-melancholic counterparts do not [40]. Thus, deficits in anticipatory reward processing seems to be a feature common to most depressed individuals, and a smaller subset of these individuals also show deficits in consummatory reward processing.

As discussed above, another aspect of reward processing is the distinction between temporally proximal and distal rewards. Healthy individuals typically ascribe more value to immediately available rewards than to future rewards of equal or even greater value, a phenomenon labeled delay discounting. Lempert and Pizzagalli [42] reported that this temporal gradient was reduced among anhedonic individuals – that is, these individuals were more apt to choose future rewards of greater value than current rewards of lesser value. The authors speculate that this reflected a reduced responsiveness to immediate reward associated with anhedonia. Although preferring a future of greater value over a lesser current reward is technically more 'rational', it is notable that this pattern of reduced delay discounting is predictive of suicide attempt lethality among depressed individuals [43].

1.3.1.2 Affective Flattening and Major Depression

The diminished interest and pleasure observed in individuals with depression may merely be part of a broad pattern of affective blunting of which diminished interest/pleasure

are merely components. That is, depression may be associated with reduced affective responding for a broad array of emotions and not just positive. Studies examining this have been mixed. While some laboratory studies have found that MDD is characterized by blunted emotional responses to both pleasant and unpleasant events (a pattern referred to as emotion context insensitivity) [44, 45], other conflicting results have been reported. In one study, depressed participants reported less happiness than controls in response to positively-valenced pictures, but comparable levels of sadness in response to negative pictures, and greater sadness than controls in response to the positive pictures (inconsistent with affective flattening) [46]. Similarly, whereas affective blunting would predict low sensitivity to reward *and* punishment, depressed individuals actually show *enhanced* punishment sensitivity in several studies (reviewed in [35]). Another study directly addressed the relationship between anhedonia and blunted affect among depressed participants and found that anhedonia and affective flattening were orthogonal constructs [47]. Thus, while studies have consistently found that depression is associated with reduced responding to positive stimuli, it is unclear whether it is also associated with reduced to negative stimuli – a pattern possibly due to differences in methodology and paradigms [48].

1.3.1.3 The Role of the Stimulus in Depressive Anhedonia

A final question concerns whether anhedonia in depression is circumscribed to loss of pleasure in specific classes of stimuli, or whether anhedonic depressed individuals experience a broad loss of pleasure in most or all stimuli. An early study suggests that the latter is the case – Fawcett and colleagues [49] found that anhedonic depressed individuals reported loss of pleasure in all areas assessed, including food, sex, social interaction, and work. Consistent with this, depressed individuals experiencing anhedonia are more likely to experience both social withdrawal and appetite loss than depressed individuals without anhedonia, suggesting that anhedonia extends to both physical and social stimuli [34].

However, these findings are qualified by two points. First, although it does appear that depressed individuals *may* experience loss of pleasure in a variety of domains; this is not the same as saying that *all* anhedonic individuals lack pleasure in *all* domains. At the individual level, some may experience a generalized loss of pleasure; some may have diminished pleasure in some domains (e.g., physical stimuli) and intact pleasure in others (e.g., social contact); while others show the opposite pattern (e.g., diminished social pleasure with intact physical pleasure) [50, 51]. In other words, individual depressed persons may experience either broad or circumscribed anhedonia, but a given depressed individual is just as likely to lose pleasure in physical, social, or intellectual stimuli.

A second caveat is that while studies investigating specific aspects of anhedonia have generally agreed that both physical and social pleasure may be impaired during depressive episodes, their long-term course may be different. For instance, a 20-year longitudinal study found that physical anhedonia in depression is stable

and trait-like, associated with depression severity, and predictive of psychosocial functioning [52]. In contrast, another longitudinal study found that currently depressed individuals experienced higher levels of social anhedonia than healthy controls, but that social anhedonia declined over a 1-year follow-up period as participants recovered from depression [53].

1.3.1.4 Can Anhedonia Improve the Classification of Depression?

As we have seen, deconstructing the broad construct of anhedonia into more specific facets can yield greater insight into depression. The reverse may also be true. Depression is a heterogeneous disorder, and presence or absence of anhedonia is a factor that may help parse this heterogeneity. That is, depressed individuals with and without anhedonia differ on a number of clinical variables, suggesting that anhedonic and non-anhedonic depression may be separate syndromes. For instance, individuals with anhedonia are more likely to experience psychomotor retardation, diurnal variation in mood, and brooding (as well as social withdrawal and appetite loss, as mentioned above) [34, 54], and tend to have poorer long-term outcome [55, 56]. Interestingly, anhedonia is *not* associated with overall depression severity [57], and is associated with *lower* rates of other symptoms, such as suicidality [34]. This suggests that anhedonia is not merely a marker of more severe depression, but instead may be associated with a qualitatively different constellation of symptoms.

The idea that anhedonic and non-anhedonic depression are separable syndromes is further supported by their associations with dimensions of personality. Although both types are associated with high neuroticism as compared to healthy individuals, anhedonia is uniquely associated with lower extraversion than controls, as well as lower neuroticism than those with non-anhedonic depression [49, 58]. Indeed, evidence reviewed extensively by Clark and Watson [59] suggests that anhedonic depression is a syndrome or dimension separate from non-anhedonic depression, while the latter is *not* separable from the general distress seen in anxiety disorders. In other words, while anhedonic depression may be a valid syndrome, non-anhedonic depression may have more in common with traditional anxiety disorders than anhedonic depression.

The *DSM-5* "melancholic features" specifier is somewhat similar to the concept of anhedonic depression [13]. However, unlike anhedonic depression, depression with melancholic features is a polythetic construct, and anhedonia per se is not sufficient (and perhaps, not even necessary) for the specifier. As such, many non-melancholic depressed individuals also demonstrate anhedonia or deficits in reward processing [52]. Multivariate genetic evidence suggests that (perhaps in contrast to anhedonic depression), melancholia may simply be a quantitatively more severe form of depression, rather than a qualitatively distinct syndrome [60]. Finally, although there is some evidence for the validity of melancholia [50], others have questioned its validity (at least in its current form) [61]. Future research should examine whether an anhedonic subtype of depression has greater validity and utility than the melancholic subtype.

1.3.2 Anhedonia and Anxiety Disorders

Compared to the large literature investigating anhedonia in depressive disorders, relatively less is known about the role of anhedonia in the anxiety disorders. This is likely due to the widely held belief that although both depression and anxiety are characterized by increased negative affect, anhedonia is thought to be a symptom that distinguishes depression from anxiety [59, 62]. However, studies have examined hedonic deficits in social anxiety and post-traumatic stress disorder (PTSD), and some work has investigated the relationship between worry and reduced positive affect. With many of these studies, however, given the sizeable overlap between depression and anxiety [59], it is unclear whether the effects are due to comorbid depression or the anxiety disorder.

1.3.2.1 Anhedonia in Social Anxiety

In studies of social anxiety, anhedonia is most often characterized as a reduction in positive affect. Additionally, to our knowledge, studies of anhedonia in social phobia have not examined the specific facets/conceptualizations of anhedonia espoused in this chapter, but rather look at anhedonia as a unitary construct (although see [63] for a discussion of how loss of interest/curiosity is different from loss pleasure in social anxiety). Overall, the symptom profile of social anxiety is similar to that of depression as it is also associated with low levels of positive affect and high levels of negative affect [64]. However, depression is more strongly associated with low positive affect than social phobia [65]. Additionally, although socially anxious individuals generally exhibit more depressive symptoms than those who are not, the presence of anhedonia in this population is not completely attributable to depressive symptoms [66].

1.3.2.2 Anhedonia and Post-Traumatic Stress Disorder

The majority of studies examining anhedonia in post-traumatic stress disorder (PTSD) have focused on the presence of emotional numbing. Emotional numbing is characterized by reduced interest and/or pleasure in previously enjoyed activities, a restricted range of emotional expressiveness, and a detachment from others [13, 67]. The presence of emotional numbing in some individuals with PTSD may contribute to the high rate of comorbidity between PTSD and depression [29]. This symptom cluster, however, is broader than just reduced positive affective experience and reflects a reduction of affective experience across different emotions. Factor analytic studies have also shown that numbing and related symptoms are separable from other symptoms of PTSD and have separate correlates [26, 28]. Emotional numbing is further delineated from anhedonia as it is associated with an increased likelihood of a comorbid anxiety disorder (and to a lesser extent a psychotic disorder), whereas anhedonia has been associated with comorbid major depressive disorder [27].

1.3.2.3 Worry and Anhedonia

Worry (i.e., a repetitive thought or series of thoughts related to potential negative future outcomes) is a core symptom of nearly every anxiety disorder. Although the content of these thoughts generally varies by disorder and feared stimuli, worry is a pervasive pattern of thinking that is often associated with increased negative affect and negative mood states [68]. Repetitive thinking of any form (whether it is anticipatory worry or rumination on past events) is known to maximize cognitive resources, which could lead to a restricted range of expressed emotion and reduced ability to experience positive affect as well. Consistent with this hypothesis, studies have shown that both rumination *and* worry, independent of one another, have been associated with decreases in positive affect [69].

In sum, hedonic deficits have been found in social anxiety disorder and post-traumatic stress disorder (and worry more broadly), but much less is known about these deficits in other anxiety disorders, specifically, obsessive-compulsive disorder, panic disorder (although see [40]), and specific phobias. Future research examining distinctive and/or overlapping features of anhedonia that may be present in anxious disorders is needed in order to establish a more complete clinical picture of this class of disorders.

1.3.3 Anhedonia and Substance Use Disorders

Research indicates that anhedonia is a common feature in patients with substance use disorders (SUD) [70, 71]. Even when controlling for overall depressive symptoms, abstinent substance users display high levels of anhedonia during acute and chronic withdrawal periods [72, 73]. Moreover, recent evidence suggests that anhedonia may actually precede substance use initiation, as drug-naïve individuals with elevated levels of baseline anhedonia report greater subjective "highs" during acute intoxication. Baseline anhedonia may also be a risk factor for the development of SUDs, as anhedonic individuals are more vulnerable to SUD onset [74, 75]. However, similar to its presentation in other psychiatric disorders, there are important nuances to the presentation of anhedonia in those with SUDs.

1.3.3.1 APA and CPA Dysfunction in Substance Use

SUDs are characterized by dysfunctional APA (i.e., wanting) and CPA (i.e., liking). Regarding APA, one key feature of substance dependence is craving – a strong desire or urge to ingest drugs. Substance users display preoccupation with drug attainment and compulsive drug seeking behaviors (and given its importance, craving was recently added to the criteria for substance use disorder in DSM5 [13]). Although reward anticipation is often conceptualized as a positive affective experience (i.e., anticipatory *positive* affect), in individuals with SUDs, craving and wanting are marked by high levels of anxiety and distress, coupled with exacerbated

biological stress reactivity [76]. In sum, people with SUDs experience high levels of wanting *without* anticipatory pleasure.

Once the drug is obtained, substance-dependent individuals report diminished CPA, which underlies the phenomenon of "chasing the first high." A large body of research suggests that diminished CPA is a function of physical tolerance, such that chronic drug administration results in adaptive changes in the central nervous system, including altered dopaminergic and serotonergic neurotransmission, which modifies the drug's psychophysiological effects over time [77, 78]. It is likely that these neuroadaptive changes result in a dampened hedonic "peak" as well as a more rapid decrease in consummatory pleasure over time. Thus, in response to drug administration, the addicted individual experiences only a small spike of hedonic pleasure that quickly dissipates as compensatory processes become more efficient at returning the body to homeostasis [79]. There is also some evidence to suggest that compensatory processes may even undershoot baseline functioning, producing a state of dysphoria, which serves to re-elicit cravings and compulsive drug seeking behaviors.

1.3.3.2 Delayed Reward Discounting in SUDs

Similar to major depression, delayed reward discounting is an additional anhedonic feature of SUDs. However, unlike individuals with major depression, who show *reduced* delayed reward discounting, individuals with SUDs exhibit enhanced reward discounting. That is, they display a deficit in appropriately guiding goal-directed behavior towards distal, relative to proximal, rewards [80, 81]. Specifically, drug dependent individuals will often select the proximal reward of drug intoxication over a variety of adaptive behaviors that would lead to deferred rewards that are actually more beneficial to them in the long run. A common example is seen in injection drug users who share hypodermic needles to achieve immediate drug intoxication instead of postponing drug use until the needles can be cleaned or replaced. This more adaptive behavior, which would still allow them to get high but would of course reduce their risk for contracting blood-borne illnesses like Human Immunodeficiency Virus (HIV). Broadly speaking, the desire/anticipation for drug intoxication is heightened, and possibly prolonged, yet the interest in long-term legal, financial, and interpersonal rewards is significantly reduced.

1.3.3.3 Generalization of Anhedonia to Other Stimuli

Data also suggest that over time, substance users experience diminished sensitivity to natural rewards, including food and sexual activity [82, 83]. This broadening of stimuli is thought to be the result of dysregulated hedonic homeostatic balance or a "set-point shift" in hedonic thresholds [32]. As was previously mentioned, repeated and prolonged substance abuse has been shown to change the functioning of reward system neurocircuitry. These changes not only affect the way the body responds to acute drug administration, but also the way all rewards are processed. Over time, substance users require higher stimulation to reach previously achieved levels of

hedonic pleasure. In other words, it is as if individuals with SUDs develop tolerance to natural rewards, in addition to addictive drugs. Together, these separate, yet overlapping, aspects of anhedonia serve to maintain drug taking behaviors and significantly contribute to the pathophysiology of SUDs.

1.3.4 The Role of Anhedonia in Eating Disorders

Eating disorders (anorexia nervosa [AN], bulimia nervosa [BN] and binge eating disorder [BED]), are increasingly pervasive disorders with high rates of comorbid depression and anxiety [84]. Recently, the role of anhedonia in eating disorders has become a topic of growing interest. However, as with the other disorders reviewed thus far, there seems to be a difference in how anhedonia and hedonic responses manifest in different eating disorders [85].

1.3.4.1 Anhedonia and Anorexia Nervosa

Anhedonia in the context of AN is often described as both a lack of desire for food and as a lack of pleasure received from food (i.e., APA and CPA). Indeed, compared to controls, studies have shown that individuals with AN report less desire and pleasure for food when presented with food either visually or olfactorily [86] – an effect that remains, even after controlling for depressive symptoms [87]. Some studies have also suggested that the observed anhedonia towards food in anorexia actually reflects a fear of weight gain towards the food and not a reduced experience of pleasure from the food [88].

Pleasure from food, however, is different from fullness/satiety, as individuals with AN report higher fullness ratings compared to controls (even after a return to normal weight), a finding which may reflect a cognitive distortion or lack of interoceptive awareness [89, 90]. It is also noteworthy that these effects may be specific to food, as some studies have reported that AN individuals rate non-food, but positive stimuli comparable to controls [87].

Neurobiological studies also support APA and CPA deficits to food in individuals with AN. Several studies have reported that while anticipating and responding to food, AN individuals and individuals who have recovered from AN exhibit hypoactivation in neural structures involved in affective processing (e.g., amygdala, anterior insula) [91–93]. Interestingly, studies have also found that even after a meal, individuals with AN, exhibited hypoactivation in some of these structures, suggesting CPA deficits [92, 94].

1.3.4.2 Anhedonia in the Context of Binge Eating

The primary symptom of BN and BED is regular binge episodes (a time period during which an individual eats more than others would consider appropriate in a given amount of time [13]). On the surface, a binge may be thought of as a focal

source of pleasure during which individuals experience CPA [95]. However, a meta-analysis of ecological momentary assessment studies found that positive affect significantly decreases (not increases) after a binge. Of course, this does not rule out the possibility that heightened positive affect occurs *during* a binge episode [96]. Indeed, a significant number of eating disordered individuals report "euphoria" during binges, although the presence of this type of CPA is higher in those with BED than in BN [97].

Binge-eaters have also exhibited abnormal neural responses during food anticipation and intake, suggesting deficits in APA and CPA circuits [98, 99]. Interestingly, one recent study suggests that the neural response during anticipation and intake of pleasurable food may be moderated by the individual's negative mood state [100].

1.3.4.3 Physical Anhedonia and Eating Disorders

It should be noted that the anhedonia in the context of eating disorders is predominantly related to one specific stimulus: food. Although individuals with eating disorders may experience anhedonia in relation to other stimuli, food is the dominant stimulus, which may be due to the fact that food intake is the primary factor in maintaining a desired weight or shape. In contrast to food, Davis and Woodside [101] note that anorexic individuals may derive *more pleasure* from exercise because it helps them burn calories and maintain a low weight. Thus, people suffering from anorexia experience anhedonia in relation to one stimulus (food) but a more pronounced hedonic response to another (exercise).

Our understanding of eating disorders is relatively limited, especially compared with other disorders [102]. Consequently, research examining the role of anhedonia in eating disorders is still in its early phases. From the current evidence, it is clear the relationship between anhedonia and eating disorders is complex and differs across the various diagnoses. More research is necessary to further understand how the hedonic response contributes to the onset, maintenance and remission of eating disorders.

1.3.5 Anhedonia and Other Disorders

Two other disorders are worth mentioning in this chapter – sexual dysfunction and schizophrenia.

1.3.5.1 Sexual Dysfunction

Sexual anhedonia is one form of physical anhedonia that is receiving increased attention in the literature, particularly in its role in sexual dysfunction. Relevant to the present review, the functions of anticipatory and consummatory positive affect

can be illuminating for disorders such as hypoactive sexual desire disorder (HSDD). In Acquired HSDD (as opposed to Lifelong HSDD), individuals report a loss of sexual desire, even though they have previously experienced it. In other words, it is possible that individuals with acquired HSDD no longer experience pleasure during sex (i.e., reduced consummatory positive affect), and therefore lose their desire for sexual interaction (i.e., reduced anticipatory positive affect) [103]. Here, a distinction could also be made between loss of interest and loss of pleasure. Individuals suffering from Acquired HSDD may not lose interest in sexual actions until they experienced a loss of pleasure during sex.

1.3.5.2 Schizophrenia

The presence of anhedonia in schizophrenia is well documented and is one of the most widely studied negative symptoms of schizophrenia and relative to many other symptoms of the disorder, is fairly resistant to treatment [104]. We mention it here briefly, with the caveat that other chapters in this volume are devoted to a more in-depth analysis of the research in this area.

The scope of the anhedonia in schizophrenia literature is so vast, that anhedonia has been conceptualized in nearly all of the aspects presented earlier in this chapter. For example, regarding time course, several studies have reported that individuals with schizophrenia exhibited deficits in anticipatory positive affect but not consummatory positive affect [105, 106]. With regard to the role of stimuli, studies have shown that anhedonia towards social stimuli/situations have different correlates than anhedonia towards physical stimuli [107, 108]. Lastly, studies of anhedonia in schizophrenia have examined whether the disorder is associated with flat or restricted range of affect or specific to low positive emotions. Specifically, while Kraeplin's classic conceptualization of schizophrenia emphasized flat affect (i.e., reduced responding to both positive and negative stimuli), modern conceptualizations generally argue that the deficit in most schizophrenic individuals is specific to positive stimuli (and perhaps even heightened for negative stimuli; [109]). Taken together, these studies suggest that the different models of how anhedonia can be parsed espoused in this chapter are particularly critical for our standing of the neurobiology of schizophrenia.

1.4 Conclusions and Future Directions

Anhedonia, or the diminished capacity to experience pleasure/reward, is an important construct in various psychopathologies. Based on the present review, the role of anhedonia in psychopathology varies across disorders and, most importantly, depends upon the conceptualization or facet of anhedonia being examined. For example, Major Depressive Disorder and Schizophrenia appear to be characterized by deficits in anticipatory positive affect more than deficits in

consummatory positive affect [37, 106]. However, some depressed individuals, such as those with melancholic depression, also experience deficits in consummatory positive affect [41]. As a second example, response to distal vs. proximal rewards is a mechanism that plays a different role in depression vs. substance use disorders [42, 81].

The important lesson to be taken from this review is that anhedonia is a nuanced construct and its different components or facets are likely to have different correlates and underlying neurobiology. Moreover, as anhedonia is one of the hardest features to treat in various psychopathologies [55, 104], a more fine-grained conceptualization of anhedonia would likely identify different targets of treatment. This line of research would, hopefully, lead to improved efficacy rates across psychopathologies.

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