



## Review

## “Don’t [ruminate], be happy”: A cognitive perspective linking depression and anhedonia

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## ABSTRACT

Anhedonia, a lack of pleasure in things an individual once enjoyed, and rumination, the process of perseverative and repetitive attention to specific thoughts, are hallmark features of depression. Though these both contribute to the same debilitating disorder, they have often been studied independently and through different theoretical lenses (e.g., biological vs. cognitive). Cognitive theories and research on rumination have largely focused on understanding negative affect in depression with much less focus on the etiology and maintenance of anhedonia. In this paper, we argue that by examining the relation between cognitive constructs and deficits in positive affect, we may better understand anhedonia in depression thereby improving prevention and intervention efforts. We review the extant literature on cognitive deficits in depression and discuss how these dysfunctions may not only lead to sustained negative affect but, importantly, interfere with an ability to attend to social and environmental cues that could restore positive affect. Specifically, we discuss how rumination is associated to deficits in working memory and propose that these deficits in working memory may contribute to anhedonia in depression. We further argue that analytical approaches such as computational modeling are needed to study these questions and, finally, discuss implications for treatment.

Major Depressive Disorder (MDD) is characterized by many debilitating symptoms, particularly across two key domains: increases in negative affect and decreases in positive affect. MDD is also chronic and recurrent. Though treatments are widely available and are helpful to some patients, up to 40–50% of patients do not respond sufficiently to either antidepressant medications (Furukawa et al., 2016; Trivedi, Greer, Grannemann, Chambliss, & Jordan, 2006) or standard-of-care psychotherapies such as cognitive behavioral therapy (Cuijpers et al., 2014; DeRubeis et al., 2005) (see Cuijpers, Karyotaki, de Wit, & Ebert, 2020 for a review). Among MDD patients who achieve remission, an estimated 40% relapse within two years (Boland, Keller, Gotlib, & Hammen, 2009). Moreover, for individuals who do respond to first-line treatments, anhedonia – the cardinal symptom of depression characterized by a lack of pleasure and/or motivation – often persists (Dunlop & Nemeroff, 2007; McCabe, Cowen, & Harmer, 2009a, 2009b; Nutt et al., 2007; Price, Cole, & Goodwin, 2009; Shelton & Tomarken, 2001). As such, researchers have been charged with the difficult and urgent task of identifying mechanisms that contribute to this disorder, particularly with regard to anhedonia.

The extant body of literature on depression has focused on two main

theoretical perspectives: cognitive and biological mechanisms underlying the disorder. Biological processes have been at the forefront of the field’s study regarding mechanisms underlying anhedonia and deficits in positive affect (e.g., Der-Avakian & Markou, 2012; Gorwood, 2022; Wise, 2008). At the same time, cognitive theories have a long history in depression research (e.g., Abramson, Metalsky, & Alloy, 1989; Beck, 1967; Brown & Harris, 1978) but have largely focused on better understanding sustained negative affect in this disorder. Even though we know anhedonia is a debilitating symptom of depression and is characterized by deficits in positive affect, most cognitive theories have focused less on understanding its etiology and maintenance. Exceptions such as reward devaluation theory (e.g., Winer & Salem, 2016) as well as recent work on dampening (Bean, Summers, & Ciesla, 2022; Vanderlind, Everaert, & Joormann, 2021; Vanderlind, Millgram, Baskin-Sommers, Clark, & Joormann, 2020) show the promise of integrating cognitive theories and work on positive affect but more needs to be done to link cognitive processes that characterize depression and anhedonia. Such an improved understanding of processes underlying anhedonia promises to help prevention and intervention efforts. As such, it is possible that by examining the relation between cognitive constructs and deficits in

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positive affect, we may better understand anhedonia in depression thereby improving prevention and intervention efforts.

One specific cognitive process that has received much attention regarding its contribution to the maintenance of depression is rumination, or the act of perseverative and repetitive attention being paid to specific—and often negative—thoughts. Critically, prior research has focused on rumination as being primarily linked to the sustaining of negative affect (e.g., Alloy & Abramson, 1988; Beck, 2002; Thomsen, 2006) but given the importance of rumination in maintenance of depression, it is possible that it not only affects negative affect but also interferes with processes important for the generation and experience of positive affect—such as the ability to learn from rewards in the environment. This paper focuses on integrating research on cognitive processes in depression with work on anhedonia to better understand how rumination might affect depressed individuals' ability to experience positive affect.

We review the extant literature on cognitive deficits and biases in depression and discuss how these dysfunctions may not only lead to sustained negative affect but, importantly, may also interfere with an ability to attend to social and environmental cues that could restore positive affect. Specifically, we will discuss how rumination may be related to deficits in working memory, and propose that these deficits in working memory may contribute to anhedonia in depression. Lastly, we note how analytical approaches such as computational modeling can be leveraged to study these questions and, finally, discuss implications for treatment.

## 1. Facets of anhedonic experience

Anhedonia is a heterogeneous construct, and is linked to many psychiatric disorders, including substance use disorders (Garfield, Lubman, & Yücel, 2014; Hatzigiakoumis, Martinotti, Di Giannantonio, & Janiri, 2011), schizophrenia (Watson & Naragon-Gainey, 2010; Wolf, 2006) and eating disorders (Tchanturia et al., 2012). For the purpose of this review, we consider the literature on anhedonia as it has been studied in major depressive disorder (see Pizzagalli, 2014; Treadway & Zald, 2011 for additional reviews) and distinguish facets of anhedonia that highlight the need for interrogation from a cognitive perspective.

Anhedonia is broadly defined as a symptom in which an individual perceives a lack of interest or pleasure in activities and experiences that were once pleasurable to them (American Psychiatric Association, 2022). Anhedonia has been tied to reward processing, which encompasses processes related to the engagement in goal-directed behavior towards rewards (appetitive motivation), responses to rewarding stimuli (reward sensitivity), and the ability to learn from rewards to adapt future behaviors (reinforcement learning) (Thomsen, Whybrow, & Kringlebach, 2015). In particular, reinforcement learning has been linked to a large body of literature on anhedonia in MDD (e.g., Pechtel, Dutra, Goetz, & Pizzagalli, 2013; Pizzagalli et al., 2008; Vrieze et al., 2013). This growing body of work has helped elucidate important nuances in reward-related deficits observed in depression.

Past frameworks had characterized anhedonia in depression exclusively as an inability to experience pleasure (e.g., Meehl, 2001; Ribot, 1896); however, more recent frameworks have begun to disentangle the many aspects of reward processing in depression, revealing how reward processing may be disrupted in multiple ways. For example, as Rizvi, Pizzagalli, Sproule, and Kennedy (2016) note in one review on the topic, “this equivocal conceptualization of anhedonia makes measurement imprecise...and refining the concept is imperative if we hope to understand the neurobiological underpinnings of anhedonia” (pg. 3). These authors and others (e.g., Treadway & Zald, 2011) further assert that distinctions within the symptom domain can be made on many levels, e.g., including *when* reward-related deficits occur in relation to receipt of reward. As such, another important distinction that may be helpful in identifying the mechanisms underlying anhedonia in depression is the difference between consummatory and anticipatory

pleasure.

Consummatory pleasure relates more closely to “liking,” satiation, or the pleasure experienced in the moment upon receipt of reward, whereas anticipatory pleasure relates more closely to “wanting,” or to the pleasure one expects to experience from a future rewarding experience. Studies indicate that these two aspects of anhedonia may be differentially impacted in depression, such that anticipatory pleasure is diminished in MDD, whereas consummatory pleasure may remain intact (e.g., Dillon et al., 2008; Shankman, Klein, Tenke, & Bruder, 2007; Treadway & Zald, 2011), and further work indicates that motivational deficits, tied to anticipatory pleasure, may also provide a useful window to understanding anhedonia in depression (Treadway, Buckholz, Schwartzman, Lambert, & Zald, 2009).

An early body of work (Berlin, Givry-Steiner, Lecrubier, & Puech, 1998; Amsterdam, Settle, Doty, Abelman, & Winokur, 1987) utilizing the sucrose sweet-taste test measured individuals' hedonic responses to sweet tastes. This test is designed to measure consummatory reward responding and has consistently demonstrated no differences between MDD patients and controls in affective responses to the receipt of reward. Thus, individuals who are depressed tend to show no differences in so-called behavioral sensitivity to rewarding stimuli. This work proposed the influential notion that there may not be depression-related deficits in consummatory pleasure.

One common task used to study reinforcement learning behaviors in MDD is the Probabilistic Reward task, or PRT (Pizzagalli, Jahn, & O'Shea, 2005a, 2005b), and it has been used to help distinguish subjects' sensitivity to rewards (consummatory) and their ability to learn from rewards. The PRT is a computer-based task in which participants are presented with one of two perceptually similar cues and are asked to use corresponding key presses to indicate which one cue had just been presented. Participants are told that they will be rewarded “sometimes” when they respond correctly, and never when they respond incorrectly. Unbeknownst to the participant, one cue (the “rich” stimulus) is more often rewarded than the other (the “lean” stimulus) throughout the task. The variable of interest in the PRT, the response bias, reflects the extent to which the learner modulates their behavior in response to reinforcement history (Pizzagalli et al., 2005a, 2005b). Importantly, individuals with depression, as compared with controls, display a marked inability to develop a response bias for the rich stimulus during the PRT (Pizzagalli et al., 2008). Notably, in a study of 23 unmedicated depressed subjects and matched controls, Pizzagalli et al. (2008) found that those with depression showed significantly reduced reward responsiveness in the PRT (as indexed by an attenuated or absent response bias). Importantly, trial-by-trial probability analyses revealed that depressed persons were responsive to the delivery of single rewards; that is, they were simply unable to integrate the values of rewards *over time* to generate a persistent response bias towards the more rewarded (rich) cue in the task. These results (i.e., Pizzagalli et al., 2008) further reflect the complex nature of anhedonia symptomatology.

In another behavioral study designed to parse consummatory and anticipatory processes in depression, 38 depressed participants and matched controls rated their liking of humorous and non-humorous cartoons (Sherdell, Waugh, & Gotlib, 2012). Participants then made a series of choices between viewing a cartoon from either group, and each choice required a specified amount of effort the participant would have to exert to view the chosen cartoon. Participants with MDD and control participants did not differ in their consummatory pleasure (i.e., reported “liking”) of the cartoons. However, whereas levels of reward “liking” predicted the amount of effort participants were willing to exert to view the cartoon, high reward “liking” (consummatory pleasure) did not predict whether depressed participants would exert effort to view the cartoon. Levels of anticipatory pleasure, on the other hand, did predict the amount of effort depressed participants would exert to view the cartoon, such that lower levels of anticipatory pleasure led to less effort exerted by participants with MDD – an effect that was not seen in control participants. This study also suggests that individuals with depression

may not have deficits in the ability to experience pleasure; rather, they may anticipate experiencing less pleasure with a given reward and thus fail to modulate their behavior as a function of past experience in order to maximize future rewards.

A third line of behavioral research, by Treadway, Bossaller, Shelton, and Zald (2012); Treadway et al. (2009). In this task, effort-based decision-making is studied as participants are presented with a series of trials in which they choose to expend more or less effort in order to gain varying levels of monetary rewards. In one study, using an unselected sample of undergraduates, the researchers found that higher levels of self-reported anhedonia were associated with decreased willingness to expend effort for rewards (Treadway et al., 2009). In another study, patients with a current MDD diagnosis were shown to exhibit decreased willingness to expend effort for rewards as compared to healthy controls as well as a decreased ability to use past information about reward magnitude history and probability of reward receipt to modify and update their future choice behavior (Treadway et al., 2009). Taken together, these studies provide stark behavioral evidence for depression-related deficits that are specific to the motivational (i.e., anticipatory) domain.

Neuroimaging research further supports the notion that different facets of anhedonia are differentially linked with symptoms seen in depression. For example, Kocsel et al. (2017), used fMRI to examine activation in reward-related areas of the midbrain while individuals with a spectrum of scores on the ruminative response scale (RRS; Treynor, Gonzalez, & Nolen-Hoeksema, 2003) completed a monetary incentive delay task (MID; Knutson, Westdorp, Kaiser, & Hommer, 2000). In the MID task, visual stimuli (e.g., shapes and colors) are used as incentive cues to convey the probability and magnitude of monetary rewards. The authors found that participants who scored high on trait rumination showed reduced midbrain activation during reward anticipation; however, they found no relation between rumination and midbrain activation in response to reward consumption. Though this study was conducted in a group of non-depressed individuals, the authors conclude that rumination may relate to the disrupted processing of anticipatory/motivational reward responses, rather than consummatory reward responses.

Taken together, the aforementioned findings support the idea that cognitive processes associated with depression, such as ruminative thinking, are related to difficulties anticipating rather than consuming rewards. These findings therefore raise the question: if individuals with depression show some intact ability to experience the benefits of reinforcement, what, then, is hindering their ability to integrate the values of rewards over time (e.g., Pizzagalli et al., 2008), anticipate pleasure (e.g., Sherdell et al., 2012), and motivate themselves to engage in goal-oriented behavior?

## 2. Reward processing in depression

Previous work has identified neurobiological correlates of anhedonia, focusing on constructs like appetitive motivation (Germans & Krings, 2000), reward sensitivity (Thomsen et al., 2015), and reinforcement learning (Huys, Pizzagalli, Bogdan, & Dayan, 2013). A consensus has emerged, stating that key brain structures, such as the basal ganglia, amygdala, medial prefrontal cortex, and orbital prefrontal cortex are critically implicated in the ability to carry out reward processing, and that this is ultimately driven by midbrain dopaminergic neurons (Haber & Knutson, 2010). More specifically, decades of research provide evidence for the integral role of the neurotransmitter dopamine, originating from the ventral tegmental area and projecting to the ventral striatum, in generating reward prediction errors (RPEs) – the difference between expected and received rewards. The RPE is the primary teaching signal of the reinforcement learning system, promoting learning from reinforcement over time (Schultz, Dayan, & Montague, 1997).

Through this work, we have learned much about the neurobiological structures and deficits in RPE-signaling that are implicated in anhedonic

symptomatology.

Response bias, as measured by the PRT, has also been tied to the neurobiological framework of reward processing described above. For example, Pizzagalli et al. (2008) showed that administration of a low-dose dopamine agonist (hypothesized to decrease dopamine signaling through presynaptic autoreceptor activation) impaired the acquisition of a response bias. These results—along with a large and influential body of research (e.g., Schultz et al., 1997; Schultz, 2007; Niv, 2009; Tobler, 2010; Glimcher, 2011)—suggest that dopamine signaling is necessary to reinforce actions that lead to reward. A recent study utilizing the PRT and positron emission topography (PET) also found that individual differences in reward learning on the PRT were related to dopamine transporter binding potential in the ventral striatum (Kaiser et al., 2018).

Although we know much about reinforcement learning in the framework of RPE and dopamine signaling—and extant literature have mostly focused on these such neurobiological correlates (see Kielisch, Valton, & Roiser, 2022 for an extensive review on this topic)—there are important individual differences in the ability to process environmental cues related to reward. Prior research has attributed some of these differences to aberrant RPE signaling (see Treadway & Zald, 2011 for review). However, there is reason to believe that additional cognitive processes (beyond canonical corticostriatal reinforcement learning) may play an important role – that is, individual differences in how humans assign values to cues in the environment may not be captured by variance in this single neural mechanism. Critically, we know also that learning from rewards is not a singular process – it also appears to rely heavily on the flexible updating of working memory (Rmus, McDougle, & Collins, 2021; Taylor et al., 2004), a cognitive construct shown to be strongly implicated in depressive symptomatology (Christopher & MacDonald, 2005; Yoon, LeMoult, & Joermann, 2014). As such, by focusing narrowly on the canonical reinforcement learning system, we may be missing a key part of the story concerning MDD and reward processing. By taking into account individual differences in cognitive processes that contribute to the maintenance, updating, and integration of action and stimulus values over time, a more complete picture may emerge.

One key cognitive process to examine in completing this picture is working memory functioning. Working memory refers to the cognitive system for temporarily storing, actively maintaining, and manipulating information across a short delay (Cowan, 2008). Working memory is also necessary to carry out temporally relevant goal-directed tasks (Miller, 2013). In the past several years, work by Collins and Frank (2012); Collins, Albrecht, Waltz, Gold, and Frank (2017); Collins and Frank (2018); Collins, Ciullo, Frank and Badre (2017) has shown that the learning during simple stimulus-response reinforcement learning paradigms is closely related to working memory. In these studies, subjects participated in a basic instrumental learning task where the number of stimuli-response pairings (i.e., the “set size”) that had to be learned at a given time was varied throughout the task. Varying cognitive load in this manner allows one to separately track the reinforcement learning and working memory systems, via behavioral (Collins & Frank, 2012, 2018) and neural signatures (Collins et al., 2017, Collins & Frank, 2018). Two pieces of recent evidence from Anne Collins and colleagues point to a complex interaction, where working memory feeds predictions to the reinforcement learning system (i.e., cooperation) but the two systems compete during decision-making (Collins & Frank, 2018). Interestingly, if working memory broadcasts predictions to the reinforcement learning system (e.g., “if you perform action X you will likely be rewarded”), computational modeling suggests that the RL system should then be less “surprised” by the working memory-predicted outcome (e.g., a reward). Indeed, this interaction has been revealed in fMRI – during learning conditions that rely heavily on working memory, reward prediction error signals in the striatum, key node in the RL network, are attenuated (Collins et al., 2017). Taken together, these findings suggest that taxing working memory should have a complex

effect on reinforcement learning; perhaps making it learn more slowly, but also allowing it to contribute more to decision-making. This work is an important first step to uncovering the interactions between working memory and reinforcement learning – rather than viewing reward learning as a monolithic process, these authors (Collins, 2019; Collins, Albrecht, et al., 2017; Collins, 2018; Collins & Frank, 2012) suggest that working memory and reinforcement learning are dynamically integrated and interact with one another (Collins & Frank, 2018), simultaneously shaping choice behavior.

Given that individuals with MDD show broad impairments across working memory and other areas of executive functioning (see Snyder, 2013 for a review), the aforementioned findings suggest that working memory processes should be examined further with respect to MDD and reward processing. Particular promise for this research may lie specifically in executive function constructs of working memory and cognitive control, which are implicated in the updating and monitoring of choice values (Domenech & Koechlin, 2015; McDougle & Collins, 2021; McDougle, Ballard, Baribault, Bishop, & Collins, 2022). For example, we know that extrastriatal regions, such as the anterior cingulate cortex (ACC) and the dorsolateral prefrontal cortex (DLPFC), are uniquely tied to updating the value of choices and/or stimuli. We also know that cognitive control, or the ability to exert mental effort needed to sustain task-relevant behavior, is thought to be necessary for carrying undisrupted RPE signals to necessary brain regions (Holroyd & Umemoto, 2016). How can we combine this knowledge to better understand the clinical presentation of depression and dissociate deficits in reinforcement learning versus executive function?

We propose that cognitive deficits may contribute to disrupted learning from rewards in MDD and as outlined below from a cognitive perspective, we suggest that rumination may play a key role in understanding these impairments.

### 3. Cognition and emotion regulation in depression

Individuals with MDD—and even those with subthreshold depressive symptoms—report a number of cognitive difficulties. In addition to issues with concentration, deficits and biases in memory and attention have been reported during depressive episodes (Trivedi & Greer, 2014). Whereas biases refer to preferences for one emotion over another (or over neutral), deficits lead to more errors and/or reduced efficiency in responding. Research has shown both global and emotion-/content-specific impairments in executive functions (see Rutherford & Joormann, 2022 for a review covering both). We know that depressed individuals show overall deficits in executive functioning (e.g., Snyder, 2013), for example, but it is likely that depression-related memory and attentional biases contribute to this (see Everaert, Koster, & Derakshan, 2012 for a review on this topic). Research on these deficits and biases suggest that they may also precede the onset of depression, indicating that they play a role in one's vulnerability to the disorder (Goodyer, Herbert, Tamplin, & Altham, 2000). Importantly, these deficits may be linked to the emotional problems that define depression: sustained negative affect and decreased positive affect (Gotlib & Joormann, 2010). In particular, cognitive biases may help explain individual differences in the ongoing maintenance of negative affect and difficulties with experiencing positive affect, which are hallmark features of depression. Mood-congruent cognitive biases maintain attention on negative stimuli in the environment, increase accessibility of negative material in memory, and result in negative interpretation of ambiguous material, all of which prolong negative affect and hinder the regulation of negative mood states.

Cognitive processes are closely related to individual differences in emotion regulation, or the ability to manage and modulate one's emotions in response to affective experiences (Gross & Thompson, 2007), and working memory in particular has been linked to emotion regulation ability in individuals with depression (see Joormann & Quinn, 2014 for a review). Emotion regulation deficits have been identified as an

important risk factor for and symptom of depression (Durbin & Shafir, 2008), and depression is frequently considered a disorder of emotion dysregulation (Joormann & Stanton, 2016). Thus, emotion regulation has provided an important bridge to understanding the role cognitive deficits and biases play in the sustained negative affect that is characteristic of depression. Rumination, for example, a process in which repetitive and perseverative attention is paid to specific thoughts, may reflect a failure of cognitive control in that attention gets “stuck” on salient, but not necessarily goal-relevant, aspects of a situation (Nolen-Hoeksema, 2000). Rumination is also associated with the onset (Nolen-Hoeksema, 2000), duration (Nolen-Hoeksema & Morrow, 1993), and severity (Just & Alloy, 1997) of depression. Cognitive reappraisal, on the other hand, a process in which an individual actively changes their thoughts about an emotional event/stimulus (Gross, 1998), has been identified as an important emotion regulation strategy that is closely related to people's ability to exert cognitive control (McRae, Ochsner, Mauss, Gabrieli, & Gross, 2008) and remove negative, repetitive content from working memory. Cognitive reappraisal is thus associated with better treatment outcomes in depression (e.g., Garnefski & Kraaij, 2006; Kraaij, Pruyboom, & Garnefski, 2002).

Effective emotion regulation requires updating the content of working memory and exerting control over mood-congruent, goal-irrelevant thoughts, and replacing them with goal-relevant information. For example, imagine the following situation:

*John arrives to work one morning after a long commute, only to realize he forgot his lunch and his wallet at home. John becomes extremely upset, and begins thinking “Why can't I do anything right? I forget everything these days! What is wrong with me? I am such a failure!” John spends most of the morning perseverating on this mistake and how he “can't do anything right” that he misses his first meeting. He has become so upset and angry at himself that by noon he has not accomplished any tasks and can't think of anything but his mistakes, so he leaves work early to go home.*

In this situation, we see that an event (John forgetting his lunch) activates an emotional response (anger and sadness), leading to an overhaul of mood-congruent thoughts in working memory (i.e., rumination). Though rumination is masked here as “problem-solving,” such as trying to understand “what's wrong” with oneself, this example demonstrates the goal-irrelevance of mood-congruent rumination. In fact, this cognitive process—often labeled ruminative brooding (Rude, Little Maestas, & Neff, 2007)—serves only to exacerbate John's anger and sadness. Alternatively, if John were to instead exert cognitive control over these thoughts and divert his attention from them (thus removing the ruminative content from working memory), he might have been able to implement goal-relevant solutions, such as asking a coworker to borrow money for lunch, and thus not miss his meeting.

As illustrated above, understanding the relation between rumination and working memory—particularly, focusing on the executive functions that subserve working memory, such as the ability to attend to salient and relevant cues, exert cognitive control, monitor and update the contents of working memory, and inhibit ruminative thoughts—is crucial for understanding how individuals regulate emotion. As such, homing in on literature that examines relations between rumination and working memory may provide a useful cognitive perspective into not only increases in negative affect, but also the lack of positive affect in depression. Specifically, because evidence suggests that working memory and reinforcement learning are dynamically integrated (e.g., Collins & Frank, 2012), rumination may indirectly affect the ability to learn from rewards and experience positive affect by interfering with working memory.

### 4. Working memory and executive function in depression

Nearly all models of working memory highlight its role in complex cognitive tasks such as planning, learning, and reasoning. Importantly,

however, working memory is capacity-limited. As such, information that is stored and manipulated in working memory should be goal-relevant. Under the purview of working memory, executive functions are defined as “general-purpose control mechanisms that modulate the operation of various cognitive subprocesses and thereby regulate the dynamics of human cognition” (Miyake et al., 2000). Many different aspects of executive functions have been proposed to relate to depression, including maintenance of information, switching between internal sets, inhibiting prepotent behaviors, selecting among different options, and monitoring representations within working memory such that the appropriate updating of working memory can occur in light of new information (Miyake et al., 2000).

A wealth of research has corroborated the theory that the above facets of working memory are highly implicated in depression and tied to rumination. Indeed, early research by Nolen-Hoeksema (1991) illustrated that an increased tendency to ruminate on negative information, combined with difficulties distracting oneself from such negative material, play a central role in the maintenance of depressed mood. It is also known that a stable tendency to respond to negative life events and negative mood states with ruminative thinking (a ruminative style) is a marker of vulnerability for developing depression.

Donaldson, Lam, and Mathews (2007) studied the effect of rumination on attention and found that MDD participants who score high on rumination tend to persevere more on negatively valenced stimuli. The authors theorize that an inability to divert attention from negative stimuli may be an initial route through which rumination affects cognition and, therefore, by which negative thinking persists. Pe et al. (2013) also studied the impact of rumination and other forms of perseverative thinking (i.e., worry) on attentional biases towards negative stimuli. Similarly, they also demonstrated that rumination is associated with a bias for negatively valenced stimuli and is negatively associated with attention to positively valenced stimuli. This maladaptive bias towards negative stimuli, coupled with a failure to acknowledge positive stimuli, may contribute to persistent rumination and the inability to disengage from ruminative cycles. These authors are not the first to recognize negative emotion-related attentional biases in depression; a large body of literature corroborates that depressed individuals may both preferentially attend to negative stimuli and be less likely to attend to positive stimuli (Armstrong & Olatunji, 2012; Bouhuys, Geerts, & Gordijn, 1999; Bradley & Mathews, 1983; Bradley, Mogg, & Williams, 1995; Gotlib et al., 2004; Gur et al., 1992; Joormann & Gotlib, 2010; Joormann & Gotlib, 2007; Winer & Salem, 2016).

Importantly, however, as Mathews and MacLeod (2005) assert in a review of this literature, and as others (e.g., LeMoult & Gotlib, 2019) have supported, the issue may not simply be that too much negative information is entering working memory and conscious awareness. Rather, a more important predictor of depression onset, severity, and chronicity may be that this information is not making its way out.

Widely accepted models of working memory and executive functions (e.g., Friedman et al., 2008; Miyake et al., 2000) focus on three specific executive functions that are necessary for optimal functioning: (1) “updating” (adding or removing) of relevant (or irrelevant) information in working memory, (2) “shifting” between tasks or mental states/sets, and (3) “inhibiting” or suppressing automatic responses to stimuli. Because working memory is capacity limited, it is important that individuals continue to monitor and update the limited information that can be held in working memory at any given time. Indeed, these functions have been targets for research in understanding cognition and depression, as they are integral processes for the monitoring and removal of negative information and the integration of positive material into working memory—two processes that, when disrupted, are illustrative of depressive symptomatology.

A body of work has shown an impairment in the ability to inhibit negative information among a variety of relevant populations, including depressed patients (Goeleven, De Raedt, Baert, & Koster, 2006) and dysphoric undergraduates (Joormann & Siemer, 2004). Furthermore,

this negative valence-specific impairment in inhibition has been shown to persist even among remitted-depressed individuals (Joormann & Gotlib, 2007; Joormann & Siemer, 2004) and never-depressed daughters of depressed mothers (Joormann, Talbot, & Gotlib, 2007). More recent work has elucidated what appears to be a unique role of rumination in this effect.

For example, one study by Yoon et al. (2014) compared working memory capabilities across individuals with MDD and social anxiety disorder (SAD), two disorders that are often found to be comorbid with one another (e.g., Adams, Balbuena, Meng, & Asmundson, 2016). Participants in this task memorized two lists of words on each trial and were told to ignore one of the lists of words. Later, participants were asked to indicate whether or not a single word belonged to the relevant of the two lists. The authors found that individuals with MDD had greater difficulty discarding and inhibiting no-longer-relevant information from working memory (i.e., greater working memory intrusion), whereas individuals with SAD showed no evidence of such interference. These authors theorize that the working memory capacities may be largely influenced by a motivation to “do well,” as would be expected for those with SAD, but perhaps not for those with motivational deficits as seen in depression. Interestingly, working memory performance in this study correlated with ruminative tendencies only for individuals with MDD, suggesting an important interaction between depressive symptomatology and rumination, which predicts working memory performance.

Similarly, De Lissnyder, Koster, Derakshan, and De Raedt (2010) used the Affective Shift Task to examine the relation between depressive symptoms and executive functioning and probed the role of rumination. In this task, participants are asked to perform an odd-one-out search based on a stimulus characteristic that was cued at the beginning of the trial (e.g., specific emotions, gender). The authors found that among all participants, depressive symptoms in general were not related to inhibition abilities and were only moderately related to set-shifting abilities. However, rumination (ruminative brooding in particular) was related to valence-specific impairments in inhibition and set shifting. That is, participants higher on rumination had more difficulty discarding information from previously negative cues, limiting their ability to both shift mental sets to respond to a new target and inhibit prepotent responses to select information in line with new goals. This work highlights a specific role of rumination in the ability to shift attention away from negative content and efficiently utilize executive function for goal-relevant purposes.

Additional studies have shown that depression and, more specifically, the tendency to ruminate, is associated with an impaired ability to remove negative information from working memory and update its contents (e.g., Joormann & Gotlib, 2008). Meta-analytic work by Yang, Cao, Shields, Teng, and Liu (2017) assessing rumination and core executive functions found robust negative associations between rumination and both shifting and inhibition abilities. This again suggests that through rumination, depressed persons do not only have too much negative irrelevant information making its way into working memory, but have difficulty switching between mental sets (i.e., away from the negative information) to address current goals.

Additional research has shown that working memory capacity itself may be reduced because of depressive cognitions (e.g., ruminative thoughts) (Hubbard et al., 2016). This work demonstrated that when a working memory span task is modified to include depressive cues, dysphoric individuals show greater deficits in working memory performance than they do on a non-modified task. Importantly, this effect is enduring, such that when dysphoric individuals receive depressive cuing in a task prior to the un-cued task, they perform worse on the un-cued task compared to dysphoric participants who received the two tasks in the opposite order. Thus, when depressive thoughts become a part of depressed individuals’ conscious awareness, working memory resources are occupied. Lyubomirsky, Kasri, and Zehm (2003) reported similar findings in a study where they asked dysphoric and non-dysphoric individuals to concentrate on either neutral, self-focused, general

emotion-focused, or symptom-focused (i.e., ruminative) thoughts before completing a series of cognitive tasks. They found that individuals who were dysphoric and asked to ruminate on their negative affect showed significantly poorer performance on the tasks, and also reported more task-irrelevant thoughts compared to non-dysphoric individuals and dysphoric individuals not asked to ruminate. Taken together, this work suggests that depressive rumination—and even the mere cuing of depressive thoughts in individuals with depressive symptomatology—has a significant deleterious effect on working memory.

## 5. “Be Happy”

The aforementioned body of work demonstrates how ruminative thoughts are tied both to depression-related outcomes and impaired working memory and executive functioning. This work has critical implications in considering how we treat the symptoms of depression. To date, however, much of the work on the treatment of depression has focused on rumination’s role in maintaining negative affect, despite individuals with MDD consistently reporting the restoration of positive affect as their primary treatment goal (Demyttenaere et al., 2015). The literature reviewed above suggests that through rumination, depressed persons do not only have too much negative irrelevant information intruding into working memory but also have difficulty switching between mental sets (i.e., away from negative information) to complete their goals.

As such—and as illustrated in Fig. 1—it is also possible that the process of rumination, which taxes core executive functions, interferes with one’s ability to learn from rewards in the environment, and thus might also constrain positive affect (i.e., anhedonia). In other words, researchers have leveraged cognitive processes to understand what makes “don’t ruminate” a difficult task, but has yet to leverage cognitive processes to gain a better understanding of what makes “be happy” equally challenging.

Past ideas about anhedonia and reward-related deficits in depression have viewed reinforcement learning as a singular process stemming from the generation of reward prediction errors which guide our future behavior (see Glimcher, 2011 for a review). However, attention must be paid to the process of rumination and how cognitive processes may play a pivotal role in the learning deficits that contribute to anhedonia. As such, future work should examine how perseverative cognition affects working memory and interferes with reinforcement learning to guide human behavior and affect in depressed individuals.

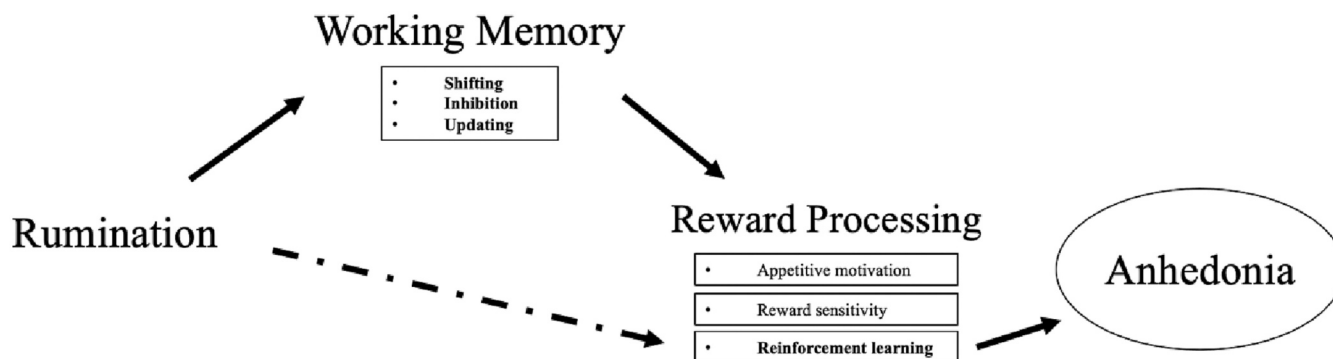
## 6. How to study the question at hand

We have made the case for a need to interrogate the relation between rumination, working memory, and reinforcement learning to better understand the maintenance of anhedonia as it occurs in depression. We

would like to also assert that computational methods may be one of many particularly helpful tools for examining this question. Computational models have been used in some studies, and not others, which we argue is one limitation to this body of work. Computational research involves using precise mathematical formulas to make sense of the behavioral data we gather. By doing so, we can probe and parse the discrete mechanisms that underlie complex behaviors. Unfortunately, by only studying the general behavioral outcomes of reinforcement learning tasks (e.g., overall accuracy), it is difficult to separate reinforcement learning effects from working memory effects (Collins & Frank, 2012; Eckstein, Wilbrecht, & Collins, 2021), a point we return to below.

To our knowledge, only two studies have directly examined rumination and reinforcement learning. In one study, Whitmer, Frank, and Gotlib (2012) experimentally induced rumination utilizing an induction protocol famously created by Nolen-Hoeksema and colleagues (Lyubomirsky & Nolen-Hoeksema, 1995; see Lyubomirsky, Layous, Chancellor, & Nelson, 2015 for a review of this literature). Dysphoric and non-dysphoric individuals participated in this experiment and were assigned to either rumination or distraction conditions. In the rumination condition, participants were instructed to concentrate on a series of prompts that were self-focused and abstract, such as, “Think about why you react the way you do.” In the distraction condition, participants were instructed to concentrate on prompts that were not self-focused, were concrete, and were neutral in valence, such as “Think about a boat slowly crossing the Atlantic.” The distraction inductions therefore served as a control condition. Participants then completed the Probabilistic Selection Task (PST; Frank, Seeberger, & O’reilly, 2004) and, although computational modeling was not performed, behavioral responses to reward and punishment were analyzed. The researchers found that dysphoric individuals in the rumination condition more often chose stimuli which rarely yielded rewards. Thus, participants demonstrated an impaired ability to learn which actions were not worthwhile. Translating this to the clinical presentation of rumination and its disruptions to learning, we can think back to our earlier example of John, the individual who forgot his lunch on his way to work. For an already dysphoric individual like John, these results help to explain his continued engagement in ruminative thinking about his forgetfulness and failures, such as “What is wrong with me?” and “Why can’t I do anything right?” despite the fact that such mental queries reliably fail to provide him reassurance or to alter his mood.

In another study, the relation between rumination and reinforcement learning was interrogated with particular attention paid to the role of selective attention. In this study, rumination was manipulated within subjects using a modified rumination induction, while participants completed a multidimensional learning task designed to rely on attention. Results of this study demonstrated that state rumination did impair performance learning, but that this impairment was not related to



**Fig. 1.** Rumination taxes facets of working memory (i.e., shifting inhibition and updating skills). Deficits in working memory interfere with the ability to properly carry out reinforcement learning. Decreased reinforcement learning ability is associated with symptoms of anhedonia. As such, rumination indirectly affects the ability to learn from reinforcements via its effects on working memory.

participants' attentional breadth, which was the authors' proposed mechanism for learning disruption (Hitchcock et al., 2022). The authors note that it is important for future research to capitalize on additional computational models, which may capture other precise processes underlying these learning deficits.

Whitmer et al. (2012) and Hitchcock et al. (2022) have provided the only evidence, to our knowledge, of investigations regarding rumination and its effect on reinforcement learning. Thus, though we have theoretical (see above sections) and empirical (e.g., Jones, Siegle, Muelly, Haggerty, & Ghinassi, 2010; Kaiser et al., 2016; Watkins & Brown, 2002) reason to believe that rumination's interference with working memory plays a pivotal role in the ability to execute successful reinforcement learning, this has yet to be clearly interrogated scientifically.

Another reason that relations among rumination, working memory, and reinforcement learning, have been so difficult to study, is that many studies on depression and cognition utilize different paradigms and computational frameworks, making it difficult to draw general conclusions. For example, Blanco, Otto, Maddox, Beavers, and Love (2013) used a reinforcement learning task and computational modeling to dissociate model-based learning (i.e., learning that uses a rich model of the task environment) from model-free learning (i.e., a simpler habitual learning system). Depressive individuals' choices were better explained by the simpler reinforcement learning model, suggesting that depression may be associated with a model-based reinforcement learning deficit (Huys et al., 2013). In another recent example, Ruppel, Stankevicius, Huys, Steele, and Seriès (2018) observed diminished memory of reward history in participants with MDD during a Pavlovian reward-learning task. Finally, a recent study using a reversal learning task revealed slower adjustment to abrupt changes in task contingencies in MDD (Mukherjee, Filipowicz, Vo, Satterthwaite, & Kable, 2020). Could these three examples – an association of depressive symptoms with attenuated model-based reinforcement learning, reduced memory of recent reward history, and disrupted reversal learning – be explained by a similar underlying deficit, perhaps involving executive function and working memory? Here, computational psychiatric approaches could help synthesize disparate results from different tasks by formalizing general cognitive systems that may be useful across those tasks.

One paradigm that shows particular promise for approaching this question involves use of a task and computational model developed by Collins and colleagues, called the Reinforcement Learning-Working Memory Task (RLWM; 2012). Importantly, this task embeds a working memory manipulation within a simple reinforcement learning paradigm, allowing researchers to separately examine the effects of these two processes. In this task, participants learn to pick one of three actions in response to stimuli over the course of 12 separate experimental "blocks." Set size in each block varies from  $n = 2$  to  $n = 5$ , allowing one to model the separate influences of capacity-limited working memory from incremental reinforcement learning systems (see Eckstein et al., 2021 for a review on this topic). This powerful task has recently been used in the clinical setting: Compared to healthy controls, patients diagnosed with schizophrenia not only perform worse (in terms of learning) overall, but their deficits can be accounted for by changes in working memory parameters (Collins et al., 2017). Interestingly, the computational model parameters and behavioral analyses in that study indicated that reward-based reinforcement learning was actually unaffected in that group. Thus, previous theories that linked schizophrenia and reinforcement learning may have conflated deficits in working memory and deficits in learning. Though it should be noted that schizophrenia is a categorically different psychiatric illness than major depression, this illustrates one example of how computational modeling can—and has been—applied to behavioral data to parse working memory and reinforcement learning dynamics and answer complex clinical questions. Unfortunately, there is no published work disentangling working memory versus reinforcement learning deficits in depression, suggesting this is an area ripe for future research. Carefully designed tasks such as RLWM and more holistic computational

frameworks should prove useful for gathering precise measurements of specific clinical deficits in depression as well.

Above work, such as that of Ruppel et al. (2018), demonstrates how broad behavioral findings, such as patients with MDD (as compared to controls) perform worse in learning overall can be attributed to specific mechanisms that underlie behavioral dysfunction (e.g., the ability to encode memory of reward and update representations of values) via computational modeling. Examples such as these highlight the need to further explore computational methods to gain a better understanding of what precisely goes awry when individuals with MDD and those who are actively ruminating exhibit learning difficulties, and how this might relate to a general impaired ability to learn from rewards in one's environment, as is fundamental to anhedonic clinical presentations.

## 7. Future directions and limitations

We have outlined a hypothesis explaining how rumination, working memory, and reinforcement learning may be linked to contribute to and maintain anhedonia in depressed individuals throughout this review. However, it is important to note a few limitations and areas for future research. Firstly, though we note that the body of work interrogating cognitive mechanisms underlying anhedonia is limited, one existent theory is worth reviewing. One important line of research has used cognition to help explain why appetitive, reward-seeking behaviors are decreased in depression and deserves mentioning. One such theory, the reward devaluation theory, proposes that depressed individuals *actively* avoid positive/rewarding stimuli due to a chronic deficit in approach motivation (Winer & Salem, 2016). Proponents of this theory have argued that due to experiences throughout development, individuals with psychopathology have learned that positive information may actually be more threatening than neutral information. This is because, unlike negative information, which cues avoidance and protective behavior, positive information promotes approach behavior, only to disappoint—or even endanger—the depressed individual if and when it proves negative or harmful. Reward devaluation theorists therefore propose that depressed individuals show biases away from positive information/stimuli as a result. This theory also suggests that individuals suffering from depression show a propensity to diminish or eliminate their positive responses to rewards when they are unable to avoid positive information altogether. The latter portion of this theory is well aligned with traditional theories of "emotional dampening," which is an emotion regulation strategy akin to rumination in which individuals down-regulate positive emotions rather than savoring them. Indeed, many studies have shown that depression and depressive symptoms (specifically anhedonia) are strongly associated with the use of positive emotion dampening (e.g., Feldman, Joermann, & Johnson, 2008; Raes, Smets, Nelis, & Schoofs, 2012; Werner-Seidler, Banks, Dunn, & Moulds, 2013) and reward devaluation theory may help to explain why.

Our proposed hypothesis differs from reward devaluation theory in a crucial regard, however, by highlighting differential deficits in anticipatory/approach motivation vs. consumption phases of reward processing. Though individuals with depression may experience an urge to "turn down" positive emotions for fear that a negative event may loom around the corner, we highlight that these individuals do have the ability to experience pleasure, nonetheless. As such, over time, principles of basic reinforcement learning would suggest these individuals would modify their behavior to maximize rewards. Unfortunately, studies showing a key deficit in the ability to anticipate and project the magnitude of a rewards in depression (e.g., Treadway et al., 2012) suggest that the value of rewards are, in fact, not being updated overall. We hypothesize that deficits in working memory due to ruminative processes play a key role in disrupting this learning process. It may be the case, however, that post-event value diminishment (as in reward devaluation) may also play a role, and this should certainly be interrogated in future research.

Secondly, in this paper, we refer to rumination broadly as the process

of perseverative, negative thinking about one's thoughts and feelings, and their causes and consequences. Research has shown, however, that rumination is a multi-faceted construct (Bernstein, Heeren, & McNally, 2019), distinguished by at least two major types of rumination: brooding and reflection (Schoofs, Hermans, & Raes, 2010). Whereas brooding tends to be characterized by negative thoughts about one's internal states and is shown to be a transdiagnostic feature of many psychiatric disorders (Watkins, 2009), reflective rumination describes a process in which one analyzes past events for problem-solving. Indeed, the latter form of rumination can be effective (e.g., Kross, 2009; Kross, Gard, Deldin, Clifton, & Ayduk, 2012a, 2012b); however, it is not generally the common form of rumination reported and treated in depression, and high rates of brooding are reported in mood disorders (Olatunji, Naragon-Gainey, & Wolitzky-Taylor, 2013). As such, though we referred to rumination as a general process here, we acknowledge that it, too, is a heterogeneous construct and the type of rumination we consider fitting most into our framework would be characterized more closely to emotion-driven brooding rather than problem-solving reflection. Further work should explore nuances in how the type of rumination one engages with is associated with executive functioning.

Thirdly, we present computational modeling in this paper as one very helpful tool for interrogating complex relationships among clinical symptoms. Nevertheless, it is important to note that interpretations of parameters derived from a model rely on the assumption that one's model is valid. Though model validity (i.e., how "accurate" a computational model is at measuring a specific phenomenon) is a difficult issue in this area of research, there are several strategies future researchers can implement to help increase the validity of their models. For example, out-of-set cross-validation is one useful way to test the ability of a model to generalize across subjects. Moreover, directly comparing the capacity of model-derived metrics (i.e., free parameters fit to subject behavior) versus behavioral summary metrics (e.g., % correct in a learning task) to predict depressive symptomatology in a sample will be key to validating models (e.g., Mukherjee et al., 2020). Additionally, testing generative performance of a given model (i.e., the ability to reproduce the behavioral effect of interest in simulated datasets) will also greatly improve model validity (e.g., Palminteri, Wyart, & Koehlin, 2017). Finally, the combined modeling of behavioral and neuroimaging data can help ground computational theories of learning, and the effects of depression on learning, in the brain (e.g., Rutledge et al., 2017).

Lastly, we would like to note and emphasize two separate points. First, many learning theories of depression have focused on how rumination is "reinforced" and maintained, because it is viewed by those with depression as having a positive value (e.g., gaining deeper insight and understanding of one's problems) (see Ramnerö, Folke, & Kanter, 2016 and Watkins & Nolen-Hoeksema, 2014 for learning theory accounts of depression). In Ramnerö et al. (2016) paper on learning theory in depression, the authors state that rumination is maintained through both negative (e.g., Martell, Addis, & Jacobson, 2001) and positive (e.g., Nolen-Hoeksema, 2013; Wray, Dougher, Hamilton, & Guinther, 2012) reinforcement. However, the authors also state that "while engaged in a ruminative process, the individual may be less sensitive to the actual contingencies that triggered the process. This will have the further effect of reducing the individual's contact with other potentially rewarding and reinforcing events and impairing the individual's ability to actively cope with the events that actually could be resolved with better contact with the situation. Such consequences may be the common denominator in depressogenic learning processes (Kanter, Busch, Weeks, & Landes, 2008; Martell et al., 2001; Nolen-Hoeksema, Wisco, & Lyubomirsky, 2008)" (pp. 76–77). It is this latter point that we aim to address in this paper.

Second, we would like to note that the model we have presented in this review is a theory outlining only one pathway through which in which rumination may lead to anhedonia (by taxing working memory and interfering with reinforcement learning processes). However, it is very much possible that other connections and feedback loops exist. For

example, rumination may not impair working memory (e.g., De Lissnyder et al., 2012; Joormann, Levens, & Gotlib, 2011; Yang et al., 2017) but deficits in working memory may also lead to an increase in ruminative thoughts (Cohen, Mor, & Henik, 2015; Hoorelbeke, Koster, Vanderhasselt, Callewaert, & Demeyer, 2015). As such, we emphasize that our model is theoretical in nature, based on extant and distinct bodies of work, rather than the results of a systematic review. It will be important for future empirical work to interrogate causality among the relationships outlined in this paper.

## 8. Implications for treatment

Having established that treatments for depression show lackluster rates of success overall and are particularly poor at restoring positive affect (Cuijpers et al., 2020), it is fundamental that we consider how a cognitive perspective of anhedonia may translate to improved treatment outcomes. While most treatments for depression have focused on low mood states rather than deficits in the appetitive symptom domain (Craske, Meuret, Ritz, Treanor, & Dour, 2016), a few treatments posit the importance of targeting anticipatory and motivational deficits related to anhedonia. For example, a common first-line therapy for individuals with MDD who exhibit distress and inhibited experiencing of positive affect is Behavioral Activation therapy (BA; Ferster, 1973; Lewinsohn, 1974; Lewinsohn & Graf, 1973). In BA, patients are instructed to use pleasant activity schedules to actively plan when, throughout a week, they can partake in rewarding activities. Thus, BA attempts to increase overt behaviors that will bring patients into contact with reinforcing environmental contingencies (Hopko, Armento, Cantu, Chambers, & Lejuez, 2003).

Meta-analytic work has shown that BA is remarkably effective for short-term treatment of anhedonia in depression (see Cuijpers, van Straten, & Warmerdam, 2007, for a review). Treatment work indicates that BA is useful insofar as it creates a structure by which depressed individuals must engage with reinforcing contingencies; the premise being, if one engages, they will experience the benefit of the reward. Nevertheless, BA is generally a short-term treatment, in part, because psychotherapists cannot sustain pleasant activity scheduling as a long-term treatment solution. Furthermore, work suggests that cognitive interference may hinder individuals with depression from actually "learning" from the reinforcements that BA brings individuals into contact with, such that behavior can be sustainably modified in the long-term (Martell, Dimidjian, & Herman-Dunn, 2013, pp. 129–148).

As cognitive deficits and biases have been shown to play a key role in depressive symptomatology, prior research has looked to trainings targeting these domains. Unfortunately, cognitive bias modifications (e.g., attention bias modification, interpretation bias modification, approach/avoidance training) have shown mixed results for depressed individuals, with meta-analytic work showing small improvements with low reliability (e.g., Fodor et al., 2020). Similarly, treatments focused on training cognitive deficits (e.g., cognitive control, working memory, motor speed, verbal fluency, etc.) through "drill-and-practice" methods have also proven largely unsuccessful at treating depressive symptoms long term (e.g., Legemaat et al., 2021). Despite the clear cognitive impairments in depression, these trainings have not been efficacious at treating depression, in part because the field is still not clear what the exact mechanisms are that maintain these impairments in depression, making them extremely difficult to target.

Taylor, Lyubomirsky, and Stein (2017) recently developed and piloted another therapy called "positive activity intervention," which was also designed to specifically target the restoration of positive affect in those with depression and anxiety. This therapy entailed a 10-session protocol of scheduled positive activity interventions to improve positive thinking, emotions and behaviors, such as practicing gratitude and acts of kindness. Though the treatment was shown to be effective within the authors' pilot study, this small cohort of 29 participants are the only reported data using this treatment, and it is not widely accessible.



Additionally, we raise similar concerns with this treatment as with behavioral activation, as this treatment does not address potential issues of cognitive interference.

Another new behavioral therapy that has been geared towards both increasing positive affect while decreasing negative affect is the Augmented Depression Treatment (ADepT), developed by Dunn et al. (2019). ADepT is characterized as a “solution-focused, cognitively augmented, behavioural activation individual therapy approach” (Dunn, Widnall, Reed, Owens, et al., 2019, pp. 2) that consists of 15 “acute treatment sessions” and up to 5 “booster” sessions. Throughout these sessions, clients are encouraged to identify goals consistent with their values and behaviorally activate towards those goals, while simultaneously identifying and “acting opposite to” negative cognitions such as rumination, avoidance thoughts, self-criticism, dampening, etc. As such, positive and negative affect are targeted in tandem to both decrease depressive symptoms and increase wellbeing. Encouragingly, when comparing the effect sizes from a pilot trial of ADepT to effect sizes in other datasets that have implemented CBT and BA, ADepT demonstrated superior outcomes in treating anhedonia (Dunn et al., 2019; Dunn, Widnall, Reed, Owens, et al., 2019). That said, RCTs are needed to replicate this finding on a larger scale.

Lastly, a similarly promising avenue for treatment that has arisen at the intersection of cognition and anhedonia is a newly developed treatment by Craske et al. (2019), Positive Affect Treatment (PAT). PAT is a behavioral and cognitive 15-week treatment that was developed specifically in light of affective neuroscience research (see Craske et al., 2016) and reinforcement learning theories, showing that three main aspects of the reward system are most implicated in anhedonia: 1) the anticipation and motivation for reward, 2) the hedonic impact of reward, and—perhaps most relevantly—3) the ability to learn instrumentally from rewards and update predictions about future rewards based on past experiences. To target these aspects, the treatment includes several sessions of three different “trainings”: behavioral training, which extends BA by requiring individuals to use recounting exercises within session to train the optimization of positive emotions; cognitive training, including exercises based on attending to the positive and identify aspects of behavior and experience that contribute to positive affect; and a compassion training, focused on cultivating positives in one’s life. Though PAT, like ADepT, is a new treatment and is not yet widely practiced, we view this as a promising solution to many of the barriers those with MDD face to restoring positive affect.

By focusing on and integrating a cognitive perspective of anhedonia into our models of treatment, we may come closer to addressing the root cause of positive affect deficits in disorders such as MDD. Without addressing rumination, for example, we expect the benefits of behavioral activation for a depressed individual to be short lived, as the cognitive interference of perseverative thinking may disrupt the ability to learn from and update the values of rewards. As such, our perspective calls for a more holistic and effective integration of cognitive and behavioral treatments—treatments that focus specifically on the presence of rumination and cognition during the consumption of rewards, and how this may affect the process of learning and subsequent anticipation of reward from future pleasurable stimuli.

## 9. Conclusion

In sum, our work highlights an increasing need to examine how cognitive processes that are hallmark to depression—particularly, the process of rumination—may contribute to and maintain anhedonia. We suggest that rumination’s relation to working memory and executive function plays an integral role in the ability to update and maintain information, interfering with the ability to produce flexible behavior and learn from reinforcements in one’s environment. Specifically, we hypothesize that when rumination, depressed persons exhibit a decreased ability to inhibit negative irrelevant information making its way into working memory and have difficulty switching between mental

sets (i.e., away from the negative information) and updating the content of working memory in order to complete necessary goals. As such, the process of rumination taxes core executive functions and working memory (by way of shifting, inhibition, and updating), therefore interfering with one’s ability to learn from rewarding cues in the environment, contributing to deficits in anticipatory pleasure as seen in depressed individuals. We propose that the burgeoning field of computational psychiatry can provide important insight into the relations among rumination, working memory, and reinforcement learning, and allow for more theoretically precise clinical frameworks. We are optimistic that such research findings will continue to lead to improved treatments for individuals experiencing anhedonia, for whom standard-of-care treatments have shown immense difficulty treating.

### Declaration of Competing Interest

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### Data availability

No data was used for the research described in the article.

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