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Diagnostic and dimensional evaluation of implicit reward learning in social anxiety disorder and major depression

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Abstract

Objective: Increasing evidence supports the presence of an anhedonic endophenotype in major depressive disorder (MDD), characterized by impairments in various components of reward processing, particularly incentive motivation, effort-based decision making, and reward learning. In addition to its prominent role in MDD, reward processing dysregulation has been proposed as a transdiagnostic risk and/or maintenance factor for a range of other forms of psychopathology. Individuals with social anxiety disorder (SAD)—a condition that frequently co-occurs with MDD—demonstrate low trait positive affectivity and altered processing of rewards and positively valenced information. However, no studies to date have directly tested reward learning—the ability to modulate behavior in response to rewards—in this population.

Materials and Methods: The current study evaluated reward learning in MDD, SAD, and healthy control subjects ($N = 90$) using a well-validated signal detection task. Given increasing data supporting transdiagnostic features of psychopathology, we also evaluated associations between anhedonia and task performance transdiagnostically in the patient sample.

Results: Contrary to expectations, results indicated no significant group differences in response bias in the full sample, suggesting no diagnostic differences in reward learning. However, dimensional analyses revealed that higher self-reported anhedonia (but not general distress or anxious arousal) was associated with worse reward learning in both the MDD and SAD groups explaining about 11% of the variance.

Conclusion: Deficits in implicit reward learning are associated with anhedonia but not necessarily with major depressive disorder as a diagnosis, which supports the use of transdiagnostic approaches to understanding psychopathology.

KEYWORDS

anxiety, assessment/diagnosis, cognition, depression, social anxiety disorder

1 | INTRODUCTION

Major depressive disorder (MDD) and social anxiety disorder (SAD) are frequently comorbid (Adams, Balbuena, Meng, & Asmundson, 2016) and are associated with marked impairment across functional domains, both separately and when co-occurring (Aderka et al., 2012; Judd et al., 2000). Although there are treatments with demonstrated efficacy (Cuijpers, Cristea, Karyotaki, Reijnders, & Huibers, 2016; Stein &

Stein, 2008), many individuals receiving first-line treatment do not experience symptom remission, and up to two thirds experience a relapse over time (DeRubeis, Siegle, & Hollon, 2008; Stein & Stein, 2008). One limitation of existing intervention approaches may be an overemphasis on tailoring treatment to discrete DSM-5 diagnostic categories. Given increasing evidence regarding shared phenotypes across traditional diagnostic boundaries (Insel et al., 2010), as well as heterogeneity within traditional DSM-5 categories (Casey et al., 2013), shifting toward transdiagnostic conceptualizations and targets may improve treatment outcomes.

A growing body of literature supports anhedonia and alterations in reward processing as a promising endophenotype for depression (Admon & Pizzagalli, 2015; Hasler, Drevets, Manji, & Charney, 2004; Whitton, Treadway, & Pizzagalli, 2015), although the ways in which anhedonia may relate to differing aspects of reward processing remain unclear (Höflich, Michenthaler, Kasper, & Lanzenberger, 2019; Kaya & McCabe, 2019). Generally, reward processing is now recognized as comprised of several interrelated, but distinct components, including anticipation and motivation to approach rewards (i.e., "wanting"), consumption of rewards (i.e., "liking"), and learning to alter future behavior following receipt of rewards (i.e., "learning"; Berridge, Robinson, & Aldridge, 2009). Research exploring reward processing in MDD suggests deficits in motivation to approach rewards and reward learning, whereas research probing consummatory pleasure in MDD suggests that this may remain intact (Admon & Pizzagalli, 2015). Accordingly, some researchers have hypothesized that anhedonia may be most proximally related to "wanting" and "learning" from rewards (Craske, Meuret, Ritz, Treanor, & Dour, 2016). However, research probing reward circuitry that may relate to anhedonia remains limited in scope (Höflich et al., 2019).

One task that is commonly used to investigate alterations in reward learning in MDD is the probabilistic reward task (PRT), which measures an individual's ability to modulate behavior in response to reinforcement (Pizzagalli, Jahn, & O'Shea, 2005). Using a signal detection-based framework, individuals are asked to distinguish between two perceptually similar stimuli and are provided with feedback on their choice for a subset of trials. Unbeknownst to the participants, correct identification of one of the stimuli (the "RICH" stimulus) is reinforced more frequently than correct identification of the other (the "LEAN" stimulus). Participants with intact reward processing demonstrate a response bias toward selecting the more frequently reinforced "RICH" stimulus (Pizzagalli et al., 2005; Tripp & Alsop, 1999). Individuals currently diagnosed with, at risk for, and remitted from MDD fail to develop a response bias on the task (Bogdan & Pizzagalli, 2006; Fletcher et al., 2015; Luking, Neiman, Luby, & Barch, 2017; Pechtel, Dutra, Goetz, & Pizzagalli, 2013; Pizzagalli, Iosifescu, Hallett, Ratner, & Fava, 2008; Vrieze et al., 2013). Importantly however, the magnitude of this effect is more pronounced for those individuals endorsing high levels of anhedonia (Fletcher et al., 2015; Vrieze et al., 2013). Follow-up work exploring the neurobiological contributions to PRT task performance implicates phasic dopamine signaling in successful reward learning on the PRT (Pizzagalli, Evins et al., 2008). Furthermore, neuroimaging studies using the PRT and related reward tasks have reported hypoactivation in neural regions that process reward, including the ventral striatum, anterior cingulate, and nucleus accumbens (Hall, Milne, & MacQueen, 2014; Kaiser et al., 2018; Wacker, Dillon, & Pizzagalli, 2009). A meta-analysis of past work on the PRT indicated that both reward sensitivity (conceptualized as gauging both aspects of consummatory pleasure, or liking, and motivation to approach rewards) and learning rate (the influence of prior rewards on future choices) influence task performance (Huys, Pizzagalli, Bogdan, & Dayan, 2013). Overall, research using the PRT has promoted advances in the field's understanding of disturbed reward processing in depression and has informed initial treatment development efforts (Craske et al., 2016).

Traditionally, anxiety disorders have been characterized by alterations in negative valence systems, with a relative neglect of the positive valence systems (Aupperle & Paulus, 2010; Insel et al., 2010). However, considering findings suggesting low trait positive affectivity in SAD (Kashdan, 2007; Kashdan, Weeks, & Savostyanova, 2011), and studies documenting biases in the processing of positive stimuli (Frewen, Dozois, Joanisse, & Neufeld, 2008; Taylor, Bomyea, & Amir, 2010), researchers have begun to explore whether anxiety may also be characterized by abnormal reward responses. Results from investigations probing reward processing in anxious samples have indeed indicated altered neural responses to social and monetary rewards (Bar-Haim et al., 2009; Forbes et al., 2006; Guyer et al., 2006; Richey et al., 2014; Silk, Davis, McMakin, Dahl, & Forbes, 2012). Furthermore, data support abnormal activation of reward-related brain regions (e.g., ventral striatum) during anticipation of social tasks and alterations in dopamine function in SAD (Mathew, Coplan, & Gorman, 2001; Schneier et al., 2008).

There are several limitations of existing work exploring reward processing in anxiety disorders. First, studies have varied considerably in the population studied (e.g., behaviorally inhibited children vs. full-threshold anxiety disorders), types of reinforcement used (monetary vs. social rewards), and the specific tasks employed. Considering this heterogeneity in study designs, it is unsurprising that the nature of observed aberrations have also been inconsistent (Bar-Haim et al., 2009; Forbes et al., 2006). Second, researchers have primarily focused on reward sensitivity, rather than reward learning, despite theoretical models highlighting the relevance of this construct (Richey et al., 2019; Schriber & Guyer, 2016). Specifically, recent developmental models of SAD propose that neurobiological processes that influence reward sensitivity and reward learning may play a role in symptoms that dynamically change across adolescence. These models first propose that early hypersensitivity to salient social cues (both rewarding and aversive) and enhancement in neurobiological processes implicated in social learning interact with the experience of repeated negative learning experiences in the social domain during adolescence (e.g., parental criticism and peer victimization). Next, repeated learning regarding the futility of coping may paradoxically result in later social anhedonia, characterized by hyposensitivity to rewards and decreased learning (Richey et al., 2019). Notably, existing literature in SAD that informs these models has

focused on reward sensitivity, rather than reward learning. Thus, there is a need for research to clarify past findings through probing differing components of reward processing in SAD.

Consistent evidence suggests that deficits in reward processing related to anhedonia are present across diagnostic categories (Balodis & Potenza, 2015; Gold, Waltz, Prentice, Morris, & Heerey, 2008; Husain & Roiser, 2018; Whitton et al., 2015). There is also significant heterogeneity in reward processing *within* diagnostic categories. For instance, anhedonia is only estimated to be present in up to 50% of individuals with MDD (Pelizza & Ferrari, 2009). Shared features across diagnostic categories and heterogeneity within DSM-5 diagnoses have resulted in an increasing emphasis on a transdiagnostic, mechanistic understanding of psychopathology, reflected in efforts such as NIMH's Research Domain Criteria (Insel et al., 2010). While the possibility of an anhedonic endophenotype has been better elucidated in depression (Pizzagalli, 2014; Treadway & Zald, 2011), this possibility has been not been rigorously explored in SAD, in other anxiety disorders, nor in combined MDD and SAD clinical samples. Accordingly, examining aberrations in reward-related processing in transdiagnostic anhedonic samples will aid in reconciling inconsistencies in past literature and result in the formulation of more targeted interventions.

The purpose of the current study was to use the PRT to test diagnostic group differences in reward learning in unmedicated individuals with MDD, SAD, and healthy control (HC) subjects. Because the PRT has been extensively tested in MDD samples, we planned to extend this literature by considering PRT performance in SAD, using the MDD group and HCs as comparison groups. We also explored a transdiagnostic perspective in the patient sample by evaluating associations between performance on the PRT and anhedonia across both diagnostic categories. The first aim was to examine differences in PRT reward learning (defined as an increase in response bias across task blocks) between diagnostic groups. We hypothesized that individuals with MDD and SAD would both independently demonstrate decreased reward learning when compared with HC participants. Consistent with a transdiagnostic approach, the second aim of the current study was to evaluate links between PRT reward learning and self-reported symptoms of anhedonia in the two patient groups. We predicted that, in the combined SAD and MDD group, greater self-reported anhedonia would be associated with decreased reward learning. Finally, given that prior work has supported the validity of the PRT in specifically probing reward processes, we expected that anhedonic symptoms would be uniquely related to PRT indicators *above and beyond* negative valence symptoms (e.g., anxiety and depressed mood).

2 | MATERIALS AND METHODS

2.1 | Participants

Participants in the current study ($N = 90$) included individuals with a principal diagnosis.[1] For SAD ($n = 34$), individuals with a principal diagnosis of MDD ($n = 33$), and HC participants ($n = 23$). Participants were recruited as part of two different treatment-related studies for anxiety and depressive disorders. Recruitment sources included clinical referrals and announcements posted in community settings or online. Inclusion criteria for the SAD group were a current principal diagnosis of SAD as defined by the SCID-I (First, Spitzer, Gibbon, & Williams, 2002) and clinician-administered Liebowitz Social Anxiety Scale (Liebowitz, 1987) score ≥50.[2] Inclusion criteria for the MDD group were a current principal diagnosis of major depressive disorder using the MINI International Neuropsychiatric Interview for DSM-5 (Sheehan et al., 1997) and a score ≥10 on the Patient Health Questionnaire-9 (See the Supporting Information for full exclusion criteria for the patient groups). Notably, a significant portion of individuals ($n = 17$) in the SAD and MDD groups currently met criteria for MDD and SAD, respectively (SAD group $n = 9$; MDD group $n = 8$).[3] For these individuals, group was determined based on the individual's principal diagnosis as determined by current impairment and distress, assessed by both participant report and interviewer judgement.

HC participants completed an initial screen verifying no lifetime history of psychopathology and endorsed no current DSM-5 diagnoses (in the past 30 days) as assessed by the MINI interview.

2.2 | Measures

2.2.1 | MINI International Neuropsychiatric Interview, Versions 5.0[4] & 7.0

The MINI was used to confirm primary diagnoses and assess co-occurring diagnoses for patient samples; in HCs, the MINI was used to rule out subjects with current DSM-5 psychopathology. Past work has supported the validity and reliability of the MINI (Sheehan et al., 1997; Verhoeven et al., 2017). In the current study, MINIs were conducted by a PhD-level clinician, a PhD student in clinical psychology, or two postbaccalaureate research coordinators. All interviewers received training in the interview protocols and met on a regular basis with the senior author for supervision and diagnostic consensus.

2.2.2 | Beck Depression Inventory-II (Cronbach's $\alpha = .95$)

The Beck Depression Inventory-II is a widely-used, 21-item self-report measurement of depressive symptoms (Beck, Steer & Brown, 1996).

[1]We defined principal diagnosis as the diagnosis that was primary to the subject's presentation and the diagnosis they rated as more severe and/or impairing.

[2]As data on individuals with SAD were collected as part of a larger trial on computerized approach/avoidance training, the LSAS cutoff was chosen to maintain consistency with past work on cognitive bias modification in SAD (Amir & Taylor, 2012).

[3]Given the potential confounding effect of co-occurring SAD and MDD in the patient groups, we re-ran analyses excluding those individuals who met criteria for the other diagnosis, as determined by the MINI. Results remained consistent in pattern.

[4]The MINI 5.0 was based on criteria for DSM IV. However, in the current study, the criteria were rescored to reflect criteria for DSM-5 diagnoses.

2.2.3 | Liebowitz Social Anxiety Scale (Cronbach's α = .97)

The Liebowitz Social Anxiety Scale is a 24-item measurement of social anxiety symptoms (Liebowitz, 1987).

2.2.4 | Mood and Anxiety Symptoms Questionnaire—Short Form (Cronbach's αs = .87–.97)

The mood and anxiety symptoms questionnaire—short form (MASQ) is a 62-item questionnaire that gauges different facets of symptoms associated with mood and anxiety disorders. The MASQ has several subscales, including anxious arousal, anhedonic depression, general distress—anxiety, and general distress—depression (Watson, Clark et al., 1995; Watson, Weber, et al., 1995).

2.2.5 | Probabilistic Reward Task

The Probabilistic reward task has been well-validated as a measure of reward learning (Pizzagalli, 2014; Pizzagalli et al., 2005; Pizzagalli, Iosifescu et al., 2008). The task requires participants to distinguish between two perceptually similar stimuli on a cartoon face (i.e., long mouth, short mouth, or long nose, short nose) that are shown on a computer screen for 100 ms. The task has three blocks of 100 trials. Participants are given feedback on 40% of trials for each block and are told that based on their performance on the task, they are able to win money. Participants are not informed that correct responses for the RICH stimulus are reinforced three times more often than the LEAN stimulus (30:10/block; Pizzagalli et al., 2005).

2.3 | Procedures

All participants attended one in-person appointment during which they provided informed consent and completed study assessments and the PRT. Procedures were approved by the local institutional review board.

2.4 | Analytic plan

Consistent with past work using the PRT (Pechtel et al., 2013; Pizzagalli, Evins, et al., 2008; Pizzagalli, Iosifescu, et al., 2008), predefined quality control procedures were performed blind to group assignments, resulting in 81 participants that were available for the main analyses (see Supporting Information for details).

We tested whether participants who were excluded from analyses following quality control checks differed in symptom and demographic variables from those who were retained. Results indicated that participants excluded from final analyses reported higher BDI scores, $t(88) = -3.11$; $p = .003$, general distress related to depression (MASQ general distress-depression), $t(14.85) = -4.30$; $p = .001$, and anhedonia (MASQ anhedonic depression), $t(18.79) = -3.54$; $p = .002$. However, these group differences were likely because all excluded participants were in the MDD and SAD subgroups. Accordingly, when the HC participants were excluded from group comparisons, the only group difference that remained significant was on the BDI, $t(65) = -2.17$; $p = .034$, suggesting that the individuals excluded through quality checks were not higher in anhedonia than other patients in the sample.

2.4.1 | Preliminary analyses

We first tested differences in overall task performance and difficulty using reaction time, hit rate, and discriminability (i.e., overall ability to discriminate between the two facial stimuli) as indicators of task performance. Discriminability was calculated for each block using the following formula[5]:

$$\log d = \frac{1}{2}\left(\frac{\text{RICHcorrect} \times \text{LEANcorrect}}{\text{RICHincorrect} \times \text{LEANincorrect}}\right).$$

We then conducted three repeated-measured analysis of variance (ANOVAs) to test for group differences in discriminability, hit rate and RT by entering Group (HC, MDD, SAD) as the between-subjects variable, and Block (Blocks 1, 2, 3) as the within-subject variable. For the models testing hit rate and RT, Stimulus Type (RICH, LEAN) was entered as a second within-subjects factor. For results from preliminary analyses testing hit rate, RT, and discriminability, see Tables S1–S3.

2.4.2 | Aim 1[6]

We calculated response bias for each block using the following formula:

$$\log b = \frac{1}{2}\left(\frac{\text{RICHcorrect} \times \text{LEANincorrect}}{\text{RICHincorrect} \times \text{LEANcorrect}}\right).$$

We then conducted a repeated-measures ANOVA with response bias as the dependent variable and block as the within-subject variable. When the assumption of sphericity was violated, we used the Greenhouse–Geisser correction.

[5]For both the log d and log b calculations, 0.5 was added to each cell in the matrix to allow computations in cases of zeros (Hautus, 1995; Pizzagalli, Evins et al., 2008).

[6]Given baseline differences in age across groups, we re-ran all main study analyses with age as a covariate, and the pattern of results remained consistent.

2.4.3 | Aim 2

To explore transdiagnostic links between reward learning on the PRT and self-reported anhedonia in the patient sample (combined SAD and MDD group), we ran a multiple regression analysis with change in response bias across blocks of the task (defined as Block 1 response bias − block 3 response bias [ΔRB]) as the dependent variable, and the MASQ anhedonic depression Subscale as the independent variable. To test the specificity of this association (Bogdan & Pizzagalli, 2006), we entered the negative valence subscales of the MASQ (anxious arousal; general distress-anxious arousal; general distress-depression) in Step 2 of the analysis.

3 | RESULTS

3.1 | Descriptive statistics

Descriptive statistics for demographic characteristics, self-reported symptoms, and psychiatric diagnoses by group are summarized in Table 1. Findings from models exploring discriminability, hit rate, and

TABLE 1 Descriptive statistics across study groups

Variable	Group			
n(%)	SAD (*n* = 34)	MDD (*n* = 33)	HC (*n* = 23)	Group differences
Gender (Male)				$\chi(4) = 0.72$
Male	13 (38.2%)	13 (39.4%)	9 (39.1%)	
Female	20 (58.8%)	19 (57.6%)	14 (60.9%)	
Neither	1 (2.9%)	1 (3.0%)	0 (0.0%)	
Race				$\chi(10) = 11.60$
Asian	13 (38.2%)	5 (15.2%)	10 (43.5%)	
Black	2 (5.9%)	1 (3.0%)	1 (4.3%)	
White	13 (38.2%)	17 (51.5%)	9 (39.1%)	
>1 Race	4 (11.8%)	5 (15.2%)	3 (13.0%)	
Other	1 (2.9%)	4 (12.1%)	0 (0.0%)	
Unknown/decline to respond	1 (2.9%)	1 (3.0%)	0 (0.0%)	
Hispanic/Latinx	11 (32.3%)	5 (15.2%)	4 (17.4%)	$\chi(2) = 3.28$
Current DSM-5 Dx				
MDD	7 (20.6%)	33 (100.0%)	0 (0.0%)	$\chi(2) = 59.51$
Panic disorder	0 (0.0%)	2 (6.1%)	0 (0.0%)	$\chi(2) = 3.53$
Agoraphobia	5 (14.7%)	2 (6.1%)	0 (0.0%)	$\chi(2) = 4.35$
SAD	34 (100.0%)	11 (33.3%)	0 (0.0%)	$\chi(2) = 60.67$
OCD	1 (2.9%)	0 (0.0%)	0 (0.0%)	$\chi(2) = 1.67$
PTSD	2 (5.9%)	0 (0.0%)	0 (0.0%)	$\chi(2) = 3.37$
GAD	9 (26.5%)	9 (27.2%)	0 (0.0%)	$\chi(2) = 7.73^{*}$
M (SD)				
Age	22.94 (5.09)	28.15 (9.38)	23.22 (3.61)	$F(2,89) = 5.99^{a,**}$
Current symptoms				
BDI	21.09 (11.73)	28.91 (8.59)	1.48 (2.52)	$F(2,89) = 64.59^{b,**}$
LSAS	85.56 (17.28)	58.21 (26.54)	7.64 (5.21)	$F(2,88) = 103.22^{c,**}$
MASQ GDA	29.24 (8.84)	23.88 (5.82)	12.82 (2.35)	$F(2,88) = 42.85^{c,**}$
MASQ GDD	34.06 (10.88)	39.76 (8.76)	13.57 (2.13)	$F(2,88) = 66.63^{b,**}$
MASQ: AA	30.00 (10.68)	25.41 (5.49)	18.09 (1.47)	$F(2,87) = 17.68^{c,**}$
MASQ: AD	77.18 (13.01)	86.30 (10.54)	35.78 (10.90)	$F(2,88) = 138.91^{b,**}$

Abbreviations: Dx, diagnoses; GAD, generalized anxiety disorder; MDD, major depressive disorder; OCD, obsessive compulsive disorder; MASQ, mood and anxiety symptoms questionnaire—short form; PTSD, posttraumatic stress disorder; SAD, social anxiety disorder; SD, standard deviation.
[a]Tukey post-hoc comparisons indicated patterns wherein MDD > SAD = HC.
[b]Tukey post-hoc comparisons indicated patterns wherein MDD > SAD > HC.
[c]Tukey post-hoc comparisons indicated patterns wherein SAD > MDD > HC, $p < .05$.
$^{*}p < .05$.
$^{**}p < .01$.

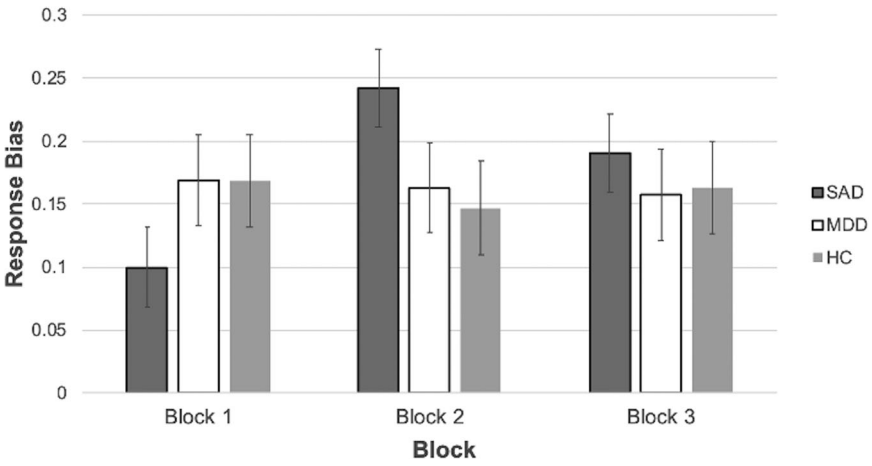

FIGURE 1 Change in response bias across PRT block, by group. HC, PRT, probabilistic reward task; MDD, major depressive disorder; SAD, social anxiety disorder

response time (see Supporting Information) generally supported the assertion that groups found the task equally challenging.

3.2 | Aim 1: Categorical analyses

To test group differences in response bias, we conducted a 3 × 3 (Group × Block) repeated-measures ANOVA (see Figure 1 and Table 2). The model indicated a significant Group × Block interaction, F (3.62, 141.26) = 3.77, p = .008, η_p^2 = 0.09. Follow-up contrasts indicated that this effect was driven by group differences in response bias change from Block 1 to Block 2, F(2, 78) = 6.92, p = .002, η_p^2 = 0.15. Inspection of marginal means suggested that this was primarily driven by an increase in response bias among the SAD group from block 1 (M = 0.10, SE = 0.03) to block 2 (M = 0.24, SE = 0.03) that was absent in the MDD and, unexpectedly, HC groups. Simple effects of block indicated no group differences within each block (ps > .05). No other main effects or interaction effects were significant.

3.3 | Aim 2: Transdiagnostic analyses

Results from the regression model (see Table 3), F (1,60) = 8.49, p = .005, Adjusted R^2 = .11, indicated the hypothesized significant association between anhedonic symptoms and reward learning across blocks, unstandardized B = −0.01; SE = 0.002, t = −2.64, p = .011. Addition of the three other MASQ subscales did not result in a significant improvement in the model, ΔF(3,51) = 0.18, p = .913, Adjusted R^2 = .05. There were no significant associations between negative valence MASQ subscales and ΔRB (ps > .05). After entering all MASQ subscales into the model, anhedonic symptoms remained significantly negatively associated with reward learning, unstandardized B = −0.01; SE = 0.002, t = −2.50, p = .016.

TABLE 2 Response bias across PRT blocks, by group

Within-subjects[a]	*df*	*F*	*p*-value	η_p^2
Block	1.81, 141.26	1.86	.164	0.023
Block × Group	3.52, 141.26	3.77	.008	0.088
Between-subjects				
Group	2, 78	0.13	.881	0.003

Abbreviation: PRT, probablistic reward task.
[a]Due to the violation of the assumption of sphericity (p < .05), we report the Greenhouse–Geisser correction.

4 | DISCUSSION

In the current study, we compared the performance of individuals with SAD, MDD, and HC subjects on a well-validated reward learning task. Contrary to expectations, no group differences emerged on task parameters. However, when taking a transdiagnostic approach to analyses in the patient subsample, significant negative associations emerged between anhedonic symptoms and reward learning. Findings are broadly consistent with a growing literature supporting a transdiagnostic, anhedonic phenotype and highlight the possibility of using characteristics such as anhedonia to better characterize heterogeneity within DSM-5 mood and anxiety diagnostic categories.

Regarding Aim 1, results identified some group differences in the development of response bias across the PRT, but in an unexpected manner. Specifically, we had hypothesized that both the SAD and MDD groups would display blunted response bias across task blocks. Instead, the only significant group difference was that the SAD group demonstrated a significant *increase* in response bias from Block 1 to Block 2. Across the remainder of the blocks, no group differences in response bias emerged. Additionally, contrary to several previous studies (Pizzagalli et al., 2005; Pizzagalli, Iosifescu et al., 2008), the current HC sample failed to show the expected increase in response bias across blocks.

However, regarding Aim 2, transdiagnostic analyses suggested that as expected, in the patient subsample, greater self-reported anhedonic symptoms were associated with reduced reward learning. Highlighting

TABLE 3 Regression analysis exploring associations between self-reported symptoms and reward learning in patient subsample

Variable	Adj. R^2	*B*	*SE*	*t*	*p*	*r*
Step 1: $F(1,54) = 6.96$, $p = .011$	.10					
Anhedonic symptoms		−0.01	.002	−2.64	.011	−.34
Step 2: $\Delta F(3,51) = 0.18$, $p = .913$	.05					
Anhedonic symptoms		−0.01	.002	−2.50	.016	−.35
Anxious arousal		.00	.004	−0.04	.971	.02
General distress-depression		.002	.003	0.47	.641	−.11
General distress-anxiety		.001	.01	0.26	.794	−.02

Note: B = unstandardized beta; r = zero order correlation with outcome variable.
Abbreviation: SE = standard error.

the specificity of these findings, other MASQ subscales—Anxious Arousal, General Distress-Depression, and General Distress-Anxiety—did not relate to reward learning, and the relationship between anhedonic symptoms and reward learning remained significant when entering the other symptoms in the regression model.

There are several explanations for our pattern of findings. Regarding lack of differences in reward learning in the SAD group, altered reward processing could be specific to disorder-relevant stimuli. Several recent studies in SAD have suggested that altered neural and behavioral responses to rewards may be specific to social rewards, rather than more general rewards (Richey et al., 2014; Richey et al., 2017). Alternatively, our lack of group-based effects could be explained by heterogeneity within both the MDD and SAD diagnostic categories, an interpretation which is also consistent with our Aim 2 findings. Notably, while the majority of PRT findings in MDD samples support blunted response bias in this group (Huys et al., 2013; Pechtel et al., 2013; Pizzagalli, Iosifescu et al., 2008; Vrieze et al., 2013), data consistently suggest that the effect is strongest among those patients reporting elevations in anhedonic symptoms (Fletcher et al., 2015; Pizzagalli, Iosifescu et al., 2008; Vrieze et al., 2013). Furthermore, existing research supports the presence of multiple phenotypes in depression, with some tentative data suggesting that reward processing dysfunction may be heterogeneous within depressed samples (Foti, Carlson, Sauder, & Proudfit, 2014). Thus, it could be the case that our MDD group was heterogeneous in its composition, obscuring response bias effects in the high anhedonia group.

It may also be the case that there was significant heterogeneity within the SAD group. Initial research exploring reward processing in SAD and anxiety more generally has been mixed, with work indicating both hyper- and hypo-responsivity to different types of reward (Anderson, Veed, Inderbitzen-Nolan, & Hansen, 2010; Bar-Haim et al., 2009; Cremers, Veer, Spinhoven, Rombouts, & Roelofs, 2015; Forbes et al., 2006). Recent theoretical perspectives drawing upon neurobiological and developmental research have outlined dynamic pathways to the development of social anhedonia and altered reward processing in SAD but note that this pathway likely represents only one SAD phenotype (Richey et al., 2019). More recently, Tung and Brown (2020) explored subgroups of patients with SAD and found that two distinct SAD risk profiles emerged—one characterized by low positive temperament and elevated negative temperament, and one characterized by normative positive temperament and elevated negative temperament (Tung & Brown, 2020). Individuals with lower positive temperament also indicated greater severity of SAD symptoms and were more likely to be men and diagnosed with MDD (Tung & Brown, 2020). Although lack of consistency in methodology precludes direct comparisons, it is possible that both classes of SAD risk were represented within our sample or that overrepresentation of the class characterized by normative positive temperament obscured any group findings. It is also worth noting that our SAD sample was primarily comprised of women, who Tung and Brown (2020) found were more likely to present with a SAD subtype characterized by normative positive affect. Altogether, given that there are likely different phenotypes within existing diagnostic categories characterized by anhedonia, future research must explore this possibility in samples that are adequately powered to undertake subgroup analyses. Further, these findings underscore the potential importance of taking a dimensional approach to understanding anhedonia across diagnostic categories.

Regardless of the reason for the lack of group-based effects on the PRT, regression analyses in the patient subsample highlight consistent links between task performance and anhedonic symptoms. These results provide support for a modest relationship between PRT performance and self-reported anhedonia. As a prior meta-analysis suggested that both reward sensitivity and learning rate contribute to performance on the task (Huys et al., 2013), the specific aspects of reward processing that may be gauged by PRT performance remains an important topic for future research. Overall, while research has begun to explore links between subcomponents of reward processing and anhedonia, neurobiological processes through which anhedonia relates to subcomponents of reward remain undercharacterized (Kaya & McCabe, 2019; Treadway & Zald, 2011). Our findings contribute to this body of work by suggesting that a behavioral indicator of reward learning relates consistently to self-reported anhedonia in a transdiagnostic sample. Though tentative, results provide initial support for clinical approaches that prioritize transdiagnostic mechanisms of psychopathology and flexible formats that can be modified for a specific clinical presentation (e.g., Unified Protocol; Farchione et al., 2012), as well as recent treatment

development efforts focused on reward processing (Craske et al., 2016, 2019; Taylor, Lyubomirsky, & Stein, 2017).

4.1 | Limitations

The current investigation possesses several noteworthy limitations. First, sample sizes for each group were small; therefore, future investigations must replicate our findings in larger samples. Heterogeneity within diagnostic groups and overlap in diagnostic categories further limit our ability to test alternative explanations for null group results. Accordingly, the need to replicate our findings and directly explore heterogeneity in large samples of individuals with SAD and MDD is an important next step.

Second, a portion of the SAD and MDD groups endorsed symptomatology of the other condition. While this likely increases sample generalizability due to common co-occurrence of these conditions (Adams et al., 2016), it does introduce a confound into our group-based analyses. However, the patterns of results remained consistent when excluding these individuals from analysis, increasing confidence in our findings. Finally, results indicated that individuals who did not pass QC thresholds for the PRT in our MDD condition endorsed significantly higher BDI scores, suggesting that individuals with greater depression may have been excluded and contributed to our lack of group-based findings.

5 | CONCLUSIONS

In the present study, we investigated reward processing in individuals with SAD, individuals with MDD, and HC individuals. Results indicated no group differences in reward learning; however, in the patient subsample, heightened self-reported anhedonia uniquely predicted poorer reward learning, over and above other non-anhedonic symptoms. Results provide further support for assertions that anhedonia and related reward disturbance may be a salient characteristic across a range of clinical presentations and represents a potential target in transdiagnostic treatments.

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CONFLICT OF INTERESTS

Charles T. Taylor declares that in the past 3 years he has been a paid consultant for Homewood Health, and receives payment for editorial work for *UpToDate*. Murray B. Stein declares that in the past 3 years he has been a paid consultant for Actelion, Aptinyx, Bionomics, Janssen, Neurocrine, and Pfizer, and receives payment for editorial work for *UpToDate* and the journals *Biological Psychiatry* and *Depression and Anxiety*. Dr. Paulus is an advisor to Spring Care, Inc., a behavioral health startup, he has received royalties for an article about methamphetamine in *UpToDate*. Over the past 3 years, Dr. Pizzagalli has received funding from NIMH, Brain and Behavior Research Foundation, the Dana Foundation, and Millennium Pharmaceuticals; consulting fees from Akili Interactive Labs, BlackThorn Therapeutics, Boehreinger Ingelheim, Compass Pathway, Posit Science, Otsuka Pharmaceuticals, and Takeda Pharmaceuticals; one honorarium from Alkermes; stock options from BlackThorn Therapeutics. Dr. Pizzagalli has a financial interest in BlackThorn Therapeutics, which has licensed the copyright to the Probabilistic Reward Task through Harvard University. Dr. Pizzagalli's interests were reviewed and are managed by McLean Hospital and Partners HealthCare in accordance with their conflict of interest policies. All other authors report no biomedical financial interests or potential conflicts of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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