Reward-Based Reinforcement Learning in Altered Among Individuals with a History of Major Depressive Disorder and Psychomotor Retardation Symptoms

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Abstract

Background: Reward-based reinforcement learning impairments are common in major depressive disorder, but it is unclear which reward-based reinforcement learning is disrupted in remitted major depression (rMDD). As the neurobiological substrates that implement reward-based RL are also strongly implicated in psychomotor retardation (PmR), the present study sought to test whether reward-based reinforcement learning is particularly altered in rMDD individuals with past PmR.

Methods: Three groups of individuals (1) rMDD with past PmR (PmR+, N=34), rMDD without past PmR (PmR-, N=44) and healthy controls (N=90) completed a reward-based reinforcement learning task. Computational modeling was applied to test for group differences in model-derived parameters – specifically, learning rate, reward sensitivity, and value estimates.

Results: Compared to controls, rMDD PmR + exhibited lower learning rates, but not reduced reward sensitivity. Relative to rMDD PmR-, rMDD PmR + exhibited reduced value estimation early, but not later, in the reinforcement learning task. Follow-up analyses indicated that the results were not due to current psychopathology symptoms.

Conclusions: Results indicate that a history of PmR predicts altered reward-based reinforcement learning in rMDD. Abnormal reward-related reinforcement learning may reflect a scar of past depressive episodes that contained psychomotor symptoms, or a trait-like deficit that preceded these episodes.

Introduction

Major Depressive Disorder (MDD) is a highly impairing and prevalent psychiatric disorder, where at least half of individuals experience multiple depressive episode in their lifetime (Kessler & Walters, 1998; Kessler et al., 1997). Indeed, the single best predictor of future depression is past depression (Lewinsohn et al., 1989; Tram & Cole, 2006). Identifying processes that are disrupted in individuals with remitted depression could reveal key mechanisms of depression relapse.

One potentially important process through which depression develops, maintains, and recurs is via aberrant reward-based reinforcement learning (RL). RL refers to the ability to learn which actions are more likely to lead to rewarding outcomes. Reward-based RL is impaired in current MDD (Kumar et al., 2008; Pizzagalli et al., 2005; Pizzagalli et al., 2008) and has been shown to predict treatment response (Vrieze et al., 2013; Whitton et al., 2020). Specifically, individuals with current MDD often fail to exhibit a bias toward more rewarding stimuli relative to controls. Computational modeling, which is increasingly being utilized to identify specific cognitive processes that are impaired in various psychopathologies (Adams et al., 2016; Huys et al., 2016), indicates that reward-based RL deficits are, in part, related to reduced reward sensitivity (for a review, see Chen et al., 2015; Huys et al., 2013).

In contrast with current MDD, reward-based RL has been studied to a lesser extent in remitted MDD (rMDD) and even fewer studies have applied computational modeling to reward-based RL in rMDD. Similar to current MDD, there is emerging evidence of reward-based RL deficits among individuals with rMDD (Pechtel et al. 2013; Whitton et al., 2016). Although a history of MDD itself may predict impaired reward-based RL, MDD is a very heterogenous

disorder (Merikangas et al., 1994; Østergaard et al., 2011), and a history of specific symptoms may be especially detrimental for reward-based RL in rMDD.

One depression symptom that may be particularly critical for reward-based RL is psychomotor retardation (PmR), which broadly describes reduced speed and amplitude in gross and fine motor activity (Schrijvers et al., 2009; Sobin & Sackeim, 1997). Although PmR has been understudied in MDD, studies of PmR are increasingly recognizing its clinical significance (Shankman et al., 2020; Walther et al., 2019). Motor symptoms not only correspond to more severe MDD (Calugi et al., 2011; Ulbricht et al., 2016) and poorer treatment response (Janzing et al., 2020; Taylor et al., 2006), but also to greater MDD recurrence (Gorwood et al., 2014). Thus, PmR may be a critical factor that relates to the development and/or recurrence of MDD.

PmR may be critical for reward-based RL due to overlapping neurocircuitry. Broadly, reward-based RL is implemented in the basal ganglia, including the dorsal striatum and ventral striatum, and regions of the prefrontal cortex (PFC; Galvan et al., 2005; O'Doherty, 2011; Pujara et al., 2016). Notably, PmR in MDD is also related to structural and functional alterations of many of these regions and related circuits (Bracht et al., 2012; Martinot et al., 2001; Naismith et al., 2002). Additionally, reward-based RL is supported by dopaminergic functioning (Samson et al., 2010), which is impaired in individuals with PmR (Martinot et al., 2001). Taken together, a history of PmR, which may be indicative of trait-like corticostriatal and/or dopaminergic dysfunction, may relate to key deficits in rMDD, including reward-based RL.

The primary goal of the present study was to identify whether reward-based RL is altered among individuals with remitted MDD with a history of PmR symptoms (rMDD PmR+) relative to (a) healthy controls and (b) rMDD without a history of PmR symptoms (rMDD PmR-). To assess reward-based RL, participants completed the Probabilistic Reward Task (PRT), and

computational modeling was applied to participants' trial-wise choices. It was hypothesized that individuals with rMDD would exhibit a reduced response bias (indicated by traditional PRT measures), as well as computational evidence of impaired reward-based RL relative to controls. Additionally, it was anticipated that differences in reward-based RL deficits would emerge for rMDD PmR+ relative to rMDD PmR-, but not rMDD PmR- relative to controls. Finally, it was expected that group differences in reward-based RL would not be accounted for by current clinical symptoms, including anhedonia.

Materials and Methods

Participants

Young adults (N=504) were recruited as part of a larger family study of transdiagnostic mechanisms of internalizing psychopathology from mental health clinics and the local Chicago, Illinois community. Primary eligibility criteria included (1) age 18-30 years old and (2) having a biological sibling age 18-30 who was also interested in participating (for full method details, see Gorka et al., 2016; Weinberg et al., 2015). Participants were oversampled during recruitment for severe internalizing psychopathology symptoms using the Depression, Anxiety, and Stress Scale (Lovibond & Lovibond, 1995; see Supplement for additional details).

Individuals were selected for the present study based on lifetime MDD and PmR symptoms using the Structured Clinical Interview for DSM-5 (SCID-5; First et al., 2015). Participants were included in one of three groups: (1) rMDD PmR+, if they met criteria for past but not current MDD and had PmR symptoms during their past major depressive episode, (2) rMDD PmR-, if they met criteria for past but not current MDD and did not have PmR symptoms during their past depressive episode, and (3) controls, if they had no lifetime history of any psychiatric disorder or PmR. Individuals who met criteria for current MDD and/or had current

PmR and/or agitation (which broadly describes increases in purposeless motor activity; Sobin & Sackeim), on the SCID-5 were excluded from the study. Participants were also excluded if they did not have viable PRT data (Pizzagalli et al., 2005; Whitton et al., 2016; see Supplement). The final sample included 34 participants in the rMDD PmR+ group, 44 in the rMDD PmR- group, and 90 in the control group (N=168).

Assessments and Measures

Clinical Diagnoses and Psychomotor Symptoms.

Diagnoses and psychomotor symptoms were assessed via the SCID-5 (see Supplement for additional details). In contrast with traditional SCID assessment procedures, interviewers assessed all symptoms of MDD for all participants, even if the cardinal symptom(s) were not endorsed. Thus, lifetime psychomotor symptoms were characterized for all participants, allowing for the exclusion of individuals who did not meet criteria for MDD but endorsed lifetime psychomotor agitation (across groups) and/or lifetime PmR in the rMDD PmR- and control groups.

Psychomotor Speed.

Condition 5 (Motor Speed) from the Delis-Kaplan Executive Function System Trail Making Test (Delis, et al., 2001) assessed current psychomotor speed. During this condition participants connect empty dots while tracing over a dotted line as quickly as possible. Reaction time was used as the dependent measure.

Inventory of Depression and Anxiety Symptoms (IDAS-II).

The IDAS-II (Watson et al., 2012) is a 99-item questionnaire that measures current symptoms of depression and anxiety. In the present study, General Depression (Well-Being items removed), Well-Being (reverse scored to capture anhedonia), Panic, and Social-Anxiety

were included as covariates of current internalizing symptoms. The observed internal consistencies were in the good-excellent range (General Depression: $\alpha=.91$, Well-Being: $\alpha=.86$, Panic: $\alpha=.80$, and Social-Anxiety: $\alpha=.86$).

Personality Inventory for DSM-5 (PID-5).

The PID-5 (Krueger et al., 2012) is a 220-item measure of pathological personality traits and has strong psychometric properties (Thomas et al., 2013; Katz et al., 2018). The PID-5's Anhedonia facet was included as a covariate to account for the trait-like tendency for individuals to experience anhedonia. The observed internal consistency was in the excellent range ($\alpha=.93$).

Probabilistic Reward Task (PRT).

The PRT (Pizzagalli et al., 2005) is a well-validated probe of reward-based RL. The task included 200 trials across two blocks. Participants are first presented with a jittered fixation cross, followed by two dots (“eyes”) and vertical line (“nose”). This is followed by either a short (10 mm) or long (11 mm) horizontal line (“mouth”; see Figure 1). Participants must select the identity of each cue by pressing a corresponding keyboard key (‘z’ or ‘/’). The cues represent a “lean” and a “rich” stimulus that are rewarded probabilistically (counterbalanced across participants), with the rich stimulus 3x more likely to deliver a reward following a correct response than the lean stimulus. During the feedback phase, participants either receive feedback indicating that they were correct and won 20 cents, or they receive no feedback.

Traditional PRT measures that were calculated included response bias and discrimination (Pizzagalli et al., 2005; Whitton et al., 2016; see Supplement for the response bias and discrimination formulas). In addition to looking at overall response bias and discrimination, differences in the two PRT measures between blocks (Block 2-Block 1) were computed for each participant (Δ response bias and Δ discrimination).

Computational Modeling of Reinforcement Learning

Several models that build on the standard Rescorla-Wagner (RW) model have been applied to the PRT task in previous research (Huys et al., 2013). These models were fit to participants' behavior, including the 'Action', 'Belief', and 'Stimulus-Action' models. All models took the following general form:

$$Q_{t+1,c} = Q_{t,c} + \alpha \times \delta_t,$$

where $Q_{t,c}$ is the *quality*, or value estimation, of cue c on trial t , α is the *learning rate* (which takes on the same value across all trials), and δ_t is the *prediction error* (PE; $\rho \times outcome_t - Q_{t,c}$) on trial t . To capture *reward-sensitivity*, parameter ρ was included in the PE term, which scales the value of the outcome, with $0 \leq \rho \leq 1$ and values closer to 1 reflecting greater reward sensitivity.

The probability of choosing key 1 ('z') or 2 ('/') was linked to participants' expectations of reward using a softmax function. Specifically, value expectations ($Q_{t,c}$) were transformed using a softmax that captures the difference between weighted (W_t) value expectations for each action (choosing the 'z' key and choosing the '/' key), with larger differences leading to an increased likelihood of selecting one action over the other:

$$p(a_t|s_t) = 1 / (1 + \exp[-(W_t(a_t, s_t) - W_t(\bar{a}_t, s_t))]),$$

where a_t reflects the selected action and $\bar{a}_t$ reflects the unselected action for the presented stimulus s_t on trial t . The weighted value estimates that are included in the softmax function for each action $W_t(a_t, s_t)$ and $W_t(\bar{a}_t, s_t)$ reflect a weighted combination of value estimates for each stimulus:

$$W_t(a_t, s_t) = \zeta \times Q_t(a_t, s_t) + (1 - \zeta) \times Q_t(a_t, \bar{s}_t),$$

where $\bar{s}_t$ reflects the stimulus that was not presented on trial t . The parameter that scales the value estimates, ζ , reflects the degree to which a learner is certain of the identity of the stimulus on screen, with $0 \leq \zeta \leq 1$ and values closer to 1 reflecting greater certainty.

For the Stimulus-Action model, $\zeta=1$ (i.e., a learner is certain which stimulus is the long versus short cue), thus nullifying the second term from the model, $(1-\zeta) \times Q_t(a_t, \bar{s}_t) = 0$. For the Action model, $\zeta=.5$ (i.e., a learner is uncertain which stimulus is the long versus short cue) and each term, $W_t(a_t, s_t)$ and $W_t(a_t, \bar{s}_t)$, is weighed equally. For the Belief model, ζ is a free parameter that is estimated for each learner, which captures individual differences in certainty about stimulus identity.

Models were fit to participants' choice behavior using maximum likelihood estimation and Akaike Information Criterion (AIC) values were used to identify the best-fitting model. Trial-wise value estimates were recovered for the best-fitting model and carried forward to group-level analyses.

Analytical Approach

Analyses examined the effect of remitted MDD with a history of PmR on reinforcement learning using linear mixed effects models. Specifically, we tested whether remitted MDD with a history of PmR symptoms (rMDD PmR+) differed from controls or rMDD without a history of PmR symptoms (rMDD PmR-) on RL using dummy coding with rMDD PmR+ as the reference group. We also examined whether behavior or modeling parameters differed for rMDD PmR- relative to controls.

First, we assessed for differences in traditional PRT behavioral measures. Using the best-fitting model, we then examined whether groups differed on model-estimated (1) learning rates, α , and (2) reward sensitivity, ρ . Because these parameters are typically correlated (Huys et al.,

2013), follow up tests examined unique effects for these parameters. Next, trial-wise value estimates were recovered for participants' choices, and we tested whether estimates varied over time across trials and/or block (trial was z-scored within each block and block was contrast coded as Block 1=-1, Block 2=1) as a function of group. Age and gender were included as covariates, family was included as a random effects factor to account for sibling relatedness (i.e., familiarity), and random slopes were included for Trial and Block. Follow-up analyses examined whether the inclusion of psychopathology covariates altered the results.

Results

Sample Characteristics

As shown in Table 1, there were no group differences in age, $F(2,165)=2.35$, $p=.098$ or self-identified gender, $\chi^2(2)=1.76$, $p=.415$, and the sample was quite diverse. Means and standard deviations for clinical measures and psychomotor speed are also reported in Table 1. Both rMDD groups endorsed more current symptoms of general depression, social anxiety, panic, and anhedonia than controls, but critically, the two rMDD groups did not differ on any of the clinical measures. Psychomotor speed did not significantly differ across groups $F(2,165)=2.85$, $p=.061$. As shown in Table 2, the only group difference on medication usage was that relative to controls, both rMDD groups had higher rates of current SSRI use, but SSRI rates did not differ between the rMDD groups, $F(2,165)=5.22$, $p=.006$.

Traditional PRT Measures

Means and standard deviations for traditional PRT measures are provided in Table 3. No significant group differences emerged for any of the traditional measures.

Model Fit

As shown in Figure 2A, the Action model provided the best fit based on AIC across all participants (log-likelihood values are provided in the Supplement). For all groups, the Action model provided a better fit than the Belief and Stimulus-Action models (see Figure 2B). Not only did the Action model provide a better fit relative to other models for all groups, model fit did not significantly differ between groups, $F(2,165)=2.38, p=.096$.

Learning Rate and Reward Sensitivity

For the learning rate (α), there was a significant effect of Group, $F(2,163)=4.19, p=.017$, which was driven by higher learning rates for controls relative to the rMDD PmR+ group, $t(163)=2.85, B=.27, p=.005$. By contrast, learning rates did not differ for rMDD PmR- relative to rMDD PmR+, $t(163)=1.43, B=.14, p=.154$. Additionally, learning rates did not differ for the rMDD PmR- group relative to controls, $t(163)=-1.40, B=-.11, p=.164$. Follow-up tests revealed that the difference in learning rates between rMDD PmR+ and controls remained after including psychopathology covariates (see Table 4). For reward sensitivity (ρ) there was no main effect of Group, $F(2,163)=1.45, p=.238$.

To identify whether the difference in learning rates between the rMDD PmR+ and control groups remained after controlling for reward sensitivity (which was significantly correlated with learning rate, $r[166]=-0.34$), the reward sensitivity parameter was included as a covariate. The difference in learning rates between controls and the rMDD PmR+ group remained significant after controlling for reward sensitivity, $B=.22, p=.023$.

Trial-wise Value Estimates for Participants' Choices

There was a significant effect of Group, $F(2, 33,586)=3.02, p=.049$, which was driven by higher value estimates for controls compared to the rMDD PmR+ group across the task, $t(33,586)=2.35, B=.16, p=.019$ (see Figure 3). Overall, value estimates did not differ for rMDD

PmR- relative to rMDD PmR+, and the rMDD PmR- group did not exhibit lower value estimates relative to controls across the task. A significant Trial x Block x Group interaction emerged, $F(2, 33,586)=3.99, p=.019$, which was evident for both rMDD PmR+ vs. controls, $t(33,586)=4.74, p=.009$, and rMDD PmR+ vs. rMDD PmR-, $t(33,586)=3.36, p=.035$.

Within the first block of the task, rMDD PmR+ differed from controls during both early (z-trials <-1; $t(3,523)=3.12, B=.22, p=.002$) and later trials (z-trials>1; $t(3,523)=2.13, B=.15, p=.033$). By contrast, the rMDD PmR+ group only differed from the rMDD PmR- group during early, $t(3,523)=1.98, B=.14, p=.048$, but not later trials, $t(3,523)=1.50, B=.11, p=.135$.

Within the second block of the task, the effect of Group for rMDD PmR+ vs. controls approached significance during early trials, $t(3,523)=1.68, B=.12, p=.094$, and reached significance during later trials, $t(3,523)=2.19, B=.16, p=.029$. Value estimates did not differ in the second block for rMDD PmR+ vs. rMDD PmR- during early or later trials, $t(3,523)=0.90, B=.06, p=.368$ and $t(3,523)=1.22, B=.09, p=.221$. There were no other group-related effects. For controls vs. rMDD PmR-, there was not a significant Trial x Block x Group interaction, $t(33,586)=-0.98, p=.328$.

Follow-up tests revealed that results remained after including psychopathology covariates (see Table 5).

Discussion

Psychomotor retardation symptom history predicted altered reward-based RL in individuals with rMDD. Whereas group differences did not reach significance for traditional PRT measures, several differences were identified using more specific parameters identified with computational modeling. These findings add to a growing literature of atypical reward-based RL

in individuals with rMDD (Pechtel et al. 2013; Whitton et al., 2016), and, furthermore, highlight a potentially important role of psychomotor retardation in reward-based RL in MDD.

Despite comparable computational model fit across groups, which indicates that participants completed the task using similar learning strategies, rMDD individuals with a history of PmR exhibited lower learning rates compared to controls. Learning rates reflect how much individuals revise their expectations based on the error in their predictions. By contrast, reward sensitivity, which reflects how much individuals weigh reward-related outcomes in their future expectations, did not differ between the rMDD PmR+ and control groups. Value estimates for participants' choices across the task provided a similar result as the learning rates, as individuals with rMDD with a history of PmR generally exhibited lower value estimates than controls throughout the task. Importantly, all results did not change after controlling for current symptoms of psychopathology, suggesting that effects were not due to residual current symptoms.

Although neither learning rates nor reward sensitivity differed between the rMDD groups, there was evidence of initial differences in value estimation during early, but not later, trials of the task. Thus, individuals with rMDD with a history of PmR exhibited difficulties with initially learning action-outcome pairings relative to rMDD without a history of PmR, but not later in the task. In contrast rMDD individuals without PmR did not differ from controls on any of the traditional or computational task measures, including learning rates and value estimates. Thus, only rMDD individuals with PmR, rather than rMDD broadly, exhibited evidence of altered reward-based RL.

Results are in line with previous research that identified reward-based RL is disrupted in individuals with a history of MDD, even after accounting for symptoms of perceived stress and

anhedonia (Petchel et al., 2013), the latter of which correlates with the reward sensitivity parameter (Huys et al., 2013; Whitton et al., 2020). Although current MDD and anhedonia have related to reduced sensitivity to rewards in previous work (Huys et al., 2013), a history of MDD has been found to only weakly relate to atypical model-derived reward sensitivity (Huys et al., 2013). That reward sensitivity is not the primary contributor to reward-based RL deficits in rMDD is further supported by present results. While this is not the first study to identify a role of other reward-based RL processes in MDD (Chen et al., 2015), to our knowledge it is the first to identify a primary role of altered learning rates in reward-based RL in individuals with rMDD who have experienced PmR.

Despite not having clinically elevated *current* PmR symptoms or significantly slower psychomotor speed, a history of PmR during a past episode of MDD predicted current alterations in reward-based RL. This suggests that difficulties in rMDD with reward-based RL are not merely the result of psychomotor slowing. As discussed above, there is evidence that corticostriatal circuitry and dopaminergic functioning that supports reward-based RL are disrupted in PmR (Bracht et al., 2012; Naismith et al., 2002). Although indirect evidence of these links, administration of a low dose dopaminergic D₂ agonist (which has antagonistic effects on behavior) has been found to disrupt learning rates, but not reward sensitivity, during reward-based RL (Huys et al., 2013; Pizzagalli et al., 2008); a pattern that emerged in our study. These findings are particularly relevant for precision medicine, which aims to identify specific prevention efforts and treatments for specific individuals (Fernandes et al., 2017). For example, computationally-derived PRT parameters could help identify which individuals may benefit from enhancing dopaminergic functioning to prevent MDD relapse.

There are several limitations that should be noted. First, psychomotor symptom history was based on a retrospective clinical assessment via the SCID. Relatedly, this study is unable to clarify whether abnormal reward-based RL processing reflects a scar of past MDD episodes that contained psychomotor symptoms or a trait-like deficit that preceded episodes. In the future, it will be important to conduct prospective studies that include fine-grained assessments of psychomotor symptoms, including laboratory-based and other instrumental measures (e.g., actigraphy, an ecologically sensitive approach to assessing movement in daily life; Shankman et al., 2020).

Conclusion

Reward-based RL is an critical cognitive process that is impaired in current and remitted depression. Among individuals with rMDD, those who have a past history of psychomotor slowing may continue to have disrupted experiential learning, which could contribute to and/or perpetuating disengagement from activities and interpersonal relationships. Targeting (a) residual PmR symptoms in individuals with a history of MDD and/or (b) reward-based RL in individuals with a history of PmR may help to prevent relapse into future depressive episodes.

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Tables

Table 1. Demographic, Cognitive, and Clinical Characteristics

	rMDD PmR+ n=34	rMDD PmR- n=44	Controls n=90
Age	22.4 (3.3)	23.3 (3.6)	22.0 (3.2)
Gender	77% female	68% female	64% female
Ethnicity			
White	38%	63%	38%
Black/African American	24%	8%	7%
Hispanic	32%	17%	19%
Asian	6%	6%	26%
Middle Eastern	-	-	8%
Mixed Race	-	6%	2%
IDAS Subscales			
Depression (Well-Being items removed) [†]	36.2 (11.3)	35.1 (12.0)	25.0 (7.0) ^{a,b}
Well-Being [†]	25.2 (5.8)	25.3 (7.4)	27.32 (6.4)
Social Anxiety [†]	11.4 (5.4)	10.4 (4.5)	7.1 (1.9) ^{a,b}
Panic [†]	11.9 (4.6)	10.3 (2.6)	9.0 (2.4) ^{a,b}
PID-5 Facets			
Anhedonia [‡]	0.73 (.51)	0.71 (.69)	0.36 (.38) ^{a,b}
Current Subthreshold Psychomotor Symptoms			
Psychomotor Retardation	n=5 (15%)	n=0	n=0
Psychomotor Agitation	n=3 (9%)	n=1 (2%)	n=0
Lifetime Subthreshold Psychomotor Symptoms			
Psychomotor Retardation	-	n=0	n=0
Psychomotor Agitation	n=0	n=9 (21%)	n=1 (1%)
Motor Speed (D-KEFS Trail Making Test, Condition 5) [§]	26.2 (12.6)	22.2 (6.5)	22.0 (8.3)

Note. IDAS=Inventory of Depression and Anxiety Symptoms; PID-5=Personality Inventory for DSM-5

[†]rMDD PmR+: n=34, rMDD PmR-: n=43, Controls: n=89

[‡]rMDD PmR+: n=34, rMDD PmR-: n=43, Controls: n=90

[§]rMDD PmR+: n=34, rMDD PmR-: n=42, Controls: n=85

^{a,b}Scores significantly differed for controls relative to the rMDD groups

Table 2. Rates of current medication use across groups.

	rMDD PmR+	rMDD PmR-	Controls
Medication			
Anxiolytics	3% (n=1)	2% (n=1)	0%
SSRI [†]	12% (n=4)	9% (n=4)	0% ^{a,b}
SNRI	0%	2% (n=1)	0%
NDRI	3% (n=1)	2% (n=1)	0%
MAOI	0%	0%	0%
Sedatives	0%	0%	0%
Hypnotics	0%	0%	0%
# of Medications	0: 88% (n=30) 1: 6% (n=2) 2: 6% (n=2)	0: 89% (n=39) 1: 9% (n=4) 2: 2% (n=1)	0: 100% (n=90)

^arMDD PmR+ vs. Controls: $t(165)=2.80$, $B=.22$, $p=.006$ ^brMDD PmR- vs. Controls: $t(165)=2.37$, $B=.19$, $p=.019$ [†]rMDD PmR+ vs. rMDD PmR-: $t(165)=0.56$, $B=.06$, $p=.572$

Table 3. Traditional Probabilistic Reward Task Measures

	rMDD PmR+	rMDD PmR-	Controls	Group Comparison
Accuracy	.76 (0.6)	.77 (0.9)	.77 (0.7)	$F(2,163)=0.04, p=.957$
Response Bias	.15 (.15)	.17 (.16)	.21 (.20)	$F(2,163)=1.37, p=.258$
ΔResponse Bias	.05 (.15)	.04 (.30)	.06 (.23)	$F(2,163)=0.07, p=.937$
Discrimination	.55 (.14)	.58 (.21)	.59 (.20)	$F(2,163)=0.57, p=.564$
ΔDiscrimination	.03 (.19)	.03 (.18)	.02 (.18)	$F(2,163)=0.30, p=.742$

Table 4. Effect of group (PmR+ versus controls) on learning rate for controlling for psychopathology covariates.

DV: α	PmR+ vs. Controls	Covariate
1.	$B=.28, p=.005$	$B=-.13, p=.096$
2.	$B=.27, p=.009$	$B=.01, p=.911$
3.	$B=.29, p=.008$	$B=.05, p=.536$
4.	$B=.24, p=.029$	$B=-.05, p=.589$
5.	$B=.28, p=.009$	$B=.02, p=.769$
1=Inventory of Depressive and Anxiety Symptoms (IDAS), Well-being (reverse-scored) 2=Personality Inventory for the DSM-5, Anhedonia 3=IDAS, Depression 4=IDAS, Social Anxiety 5=IDAS, Panic		

Table 5. Trial-wise value estimates for participants' choices controlling for psychopathology covariates.

DV: V PmR+ vs. Controls	PmR+ vs. PmR-
Block 1, Early Trials	Block 1, Early Trials
1. $B=.23, p=.001$	1. $B=.15, p=.036$
2. $B=.23, p=.003$	2. $B=.16, p=.035$
3. $B=.23, p=.006$	3. $B=.16, p=.035$
4. $B=.20, p=.016$	4. $B=.14, p=.043$
5. $B=.22, p=.005$	5. $B=.16, p=.033$
Block 1, Late Trials	
1. $B=.17, p=.015$	
2. $B=.17, p=.019$	
3. $B=.15, p=.049$	
4. $B=.15, p=.047$	
5. $B=.15, p=.049$	
Block 2, Late Trials	
1. $B=.17, p=.018$	
2. $B=.17, p=.024$	
3. $B=.14, p=.049$	
4. $B=.15, p=.048$	
5. $B=.15, p=.049$	
1=IDAS Well-being (reverse-scored) 2=PID-5 Anhedonia 3=IDAS Depression 4=IDAS Social Anxiety 5=IDAS Panic	

Figures

Figure 1.

Schematic representation of a trial from the Probabilistic Reward Task.

Figure 2.

A. Summed Akaike information criterion (AIC) values for the Stimulus-Action, Action, and Belief Models across participants. AIC values indicate that participants' behavior was better captured by the Action Model than the Stimulus-Action and Belief Models. B. Average AIC values for the Action, Stimulus-Action, and Belief Models for each group. AIC values indicate that for each group, participants' behavior was better captured by the Action Model than the Stimulus-Action and Belief Models.

Figure 3.

Model-derived trial by trial value expectations (V) averaged across the remitted major depressive disorder (rMDD) and control groups.