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Dissociable Roles of Reward Prediction Error in the Contrasting Mood Dynamics of Depression and Anxiety

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eLife Assessment

This **important** study uses a tripartite transdiagnostic computational framework to distinguish depression-specific, anxiety-specific, and shared psychopathology dimensions, in their relationships to mood variability and mood reactivity to reward prediction errors across multiple large non-clinical cohorts and a clinical sample. The evidence is **convincing** overall because the study combines large samples, a well-characterized gambling task and in-depth computational and psychometric analyses, and it replicates the depression-specific association with blunted reward prediction error-sensitivity in a clinical sample. However, the anxiety-specific effects are less consistently supported across individual datasets, may be underpowered in the clinical cohort because of comorbidity, and some aspects of the factor-analytic, risk-attitude, and mediation analyses would benefit from clearer explanation. These findings advance a mechanistic account of how distinct symptom dimensions differentially shape reward-based mood updating and variability, providing a principled framework for future transdiagnostic modeling.

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Abstract

Mood fluctuations, central to human experience, are profoundly influenced by reward prediction error (RPE). Although depression and anxiety traditionally exhibit contrasting mood fluctuations, their interrelated nature has made it challenging to pinpoint their specific roles in RPE-induced mood variations. In this study, we employed a computational model of momentary mood within a gambling task, involving 2,043 participants across five experiments. These participants also completed a series of questionnaires designed to allow us to differentiate the influences of anxiety- and depression-specific traits through bifactor modelling. Results showed that depression was associated with dampened mood fluctuations due to mood hyposensitivity to RPE. In contrast, anxiety correlated with heightened mood fluctuations stemming from mood hypersensitivity to RPE. We also validated deficit mood sensitivity to RPE with depression in patients with affective disorders, confirming the clinical utility of this affective mental parameter. Moreover, the shared depression/anxiety component was linked to lower affective baseline and greater risk aversion.

Collectively, our results uncover computational dissociation of depression vs. anxiety using RPE-based mood modeling and present multi-dimensional computational signatures for depression and anxiety, with clinical relevance for management of mood disorders.

Introduction

Happiness is a central component of human experience, providing a "measure of right and wrong" and guiding actions undertaken to maximize well-being[1,2]. Yet, humans often fail to do so as evidenced by the high prevalence of mood disorders such as depression and anxiety. It is therefore crucial to know what causes mood fluctuations to identify what promote happiness and what triggers mood disorders, and how.

Mood dynamics are strongly influenced by reward prediction error (RPE) — the discrepancy between expected and actual outcomes[3–9]. The modulatory role of RPE is particularly prominent in uncertain environments, such as gambling scenarios, where it significantly impacts mood fluctuations[8,10–13]. Despite the critical role of RPE-induced mood fluctuations in adaptive behavior[4,7,14,15], atypical mood dynamics are considered risk factors for the development of affective disorders[4,16–18]. Specifically, depression, often associated with blunted emotional response[19,20], may suppress mood fluctuations[21–25]. In contrast, anxiety, characterized by exaggerated responses to uncertainty[26], might intensify them[27,28]. However, distinguishing the unique effects of depression and anxiety on mood dynamics presents a significant challenge, owing to their overlapping symptoms and entangled nature[29,30].

Recent advances have been made to understand the unique qualities and differential influences on decision-making based on the bifactor validation of a tripartite model of depression and anxiety[31,32], which segregates symptom variance into common, depression-specific, and anxiety-specific components[30,33,34]. For example, Gagne et.al., (2022) has utilized bifactor analysis to demonstrate that depression negatively influences prior belief, while anxiety positively contributes to negative bias for belief updating[31]. Intriguingly, mood sensitivity to RPE seems intact in both patients with major depressive disorder (MDD) and individuals scoring high in depression questionnaires[12,25,35], contrary to the hypothesized decrease in mood patterns in depression[25]. Individuals with anxiety not only show heightened vigilance before an outcome is known[26] but also assign greater precision to the information that resolves that uncertainty[36,37]. This precision-weighting amplifies the affective influence of RPE, leading to larger mood shifts when outcomes deviate from expectations[38,39]. Given the opposite impacts of depression and anxiety on mood fluctuations[23,24,27,28], an open possibility for the observed intact mood fluctuations[25] is their opposite roles in mood sensitivity to RPE, which may counteract each other. To test this hypothesis, we turn to the bifactor model to disentangle these interrelated influences.

This study utilizes a computational model of momentary mood in a gambling task involving five experiments and 2,043 participants who also completed a series of questionnaires, permitting bifactor analysis to disassociate influences from anxiety and depression-specific traits on mood fluctuations. We measured momentary mood by asking participants "How happy are you at the moment?". This measurement has also been referred to as happiness or momentary subjective well-being[8,14,15,40]. Although mood is thought to persist for hours, days, or even weeks[4,5,7,41], momentary mood, measured over the timescale in the laboratory setting, represents the accumulation of the impact of multiple events[4,7,8,11–13,25,35,42] at the scale of minutes. Momentary well-being external validity is demonstrated e.g., through its association with depression symptoms[12,25]. We first validate the tripartite model of depression and anxiety before exploring their specific roles in mood fluctuations (n = 901). Next, we measured mood sensitivity to RPE in a gambling task, and investigated its relation to anxiety vs. depression-specific traits based on the tripartite model in laboratory test (n = 44) and another two online experiment settings (n = 747/235). In the final experiment (n = 116), we confirmed these results in the clinical setting (i.e., patients with affective disorders), revealing dissociable roles of RPE in the contrasting mood dynamics of depression and anxiety.

Results

Experimental Protocol

After completing a battery of questionnaires[32] (see Methods), participants performed a gambling task with momentary mood ratings[8,10,13]. Within this task, subjects were asked to make decisions between certain vs. gamble options (2 possible outcomes, 50% probability for each). Participants were instructed to assess and rate their mood every 2-3 trials (Figure 1). Detailed participant demographics are outlined in Table 1. The choice (e.g., gambling rates) and mood data (e.g., the initial mood, mean mood, and mood variation) showed similar patterns with previous studies on measurements of momentary moods during the gambling task (Figure S2 & S3)[10,43]. We also replicated classic mood-relevant effects[12,40]: better mood after gain than loss and mood drifted over time (*ps* <0.001; Figure S4).

Table 1. Basic demographic details.

	Psychometric dataset (N = 901)	Laboratory dataset (N=44)	Online dataset 1 (N = 747)	Online dataset 2 (N = 235)	Clinical Dataset (N = 116)
Gender (female)	676	18	500	143	89
Age	22.04±2.10	20.05±1.70	20.90±2.41	21.67±2.52	16.40±3.86
MASQaa	24.70±8.13	22.98±6.14	24.16±7.60	23.92±7.62	43.51±10.51
TAIanx	16.06±4.86	15.00±4.95	15.69±4.97	15.99±5.34	23.35±5.44
CESD	35.28±10.72	33.30±10.73	34.82±10.74	35.06±11.41	55.64±13.04
BDI	11.38±9.35	7.91±8.11	10.04±8.94	10.26±9.18	22.66±17.91
BFIn	33.99±8.82	30.52±8.26	32.82±9.37	32.79±9.81	43.75±9.31
PSWQ	48.28±11.61	46.89±12.52	48.25±12.15	48.36±13.29	59.18±12.87
MASQad	61.49±14.00	60.23±16.63	58.07±15.30	59.57±15.71	77.16±15.39
TAIdep	29.39±5.47	27.32±6.16	27.61±6.27	27.85±6.34	35.97±4.91

Descriptive data are presented as mean±SD. Abbreviations: MASQaa, the subscale of anxious arousal in the Mood and Anxiety Symptoms Questionnaire; TAIanx, the subscale of anxiety in the Trait Anxiety Inventory; CESD, Center for Epidemiologic Studies Depression Scale; BDI, Beck Depression Inventory; BFIn, the subscale of neuroticism in the Big Five Inventory; PSWQ, Penn State Worry Questionnaire; MASQad, the subscale of anhedonic depression in the Mood and Anxiety Symptoms Questionnaire; TAIdep, the subscale of depression in the Trait Anxiety Inventory.

To confirm model-based findings, we used the classic mood model assuming that momentary moods are contributed by the recency-weighted average of the chosen certain reward (CR), expected value of the chosen gamble (EV), and reward prediction error (RPE; Equation 1; Figure 1H)[8,13]. RPE was defined as the difference between the obtained and expected value. We also incorporated a drift parameter to account for the gradual change in happiness over time [40,42].

$$\begin{aligned}\text{Happiness}(t) = \beta_0 + \beta_{CR}\sum_{j=1}^{t}\gamma^{t-j}CR_j + \beta_{EV}\sum_{j=1}^{t}\gamma^{t-j}EV_j \\ + \beta_{RPE}\sum_{j=1}^{t}\gamma^{t-j}RPE_j + \beta_t t\end{aligned} \quad (1)$$

This model explained momentary moods well for all datasets (r^2: mean ± SD = 0.67 ± 0.20 for the laboratory dataset, 0.69 ± 0.17 for the online dataset 1, and 0.64 ± 0.19 for the online dataset 2, and 0.47 ± 0.21 for the clinical dataset; see Supplementary Materials Note 2 and Note 5 for our model space; see Table S2 & Table S4 for model comparisons), with good performance for parameter recovery (Figure S5). For all datasets with the gambling task, we replicated previous mood model-based findings[8,13]: 1) the RPE weight was significantly higher than the EV weight (ts > 3.84, *ps* < 0.001; Figure 1I), suggesting that moods are more sensitive to the prediction error of experiential outcome than the expectation of value; 2) the baseline mood parameter β_0 was positively correlated with the initial mood before the experiment (*rs* > 0.25, *ps* < 0.006), further validating this mood model. We also replicated previous depression-related findings[25]: depression

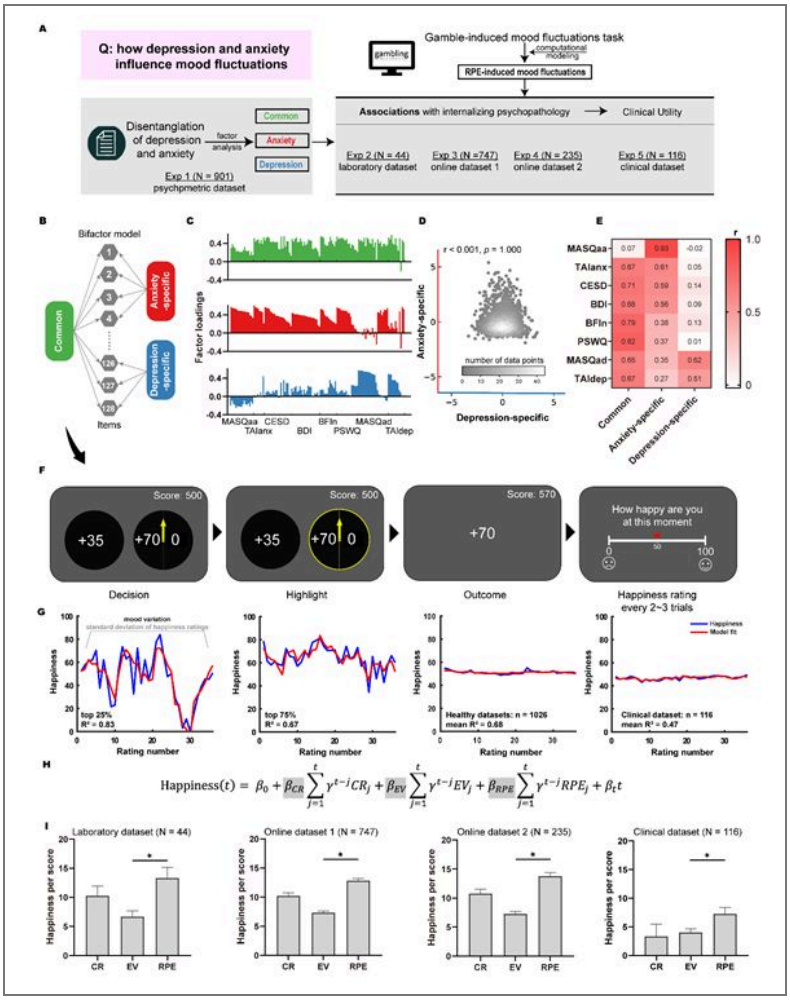

Figure 1. Experimental protocol.

A) Study outline. This study aims to answer how depression and anxiety influence mood fluctuations. The first experiment assesses the bifactor structure that disentangling depression and anxiety in the psychometric dataset (N = 901). The second experiment with this battery of questionnaires and the gambling-induced mood fluctuations task tests correlational associations of depression and anxiety with RPE-induced mood fluctuations (the laboratory dataset, N = 44). The third and fourth experiments replicates the 2nd experiment in the online context (the online dataset 1, N = 747; the online dataset 2, N = 235). The fifth experiment with the mood fluctuations task tests the potential treatment of mood stabilizer on RPE-induced mood fluctuations in the clinical dataset (N = 61). B) Mapping between three factors and 128 items in the bifactor model. C) Factor loadings of items on the common factor, the anxiety-specific factor, and the depression-specific factor. D) Orthogonal nature between the common factor and the anxiety-specific factor and between the common factor and the depression-specific factor in a total of 1950 participants for each dataset. E) Mean correlations of factor scores with questionnaire scores. Overall, the common factor showed high correlations with all questionnaires. The depression-specific factor was mainly contributed by the TAIdep and the MASQad, while the anxiety-specific factor was mainly contributed by the remained questionnaires. These correlational results confirmed the bifactor structure underlying anxiety and depression. F) Gambling task design. On each trial, participants were asked to choose between a certain option and a gambling option. Once selected, the corresponding outcome was resolved in the center of the screen. The cumulative score was always shown in the right-upper corner. Every 2 or 3 trials, participants were asked to complete a self-pace rating of "How happy are you at the moment" on a slider from 0 (very unhappy) to 100 (very happy). G) Time dynamics of happiness ratings for the individual with mood variation in the top 25% (the first panel), the individual with mood variation in the top 75% (the second panel), healthy datasets, and the clinical dataset. H) Momentary mood model. I) Results of momentary mood model for each dataset. Abbreviations: MASQaa, the subscale of anxious arousal in the Mood and Anxiety Symptoms Questionnaire; TAIanx, the subscale of anxiety in the Trait Anxiety Inventory; CESD, Center for Epidemiologic Studies Depression Scale; BDI, Beck Depression Inventory; BFIn, the subscale of neuroticism in the Big Five Inventory; PSWQ, Penn State Worry Questionnaire; MASQad, the subscale of anhedonic depression in the Mood and Anxiety Symptoms Questionnaire; TAIdep, the subscale of depression in the Trait Anxiety Inventory; CR, certain reward; EV, expected value; RPE, reward prediction error.

symptom measured by Beck Depression Inventory (BDI) was negatively correlated with the baseline mood parameter β_0 (r = -0.173, p < 0.001; Figure S6). In sum, these confirmatory results suggest 1) engagements of questionnaires and tasks for all included participants, 2) the important contribution of RPE to momentary mood fluctuations, and 3) robust relationships between depression measured by BDI and mood baseline.

Depression links to blunt mood fluctuations through hypo-sensitivity to RPE

We first examined how depression influences mood fluctuations and the underlying computational mechanism. It has been shown that depression and anxiety often co-occur at the symptom level[29,34,44,45], e.g., high correlation between depression measured by BDI and anxiety measured by the subscale of anxiety in the Trait Anxiety Inventory (r = 0.75, p < 0.001). To differentiate unique influence of anxiety and depression-specific factor, we applied the validated bifactor structure from the psychometric dataset (Figure 1C) to decompose depression/anxiety symptoms into three orthogonal components: the common factor, the anxiety-specific factor, and the depression-specific factor (-0.001 < rs < 0.001, ps = 1.000; see Supplementary Materials Note 1 for psychometric properties of the tripartite model of depression and anxiety in our sample; n = 901). To test the contribution of depression-specific loading on mood fluctuations, we performed correlation analyses between the depression-specific factor and mood variation (i.e., standard deviation of happiness ratings). We found convergent results across the laboratory dataset, the online dataset 1 and online dataset 2. Specifically, results showed significantly negative correlations of the depression-specific score with mood variation (rs < -0.13, ps < 0.041; Figure 2ABC). Model-based correlational analyses further showed significantly negative correlations between the depression-specific factor and mood sensitivity to RPE (β_{RPE}; rs < -0.14, ps <0.019; Figure 2EFG), which remained significant after controlling for gender, age, earning, and mood drift (ps < 0.035). These results hold with bootstrap validation, indicating reduced mood fluctuations and decreased impact of RPE on mood with depression loading. Given the high correlation between β_{RPE} and mood variation (rs > 0.67, ps < 0.001), we further conducted mediation analysis among the depression loading, β_{RPE}, and mood variation, with the assumption of the mediating effect of mood parameter of RPE on the association between depression and mood variation. Result supported our hypothesis (ps < 0.015; Figure 2I), suggesting that higher depression loading flats momentary mood variation by reducing sensitivity to RPE. See Table S7 for statistical values for each dataset. Note that this mediation analysis is in line with previous literature in computational psychiatry[46]. Although both mood fluctuations and mood sensitivity to RPE are indeed derived from the same happiness data during the gambling task, these two measures quantify conceptually distinct aspects of mood dynamics: mood fluctuation captures the overall variability in mood ratings across trials, whereas mood sensitivity to RPE specifically quantifies mood sensitivity to prediction errors on a trial-by-trial basis. To further test whether the impact of depression on mood fluctuations was specific to RPE-based mood sensitivity, we conducted a linear regression against depression-specific factor with mood sensitivity to CR, EV, and RPE as regressors for the combined dataset (N = 1026). We observed deficient RPE-specific mood sensitivity with depression loading (CR: t = 0.274, p = 0.784; EV: t = -1.069, p = 0.285; RPE: t = -3.282, p = 0.001; Figure 2K).

Anxiety relates to intensified mood fluctuations via hyper-sensitivity to RPE

We then examined how anxiety influences mood fluctuations. We observed signs of positive correlations of the anxiety-specific factor with mood variation (the laboratory dataset: r = 0.10, p = 0.531; the online dataset 1: r = 0.08, p = 0.026; the online dataset 2: r = 0.19, p = 0.004), and signs of positive correlations of the anxiety-specific factor with β_{RPE} (the laboratory dataset: r = 0.04, p = 0.820; the online dataset 1: r = 0.05, p = 0.216; the online dataset 2: r = 0.19, p = 0.004; Figure 2A-C & 2E-G). Note that the phrase “signs of positive correlations” was intended to describe the directionality (i.e., positive r values) of the observed relationships across datasets, regardless of

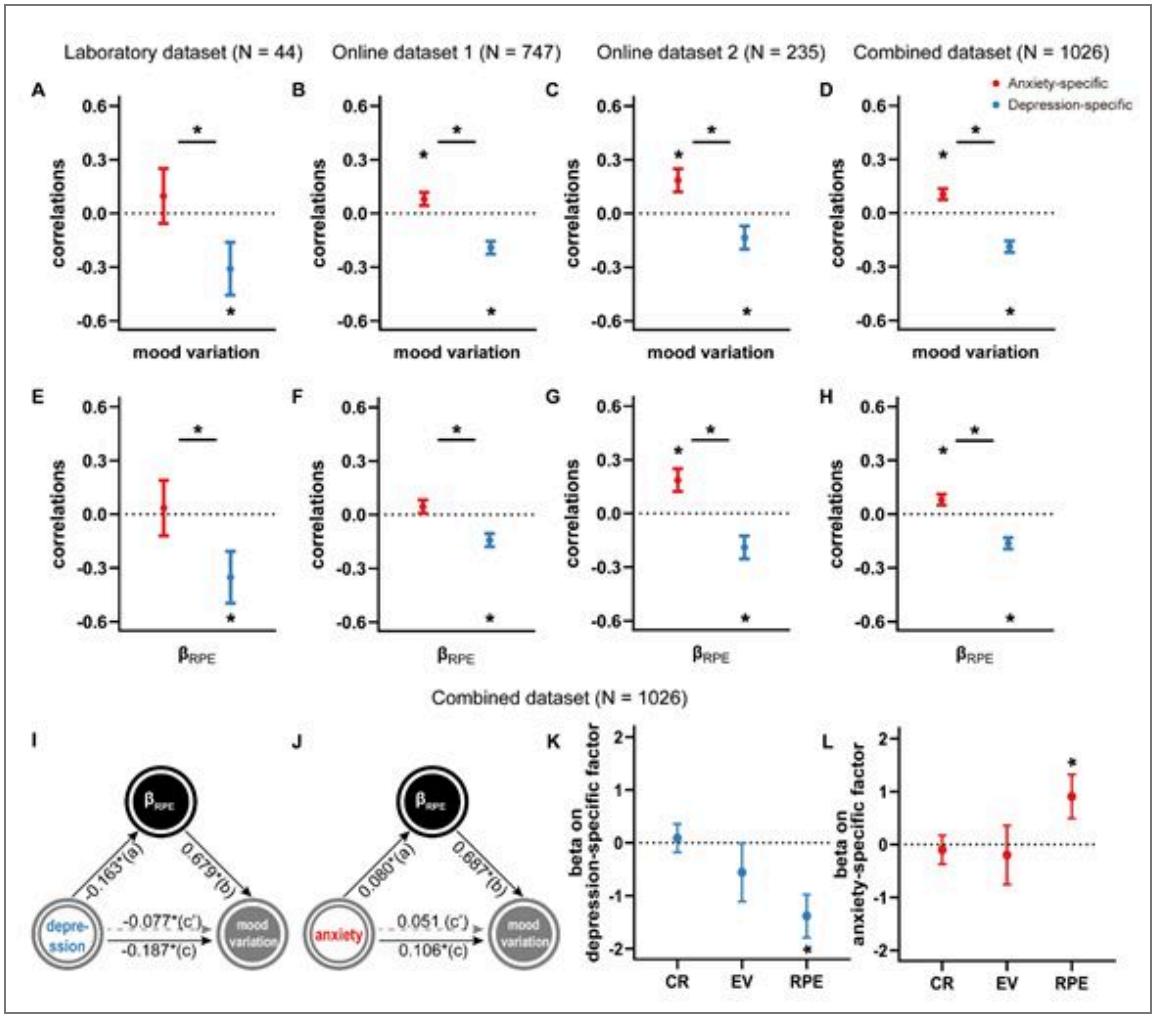

Figure 2. Results for depression vs. anxiety on mood fluctuations.

Correlations of depression and anxiety factor score with mood variation and mood parameter of RPE (β_{RPE}) for the laboratory dataset (AE), the online dataset 1 (BF), the online dataset 2 (CG), and the combined dataset (N =1026; DH). I) The lower RPE-weighted mood parameter mediated the effects of depression on decreased mood fluctuations. J) The higher RPE-weighted mood parameter mediated the effects of anxiety on increased mood fluctuations. K) Depression-specific factor was specifically relevant to decreased mood sensitivity to RPE among mood parameters. L) Anxiety-specific factor was specifically relevant to increased mood sensitivity to RPE among mood parameters. The regression coefficients were represented by mean ± se, which were estimated by bootstrap. Abbreviations: CR, certain reward; EV, expected value; RPE, reward prediction error; *p<0.05.

their statistical significance, given inconsistent results at the statistical level. Comparison of correlations showed significant differences both between depression-mood variation and anxiety-mood variation associations (for the laboratory dataset: Z = -1.84, p = 0.033; for the online dataset 1: Z = -5.36, p < 0.001; for the online dataset 2: Z = -3.42, p < 0.001) and between depression-β_{RPE} and anxiety-β_{RPE} associations (for the laboratory dataset: Z = -1.77, p = 0.038; for the online dataset 1: Z = -3.67, p < 0.001; for the online dataset 2: Z = -4.00, p < 0.001; Figure 2A-C & 2E-G), suggesting different roles of depression and anxiety in mood fluctuations. Given that recent evidence suggested that reliable individual difference can be tested with large data size[47] and that anxiety spanned a wide variety of symptoms[48], we combined data from the laboratory dataset, online dataset 1, and the online dataset 2 (total N = 1026). Linear mixed-effect models against mood variation and β_{RPE} with all three factors, with dataset as a random factor, showed significant positive association between the anxiety-specific factor and mood variation (t = 3.46, p < 0.001), while significant negative association between the depression-specific factor and mood variation (t = -6.13, p < 0.001). Regarding RPE-based mood sensitivity, we found the anxiety-specific factor was significantly positively correlated with β_{RPE} (t = 2.60, p = 0.009), while the depression-specific factor was significantly negatively correlated with β_{RPE} (t = -5.30, p < 0.001). These results remained significant after controlling for gender, age, earning, and mood drift (ps < 0.001). Mediation analyses further showed that hyper-sensitivity of mood to RPE mediated higher mood fluctuations with anxiety loading (a×b = 0.055, 95% CI = [0.013, 0.097], p = 0.010; Figure 2J). Moreover, linear regression against anxiety-specific factor with mood sensitivity to CR, EV, and RPE as regressors showed RPE-specific mood hyper-sensitivity with anxiety loading (CR: t = -0.36, p = 0.718; EV: t = -0.35, p = 0.724; RPE: t = 2.19, p = 0.028; Figure 2L). To rule out potential confounds of subjective calibration of rating scales, for example, individuals with nonlinear utility functions (e.g., risk-seeking participants) could display disproportionately high mood responses for large relative to small wins, we conducted several complementary analyses, including re-analyzing two open datasets using similar risk-based decision-making tasks with repeated happiness ratings: Vanhasbroeck et al. (2021; n = 49) and Rutledge's smartphone App dataset (n = 46,204). See Supplementary Note 3 for details. Collectively, individual differences in risk preference did not significantly affect the calibration of mood ratings or alter our primary findings regarding distinct mood dynamics in anxiety versus depression. Instead, these results possibly suggest dissociable processes for decision-making (i.e., value perception) and mood dynamics. Together, these results show that depression and anxiety play opposite roles in mood dynamics via RPE-specific mood sensitivity.

Clinical utility for deficit RPE-based mood fluctuations with depression

To test whether abnormalities of mood fluctuations driven by RPE can serve as a clinically relevant computational marker for depression and anxiety, we recruited patients with affective disorders (n = 116) to complete the battery of questionnaires and the gambling-momentary mood rating task (Figure 1). For demographic, psychological and clinical information, see Table 1 and Table S8. We observed significantly negative correlations of the depression-specific score with mood variation (r = -0.239, p = 0.009) and RPE-based mood parameter (r = -0.216, p = 0.020), which remained significant after controlling for demographic and clinical characteristics, earning, and mood drift (ps < 0.05). These results hold with bootstrap validation. Mediation analyses further showed that hypo-sensitivity of mood to RPE mediated lower mood fluctuations with depression loading (a×b = -0.141, 95% CI = [-0.261, -0.038], p = 0.021; Figure 3). However, we did not observe any significant correlation with anxiety (mood variation: r = -0.092, p = 0.327; RPE-based mood parameter: r = -0.095, p = 0.311).

Exploratory analysis of the common factor

Given the theoretical relevance of the common factor—often linked to shared symptoms such as somatic complaints, sleep disturbances, and cognitive impairments[34]—as well as the potential for non-linear associations with symptom severity, we conducted exploratory analyses to examine its

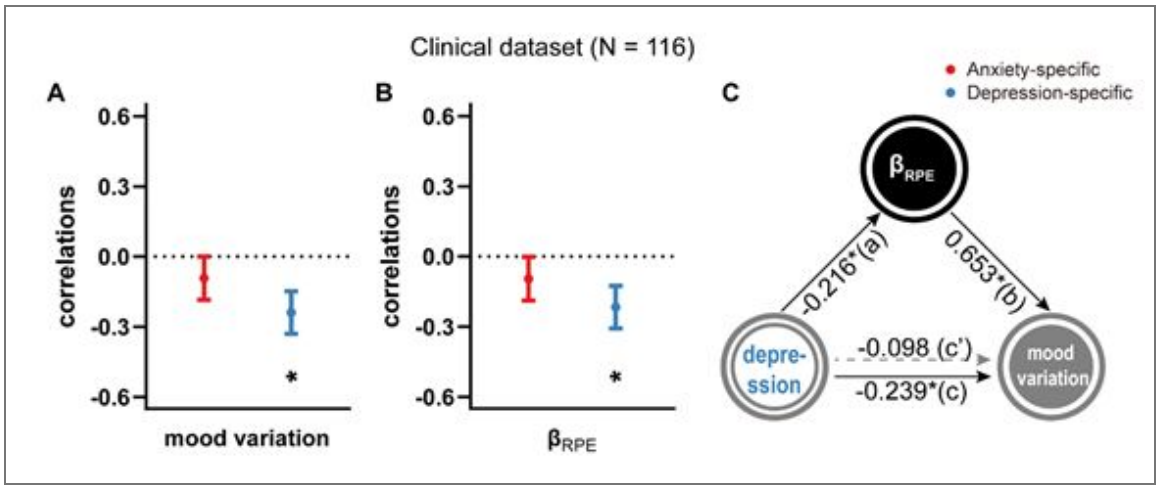

Figure 3. Clinical validation for deficit RPE-based mood fluctuations with depression.

AB) Correlations of depression and anxiety factor score with mood variation and mood parameter of RPE (β_{RPE}) for the clinical dataset. C) The mediation model among depression, β_{RPE}, and mood variation in the clinical population. The regression coefficients were represented by mean ± se, which were estimated by bootstrap. Abbreviations: RPE, reward prediction error; *p<0.05.

relationship with choice and mood parameters in both healthy and clinical datasets. To ensure robustness and reduce the risk of false positives, we report only findings that were consistent across datasets. Model comparisons using BIC consistently favored linear over non-linear models in capturing associations between the common factor and parameters (Table 2). Specifically, we observed significant negative associations between the common factor and two key variables: the baseline mood parameter and risk attitude for gain (Figure 4). Note these parameters were not consistently related to either the depression-specific or the anxiety-specific factor (depression × baseline mood parameter : r = -0.12, p < 0.001 for the healthy dataset and r = -0.08, p = 0.934 for the clinical dataset; anxiety × baseline mood parameter : r = -0.14, p < 0.001 for the healthy dataset and r = -0.15, p = 0.104 for the clinical dataset; depression × risk attitude for gain : r = -0.02, p = 0.505 for the healthy dataset and r = -0.00, p = 0.961 for the clinical dataset; anxiety × risk attitude for gain : r = 0.01, p = 0.846 for the healthy dataset and r = 0.04, p = 0.688 for the clinical dataset). These findings suggest that higher levels of general internalizing psychopathology are associated with a lower affective setpoint and increased risk aversion, consistent with transdiagnostic features observed across mood and anxiety disorders.

Table 2. Exploratory analysis for the g factor.

Models	Healthy dataset (n=1026)		Clinical dataset (n=116)	
	BIC	Results	BIC	Results
β_0~ common	**-830.23**	b = -0.03, t = -6.54, p < 0.001, 95% CI = [-0.042 -0.022]	**-113.74**	b = -0.03, t = -2.05, p = 0.043, 95% CI = [-0.052 -0.001]
β_0 ~ common + common^2	-823.74		-110.91	
α_{gain} ~ common	**551.76**	b = -0.03, t = -3.08, p = 0.002, 95% CI = [-0.049 -0.011]	**101.7**	b = -0.11, t = -3.24, p = 0.002, 95% CI = [-0.172 -0.041]
α_{gain} ~ common + common^2	553.73		106.12	

Note: For healthy dataset, we added datasets as random variable. β_0, the mood baseline parameter; α_{gain}, risk attitude for gain.

Discussion

Although atypical mood dynamics are obviously at the core of depression and anxiety[21,23,27,28], little is known about their underlying computational mechanisms. Our study offers computational insights into the differing impacts of depression and anxiety on mood fluctuations. Through the orthogonal decomposition of depression and anxiety, we were able to distinguish their unique effects on mood dynamics. Depression was found to be related with blunt mood fluctuations through a decrease in mood sensitivity to RPE, whereas anxiety was linked to intensified mood fluctuations via a heightened sensitivity to RPE. Of substantial note is our finding that we validated deficit mood sensitivity to RPE with depression in patients with affective disorders, identifying clinical utility for our affective computational markers. Moreover, the shared depression/anxiety component was linked to lower affective baseline and greater risk aversion.

Depression and anxiety, though often coexisting[29,30], have contrasting influences on mood dynamics, aligning with their core characterizers of mood problems[24,27,28]. Our computational model not only validates the significant role of RPE in mood dynamics but also highlights the divergent mediating roles of RPE-induced mood sensitivity in the effects of depression and anxiety on mood fluctuations. The opposite impacts of depression and anxiety on mood sensitivity to RPE may complement the previous finding of intact RPE-induced mood fluctuation in depression[12,25,35]. This further underscores the necessity of decomposing depression and anxiety for precise measurement in mood studies, which can enhance our understanding of their unique impacts on emotion processing and cognitive flexibility. For example, using bifactor analysis, Gagne et.al., (2020) has shown that the common factor, but not the anxiety-specific factor, contributes to maladaptation to environmental volatility[32], which complemented previous findings that trait anxiety influenced inflexible adjustment to volatility[49].

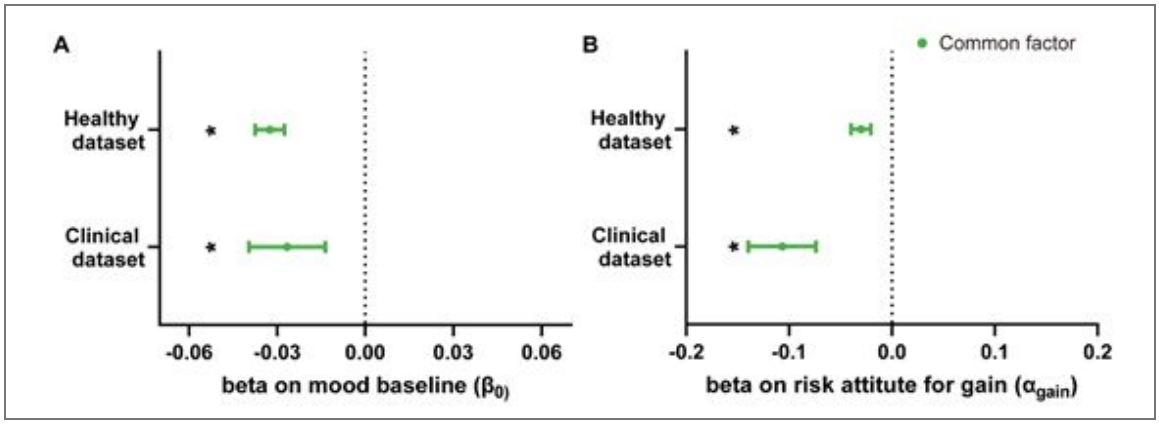

Figure 4. Consistent results for the common factor across the healthy and clinical dataset.

Negative associations of the common factor with the baseline mood parameter A) and risk attitude for gain B). The regression coefficients were represented by mean ± se. *p<0.05.

This study bridges multiple fields of research on depression. Depression, or anhedonia, describes disability to feel pleasure[50]. From the perspective of affective science, researchers used experience sampling technique across several weeks or months[5,7,27,51], showing flattened mood fluctuations in depression[23,24]. Instead, in the field of decision making and learning, most studies adopted laboratory-based learning or gambling tasks[52,53], revealing alterations in reward function in individuals suffering from depression[52,53]. However, how their reward processing transfers into aberrant hedonic experience remains largely unknown. Our study employed a gambling task with momentary mood ratings, allowing us to examine reward-induced mood dynamics. In line with previous literature from different fields[20,24], this study provides evidence for mood hypo-sensitivity to reward prediction error with depressive symptoms.

In addition to confirming the putative role of anxiety in intensified mood fluctuations[26–28], we provided its underlying computational account: heightened mood sensitivity to RPE. Our study provides a computational account of this phenomenon, showing that anxiety is associated with heightened mood sensitivity to RPE, but not to value expectation. Anxiety is marked by exaggerated anticipatory responses to uncertainty and excessive affective reactivity[26]. Since RPE signals the resolution of uncertainty, our findings suggest a mechanistic pathway through which anxiety influences uncertain-resolved mood—by amplifying affective responses to unexpected outcomes. In this way, individuals with higher anxiety may exhibit a stronger need to resolve uncertainty[49], leading to increased mood reactivity to RPE. Moreover, our results were different to Browning et al. (2015)[49], who reported impaired learning from uncertainty in anxiety. However, the two findings may reflect different stages or aspects of processing: while Browning et al. focused on learning rate adaptation in response to environmental volatility, our study targets affective responsiveness to outcome-level surprises. Thus, it is possible that anxious individuals exhibit reduced cognitive flexibility in belief updating, yet simultaneously increased emotional reactivity to prediction errors—highlighting a dissociation between cognitive and affective systems. Notably, the pattern of heightened RPE sensitivity observed in the healthy population was not replicated in the clinical sample. This discontinuity may reflect non-linear or stage-dependent features of anxiety and depression, or potential floor effects in affective reactivity among patients with severe symptoms. Alternatively, it may suggest that dimensional models derived from healthy populations do not always generalize straightforwardly to clinical settings. Another possibility is the disruption of mood homeostasis in clinical population (a sign for negative correlation with anxiety)[39,54]. In healthy individuals, opposing influences of depression and anxiety on mood variation may work in tandem to maintain emotional equilibrium. In contrast, individuals with affective disorders may lack the regulatory balance required to stabilize mood in the face of competing emotional signals.

The common factor—reflecting general internalizing psychopathology—was robustly and consistently associated with both a lower baseline mood (i.e., affective setpoint) and increased risk aversion for potential gains. Regarding the former, previous large-scale studies have established a reliable association between lower affective setpoint and elevated depressive symptoms, including individuals scoring high on the BDI and patients diagnosed with major depressive disorder (MDD)[25]. Expanding on this, our bifactor approach decomposed depression and anxiety into three orthogonal components and revealed that only the common factor, not the depression- or anxiety-specific components, was significantly associated with a lower mood baseline. This finding suggests that it is general emotional distress—rather than core features such as anhedonia or pathological worry—that exerts a broader influence on individuals' affective setpoints[4]. With respect to decision-making, prior literature has linked pathological anxiety with enhanced risk aversion[55]. In line with this, our results again point to the common factor as the driving force, rather than anxiety-specific variance per se. This suggests that heightened risk aversion is a transdiagnostic feature of internalizing psychopathology, rather than uniquely attributable to anxiety. Notably, model comparison favored linear over non-linear associations, indicating that even subclinical levels of general internalizing symptoms can exert measurable influences on mood and decision processes. Taken together, these findings underscore the importance of incorporating general psychopathology dimensions into computational models of affect and choice.

Several limitations of the present study should be noted. First, although the r-to-z transformed correlation comparisons appeared more consistent than individual zero-order correlations for anxiety-related results, this pattern may suggest potential confounding due to scale-related variability—that is, differences in how participants use mood rating scales. While our additional analyses, including those conducted on two publicly available datasets with similar paradigms (Vanhasbroeck et al., 2021[13], n = 49; Rutledge et al., n = 46,204), did not support this explanation (see Supplementary Note 3), future studies would benefit from task designs that better control for scale sensitivity and enhance mood responsiveness to risk, possibly by incorporating more emotionally salient or high-stakes decision contexts. Second, adolescence represents a critical developmental period for the emergence of emotional problems, and the transition from anxiety to depression is a well-documented trajectory during this stage. However, how affective computational parameters such as mood sensitivity to RPE evolve during this transition remains unclear. Specifically, questions such as whether mood homeostasis becomes more vulnerable in adolescence, or whether the mechanisms underlying mood regulation shift qualitatively during this period, are of high relevance. Addressing these developmental questions could inform preventive strategies and improve early interventions aimed at reducing maladaptive emotional reactivity—particularly in adolescents who are at heightened risk for internalizing psychopathology.

In conclusion, our findings emphasize the distinct effects of depression and anxiety on mood fluctuations, underlining the essential role of symptom variance partition in affective science. The specific association we identified between depression and RPE-induced mood sensitivity has significant implications for the development of computational mechanism-based treatments for affective disorders, providing a promising direction for future research and clinical treatment.

Methods

A total of 2634 participants via online platforms (questionnaires from *https://www.wjx.cn* and tasks from *https://www.naodao.com*) took part in four experiments, including a psychometric experiment, a laboratory experiment, an online replication experiment, and an online intervention experiment. The study was approved by the Ethics Committee of Beijing Normal University. Written informed consent was obtained from all participants before each experiment. Finally, participants were paid for the basic participation fee and a bonus based on their task performance, which was instructed before the experiment.

Participants

Data from 1145 participants was collected in the psychometric experiment. Participants were excluded if 1) they failed any of the attentional checks (4 items); 2) they made the same choices for all items; 3) they responded with extreme inconsistency in two similar questionnaires (difference in z-scores out of ±2). The final sample for the psychometric dataset consisted of 901 participants. We recruited 59, 1087, and 343 participants for the laboratory experiment, the online replication experiment, and the online intervention experiment, respectively. Participants were excluded if 1) they failed any of the attentional checks in questionnaires (4 items) 2) they failed any of the catch trials (4 trials) and 3) they responded too fast (reaction time for decision < 200 ms) in more than 10% of the 90 trials. The final sample for the laboratory dataset, the online dataset 1, and the online dataset 2 included 44, 747, and 235 participants, separately. See Table 1 for demographic information.

Measurements of anxiety and depression

In line with previous literature orthogonally decomposing anxiety and depression[31,32], participants completed a set of Chinese version questionnaires of anxiety and depression. These measurements included the Mood and Anxiety Symptoms Questionnaire (MASQ; 62 items)[56], the Trait subscale of the State-Trait Anxiety Inventory (TAI; 20 items)[57], the Beck Depression Inventory (BDI; 21 items)[58], the Penn State Worry Questionnaire (PSWQ; 16 items)[59], the Center for Epidemiologic Studies Depression Scale (CESD; 20 items)[60], and the Big Five Inventory-2 (BFI; 60 items)[61]. Each item in the TAI, BDI, and CESD was rated on a four-point Likert scale, while five-point Likert rating scale was used for the MASQ, PSWQ, and BFI. There were four items for attentional checks, which required the participants to make a specific choice and were embedded in the entire measurements, e.g., “please select the second option for this item”.

Patients with affective disorders

We also recruited 121 patients with affective disorder, including major depressive disorder, anxiety disorder, and bipolar disorder. After excluding participants without mood variance, the final sample included 116 patients. See Table 1 and Table S8 for details.

Experimental Procedure

Participants were asked to make a choice between a certain option and a gamble (50% probability for each outcome) and to rate their momentary moods. Before the task protocol, participants were asked to rate their current happiness that we consider as their initial mood. At the beginning of the task, participants were endowed with 500 points. Each trial started with two options (a gamble option and a certain option) that presented randomly on each side (Figure 1F). Upon response, the chosen option would be highlighted in yellow for 0.5 s. Then the corresponding outcome at the screen center was presented for 1 s, followed by a fixation cross with a random duration (0.6~1.4 s). If the gamble was chosen, participants had equal probability to obtain each outcome. The obtained outcome would be accumulated to their total score, which was presenting at the top-right concern. Every 2~3 trials, participants rated “how happy are you at this moment” from 0 (very unhappy) to 100 (very happy) by moving a slider anchoring at midpoint (i.e., 50). Upon identifying their current mood, a fixation cross was presented with a random duration (0.6~1.4 s). Please note that the slider in the laboratory experiment was anchoring at the midpoint, i.e., 50. To exclude the potential anchoring effect, we did not set an anchor in the online replication experiment to check the robustness of our findings. This task consisted of 90 randomly presented trials, including 30 mixed trials, 30 gain trials, and 30 loss trials. In mixed trials, participants made a choice between a certain amount 0 and a gamble with a gain amount {40, 45, or 75} and a loss amount determined by a multiplier {0.2, 0.34, 0.5, 0.64, 0.77, 0.89, 1, 1.1, 1.35, or 2} on the gain amount. In gain trials, there was a certain gain amount {35, 45, or 55} and a gamble with 0 and a gain amount determined by a multiplier {1.68, 1.82, 2, 2.22, 2.48, 2.8, 3.16, 3.6, 4.2, or 5} on the certain gain amount. In loss trials, there were a certain loss amount {-35, -45, or -55} and a gamble with 0 and a loss amount determined by a multiplier {1.68, 1.82, 2, 2.22, 2.48, 2.8, 3.16, 3.6, 4.2, or 5} on the

certain loss amount. Many amounts and multipliers were used to accommodate a wide range of risk and loss sensitivity, as in previous literature[8,10]. We also set 4 trials embedded in the entire task for attentional checks. For example, participants were asked to make a choice between a certain gain 20 and a gamble 35/55, where the correct response for this trial was the gamble choice. All experimental procedures were programmed using Psychopy3 (2021.2.3) builder and hosted on *https://www.naodao.com*.

Model fitting

We fit model parameters by using the method of maximum likelihood estimation (MLE) with fmincon function of MATLAB (version R2015a) at the individual level. To avoid local minimum, we ran this optimization function with random starting locations 50 times. Bayesian information criteria (BIC) were used to compare model fits.

Mediation model

The mediation model was conducted using a sequence of regression steps. First, a regression of the independent variable depression on the dependent variable mood variation was performed; Second, a regression of the independent variable depression on the mediator variable β_{RPE} was performed; Next, a regression of the independent variable depression and the mediator variable on the dependent variable mood variation β_{RPE} was performed; Finally, the indirect and total effects were estimated.

Statistical analysis

We performed correlations among depression/anxiety factor scores, behavioral indices, and parameters using Matlab R2015a. To further validate our main correlational results, the percentile bootstrap CIs were estimated using the R package 'boot' and 5000 bootstrap resamples. We used an online calculator (*https://www.psychometrica.de/correlation.html*) to examine differences between two correlation coefficients. All reported tests are two-tailed. We set the significance level at $p = 0.05$.

Data availability

The data and code that support the findings of this study are available from *https://github.com/ZhihaoWangpsyer/depression_anxiety_mood*.

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Additional files

Supplementary material

Additional information

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Peer reviews

Reviewer #1 (Public review):

This is a very interesting paper. The research question is intriguing, allowing the authors to address commonly observed comorbidities between depression and anxiety and their dissociable and opposite relationship to mood fluctuations and sensitivity to reward prediction errors. The computational analyses are very in-depth, including many state-of-the-art checks and validations. Another strength is the inclusion of several large or very large samples, including a patient sample in addition to the general population sample.

I have the following questions:

(1) Factor analysis I found the hierarchical organization of the factors interesting. While this is a very common procedure in, for example, the field of intelligence (producing sub-scores and a general g factor), it is not yet very commonly used in the field of computational psychiatry (though it has been validated before for anxiety/depression, so it is used here with good reason). I was also impressed by the methodological depth. In particular, it was of note how thoroughly done it was (for example, repeating the EFA on the second half of the data

set). I have one question though: is the sample size too small for the exploratory analyses, given the number of items? Given the stability across the half-split, I imagine it is not. Perhaps the authors could spell out how many items, what would be the recommended standard for a subject-to-item ratio, and comment on this. A very technical point, the authors should specify how they extracted the factor scores from the other data sets (is it using the Thurstone or Bartlett method)? From experience (though not doing a hierarchical factor analysis), Bartlett can be somewhat better compared to the default (Thurstone) - better as in the resulting factors more closely recapitulating the factor correlations in the original sample (and independence of responses of other participants in a sample for computing a person's factor score). Could you also comment on similarities or divergences in this hierarchical factor analysis approach from another one recently used transdiagnostically in Wise et al. (2026, Translational Psychiatry)?

(2) Linking factors to task parameters As I understand it, the authors relate the orthogonalized depression/anxiety to task parameters (sensitivity to RPEs on mood and mood variations) using correlations. In order to have a better understanding of how this relates to other commonly used approaches, I would pose two questions:

(i) What are the correlations when the full (non-orthogonalized) factor scores for depression and anxiety are used? Are the signs the same? (ii) What are the results when, instead of the independent correlations, the authors perform b_RPE ~ anxiety + depression (again using the non-orthogonalized factors)?

I'm assuming all of these analyses should give the same results if the authors' hypothesis of opposing effects of anxiety and depression holds true.

Minor comments:

(1) The authors should write down when the data were collected for each study. This is because AI capabilities have massively increased since ~2020 in quite specific steps (with the public release of new AI models), meaning that AI is likely to have been able to do tasks and questionnaires without detection if data were collected recently.

(2) The authors should include a statement in the methods section that checks for AI were done. If none yet, could you do any? Recent papers (Westwood, PNAS 2025; van der Stigchel PNAS, 2026) point to the risk since at least the release of o4-mini (used in the cited paper to create very human-like behaviour).

(3) It would have been good to collect questionnaires of other, thought to be unrelated psychiatric traits, like compulsivity or schizophrenia symptoms, to check the specificity of the results, also under the assumption that higher scores on either of these skewed questionnaires can pick up individual differences in 'bad questionnaire completion'. The authors should comment on the absence of other questionnaires in the discussion in the limitations section.

(4) The authors could include a more explicit sentence in the abstract stating that the anxiety result did not hold up in the clinical population.

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Reviewer #2 (Public review):

Summary:

Despite their common co-occurrence, depression and anxiety are known to alter mood fluctuations in opposite ways. Here, the authors aimed at distinguishing depression-specific from anxiety-specific from psychopathology-general effects of reward processing on mood

fluctuations, focusing on reward prediction errors (RPEs), which are known to be linked to mood fluctuations. This mechanistic study aims at uncovering the process through which these psychopathologies are associated with mood modulations. The authors were able to appropriately test their hypothesis and obtained results corroborating their conclusions.

This work provides a convincing demonstration of the relevance of computational psychiatry (Huys et al, 2016) and the use of decision neuroscience to shed light on the interplay of anxiety, depression, and mood.

Strengths:

The authors used a tripartite model to distinguish depression vs anxiety, as well as a computational model distinguishing reward expectation (EV in the model) from outcome processing through RPE, which are two sequential cognitive processes.

The manuscript adequately addresses the concerns one would have regarding risk-attitudes and regarding referring to trending statistical results.

Weaknesses:

The sample size of the clinical sample (N=116) may not be sufficient to detect anxiety-specific effects due to the high rate of comorbid anxious depression. It would be beneficial to include the number of MDD vs GAD vs anxious depression diagnoses in the clinical population, as this would likely shine light on the power limitations.

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Reviewer #3 (Public review):

Summary:

In this submission, Wang and colleagues jointly examine the association between depression and anxiety symptoms and individuals' affective reactivity to reward prediction errors in Ruttledge et al.'s gambling paradigm. Taking a bifactor approach to anxiety and depression in several non-clinical (and one clinical sample), the authors find that anxiety-specific symptoms relate to over-reactivity of mood to reward prediction errors (RPEs) as well as heightened mood variability, while depression-specific symptoms relate to blunted mood sensitivity to RPEs. These depression- but not anxiety-specific relationships replicated in patient samples.

Strengths:

I was impressed that the data-driven, transdiagnostic approach employed by the authors uncovered specific relationships between anxiety and depression-specific factors and RPE reactivity in a well characterized task and computational model, especially in a non-clinical sample. This sheds new light on how these affective processes may be perturbed-and importantly, in different ways-by anxiety and depression symptoms. Likewise, the replication of the depression-specific finding (RPE hypo-reactivity) in a clinical sample was nice to see.

Weaknesses:

(1) While the anxiety- and depression-specific factors had differential effects on mood variability (Figure 2A-D) and RPE reactivity (Figure 2E-G) in all samples, such that the correlations between the two factors and these mood parameters were significantly different, the anxiety factor was not consistently (significantly) associated with either mood-related parameter across samples. However, the authors resolve anxiety-specific predictive effects when they collapse across datasets. While it is intuitive that achieving a larger effective

sample size would afford the power necessary to detect such individual differences, this struck me as a major caveat for this set of results.

(2) The authors observe associations between the 'common factor' of depression and anxiety and risk-attitude tendencies, presumably the alpha (exponent) parameter in a prospect theory-type subjective value model. But where is this analysis explained? (i.e. how was this model formulated and how were risk attitude parameters estimated?) And what is the interpretation of this finding - is there precedent for looking at risk attitudes in this task? And why would these predictive effects only be observed in relation to the common, but not unique, factors of anxiety and depression?

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