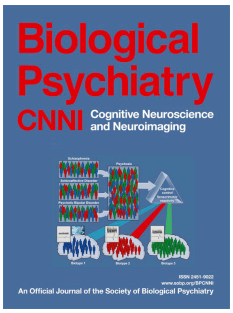


Adaptive suppression of motivational biases increases with mood/anxiety traits

Vanessa Scholz, Johannes Algermissen, Mojtaba Rostami Kandroodi, Claire Gillan, Hanneke Eveline Maria den Ouden

PII: S2451-9022(26)00175-8

DOI: https://doi.org/10.1016/j.bpsc.2026.06.001

Reference: BPSC 1627

To appear in: *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging*

Received Date: 23 May 2025

Revised Date: 1 June 2026

Accepted Date: 5 June 2026

Please cite this article as: Scholz V., Algermissen J., Kandroodi M.R., Gillan C. & Maria den Ouden H.E., Adaptive suppression of motivational biases increases with mood/anxiety traits, *Biological Psychiatry: Cognitive Neuroscience and Neuroimaging* (2026), doi: https://doi.org/10.1016/j.bpsc.2026.06.001.

Adaptive suppression of motivational biases increases with mood/anxiety traits

Vanessa Scholz[1,2], Johannes Algermissen[1,3], Mojtaba Rostami Kandroodi[1,4], Claire Gillan [5,6,7], Hanneke Eveline Maria den Ouden[1]

[1]Donders Institute for Brain, Cognition and Behaviour, Radboud University, Nijmegen, The Netherlands

[2] Department of Child and Adolescent Psychiatry, Psychosomatics and Psychotherapy, Centre of Mental Health, University of Würzburg, Germany

[3] Department of Experimental Psychology, University of Oxford, UK

[4] Department of Cognitive Science and Artificial Intelligence, Tilburg University, Tilburg, The Netherlands

[5] School of Psychology, Trinity College Dublin, Dublin, Ireland

[6] Trinity College Institute for Neuroscience, Trinity College Dublin, Dublin, Ireland

[7] FutureNeuro SFI Research Centre, Trinity College Dublin, Dublin, Ireland

Corresponding authors:

Hanneke den Ouden
Thomas van Aquinostraat 4,
6525 GD Nijmegen, the Netherlands
hanneke.denouden@donders.ru.nl

Vanessa Scholz
Magarete Hoeppel Platz 1
97080 Wuerzburg, Germany
scholz_v@ukw.de

Running title: Increased bias suppression in mood-anxiety symptoms

Keywords: motivational bias, decision-making, depression and anxiety, compulsivity, computational psychiatry

Words: 4502
Figures: 4
Tables: 1

Abstract

Background: Changes in motivational biases appear central to many psychiatric disorders including depression, anxiety, and substance abuse. Motivational biases, theorized to embody default response strategies helpful in uncertain or novel situations, invigorate responding when promised rewards and hold us back facing punishment. However, it remains unclear how specific changes in biases are to certain disorders. Here, we relate motivational biases to continuous, dimensional measures of psychiatric symptoms focusing on depression and anxiety (AD) and compulsions and intrusive thoughts (CIT).

Methods: We recruited 500 participants online from the general population, who performed a motivational Go/NoGo task, a global measure of cognitive ability (I-CAR), and clinical questionnaires. We used factor analysis, mixed-effects models and computational modelling to link transdiagnostic symptom dimensions to motivational biases.

Results: Higher AD symptoms predicted better performance on trials where action requirements are not aligned with motivational biases, requiring their suppression. In contrast, higher CIT scores were associated with overall worse performance, likely reflecting a more general performance deficit. Higher AD symptoms were linked to a more frequent expression of a novel computational model capturing the trial-by-trial up-and-downregulation of the impact of motivational biases. The opposite was found for the CIT dimension.

Discussion: The dissociable patterns for AD and CIT suggest dimension-specific alterations in motivational bias rather than reflecting a generalized psychopathology factor. We speculate that AD individuals, when required to exert effort, might recruit excessive cognitive effort for suppressing motivational biases. This more 'controlled' mode of decision-making might improve performance, yet also exhaust participants and contribute to symptoms.

Introduction

Decision-making involves learning which actions lead to desired outcomes and which ones help to avoid harmful events. However, in addition to such adaptively learnt responses, cues or contexts signaling the valence of prospective outcomes also shape responding in a Pavlovian manner (1–4). Pavlovian action biases, e.g. invigoration in a rewarding context and inhibition in a punishment context, have been proposed to provide sensible 'default' actions that are helpful for example in novel environments, or when speed is of the essence (5). Behaviour is regulated by an intricate interplay of these Pavlovian and instrumental controllers (5). Most of the time, these controllers concur, so that their interaction is cooperative (3,6). For example, a goal-directed approach to obtain a reward is enhanced by Pavlovian invigoration elicited by the reward cue (7,8). However, sometimes, Pavlovian response tendencies are at odds with optimal, learnt responses, leading to interference with instrumental control (9,10). Such Pavlovian interference can lead to detrimental consequences and has recently been implicated in the aetiology and prognosis of several psychiatric disorders including addiction, depression and personality disorders (11–15).

Many psychiatric disorders are characterized by rigid, stimulus-driven behaviours that individuals fail to exert control over. Examples include drug craving in addiction and substance abuse, repetitive behaviours in obsessive-compulsive disorders, or reflexive avoidance behaviour in anxiety and depression. These behaviours can be interpreted as instances of altered motivational biases, and various branches of human and animal research have proposed phenomena akin to motivational biases to lie at the roots of these psychiatric disorders. For example, the phenomenon of sign-tracking in which animals approach reward-predictive cues rather than rewards themselves, might reflect aberrantly strong appetitive biases (16,17). It has been linked to alcohol and drug addiction in both animals (18–20) and humans (11,21,22). Conversely, individuals with anhedonia lack the automatic attraction by such appetitive cues (23,24). Indeed, past research has shown lower motivational biases in depression (25,26), with relatively conserved biases predicting eventual remission (26). In the aversive domain, stronger inhibition induced by aversive cues has been observed in social anxiety (27,28) and depression (29), suggesting that motivational biases might underlie freezing and withdrawal behaviour in these disorders (30,31). Conversely, pathologically strong invigoration of behaviour by aversive cues has been observed in PTSD (32) and suicidal ideation (33). In sum, various psychiatric disorders show altered stimulus-driven behaviour as reflected in motivational biases. As such, these biases may constitute a valuable neurocognitive test bed to better understand these disorders.

In order to suppress motivational biases in situations in which they are maladaptive, individuals need to recruit cognitive control (2,34,35), a process considered to be effortful. In choices between different levels of effort, depressed individuals typically avoid more effortful options (23,30,36–38). Still, when actually obliged to perform cognitively demanding tasks, depressed individuals paradoxically invest more effort and use slower, more deliberate and effortful strategies compared to healthy controls (39–42). This overexertion may help explain why they tend to avoid such tasks, as they might realistically anticipate them to be particularly strenuous given their tendency to overinvest cognitive effort. However, it remains unclear why they adopt these effort-intensive strategies in the first place. Hence, apart from alterations in motivational biases themselves, psychiatric patients might also differ in the amount of control they exert to suppress these biases.

Given that motivational biases are prominent in various psychiatric disorders, the question arises whether these psychiatric disorders share a common underlying trait that is specifically associated with altered motivational bias, or whether they arise under high levels of psychopathology more generally. In other words, patients suffering from mental health problems might show unspecific increased reliance on low-resource-intensive default behavioural strategies and failure to override such default strategies when necessary (43). Alternatively, what initially appears as a lack of disease specificity might in fact arise from the presence of distinct clusters of symptoms in multiple psychiatric disorders

(44–46). Rather than making categorical distinctions between psychiatric disorders as currently defined by diagnostic systems such as DSM-5 (47) or ICD-10 (48), changes in motivational biases may perhaps be better conceptualized as relating to a distinct (group of) symptoms that is common to several disorders, such as apathy or compulsivity.

In this study, we investigated the link between the ability to adaptively control motivational biases and previously defined transdiagnostic psychiatric symptom dimensions capturing mood/anxiety symptoms and impulsive/compulsive symptoms. For this purpose, we recruited a large online sample that completed a previously used questionnaire battery (49). In addition, they completed a well-established probabilistic Motivational Go/NoGo Learning Task that reliably elicits motivational biases in choice behaviour and reaction times. Participants had to learn by trial and error to execute (Go) or withhold (NoGo) a response to acquire a desired outcome. Some cues offered the chance to win a reward ("Win" cues) while others signaled the need to avoid a punishment ("Avoid" cues). Participants typically show better performance for bias-congruent cues (i.e. perform a Go response to Win cues and a NoGo response to Avoid cues) than to bias-incongruent cues (NoGo to Win cues, Go to Avoid cues) (50,51). We quantified motivational biases as the relative performance difference between bias-congruent vs. incongruent cues. Relating individual differences in motivational biases to two transdiagnostic symptom dimensions, namely anxiety/depression (AD) vs. compulsions and intrusive thoughts (CIT), we observed a double dissociation: On the one hand, increased anxious-depressed symptoms predicted stronger suppression of motivational biases for incongruent cues, improving performance specifically for these more difficult conditions. This pattern of behaviour was captured by a novel, exploratory computational model, in which the value of up-and downregulating the Pavlovian choice biases is itself learned. In contrast, higher CIT symptoms were associated with a nonspecific performance deficit across both bias-congruent and incongruent task cues and with reduced expression of this model. This dissociable pattern argues against a unitary, generalized psychopathology account and instead suggests that alterations in motivational bias reflect dimension-specific computational mechanisms.

Materials and Methods

Participants

We recruited 500 participants via Prolific (https://www.prolific.ac.uk) constituting a general-population sample not specifically pre-screened for clinical criteria. Participants indicated English as first language and had (corrected to) normal vision. Participation in previous studies precluded inclusion. Participants were reimbursed with a base rate of £5.83/hour for study completion and could earn a performance-based bonus of up to £1.00. Before participation, all subjects provided informed consent and study procedures were approved by the Ethics Review Committee, Faculty of Social Sciences, Radboud University (ID:17U.003546). Briefly, participants completed a Motivational Go/NoGo (MGNG) Task, a reversal learning task (to be reported elsewhere), demographic questions, a subset of the International Cognitive Ability Resource (I-CAR) Test, a cognitive ability test (52), and a battery of clinical questionnaires (Fig.1). A final sample of n=481 participants remained (4% exclusion rate; demographic details in Table 1) (see SI and preregistration https://osf.io/nht4u/ for more details on exclusion and quality assessment).

MGNG task

The well-established MGNG task robustly evokes motivational biases (50,51). On each trial, a cue was shown and participants had to decide whether to respond (Go) or not (NoGo). For each cue, participants could either receive a reward (Win cues), or avoid loss (Avoid cues) (Fig.1). Feedback followed a probabilistic schedule with validity set to 75%. Participants completed two blocks of 200 trials (four cues/block), presented in pseudorandomised order with max 2 cue repetitions, which took 25–30 minutes to complete (details see SI).

Cognitive Test and Clinical Questionnaires

Participants completed the I-CAR, (63) containing 16 items from four areas (see Fig1). I-CAR scores were used to ensure associations with psychopathology were not accounted for by differences in cognitive ability. We did not enforce any exclusion criteria based on ICAR scores, given that the ICAR does not provide standardized norms or cut-off values (c.f. 63). Participants then completed a well-established questionnaire battery (49), with a preregistered addition of the Almost Perfect Scale (APS) (54) and Padua Inventory (59). Together, these questionnaires assessed a range of clinical symptoms focusing on mood-anxiety and compulsive symptoms (Table 1). It also contained two catch items to assess compliance. Information on clinical relevance of the assessed symptoms is reported in Table S1.

---------- INSERT FIG 1 HERE ----------

Figure 1 Task Design and Effects. A) Experimental timeline for the online experiment. The total time to complete the experimental procedure was approximately 75 minutes, but depended on the self-paced questionnaires. **B)** Go NoGo task trial sequence for each of the four cue types: The Go2Win and NoGo2Avoid cues are bias-congruent, as their instrumental action requirement is in line with the stimulus-response coupling dictated by the motivational bias. Go2Avoid and NoGo2Win are bias-incongruent and usually harder for participants to learn. On each trial, a cue is presented for 1300 ms. During this window, subjects can decide to make a Go response by pressing a button or make a NoGo response by withholding a response. After a 200 ms. blank screen, subjects are presented with the outcome (reward, neutral, punishment). The inter-trial-interval (ITI) varied within the range of 750-1250 ms. in steps of 250ms. **C)** Trial-by-trial probability of Go responses p(Go) across participants (±SEM) for Go cues (solid lines) and NoGo cues (dashed lines) visualized with a 5-trial sliding average. From trial 1, participants show a clear tendency to make more Go responses for Win cues compared to Avoid punishment cues (green lines above red lines) reflecting the motivational bias. Successful learning is evident from the increasing difference in p(Go) for Go relative to NoGo cues. **D-F)** Average performance across participants (± 1 SEM) for each cue condition, grey dots indicate individuals. **D**) The proportion of Go responses for Go cues is higher compared to NoGo cues, showing that participants successfully learned the task, and higher for Win than Avoid cues, reflecting the motivational bias. **(E)** Proportion of correct responses for congruent vs incongruent cues illustrate the degree to which motivational bias hampers responding on incongruent trials. Note that, p(Go) and accuracy, p(Correct), are identical for Go cues, but are the inverse for NoGo cues. (**F**) Reaction times. Participants are faster for correct (relative to incorrect) Go responses, indicating learning, as well as faster to make Go responses for Win (relative to Avoid) cues, reflecting the motivational bias.

Data Analysis

Transdiagnostic factors

Many clinical mental health questionnaires show strong inter-correlations reflecting an overlap of underlying constructs (49,63). Employing factor analysis (FA) for the purpose of dimensionality reduction can alleviate this problem by identifying distinct latent constructs. As pre-registered, we added the APS (which has been related to Obsessive-Compulsive symptoms (67,68)), and Padua Inventory (PI) (59). Rationale for this addition was to dissociate obsessive and compulsive symptoms to reveal putative distinct associations with motivational bias. Unfortunately, the addition of these 62 items led to so-called 'bloated' specific factors, which can emerge when one facet is over-represented, here the OCD domain (69). We therefore report results from using the factor solution from the established questionnaire set. For completeness, results using the 'extended' FA with bloated factors are reported in the SI (*supplementary questionnaire effects and dimension scores*). Importantly, the conclusions remain the same.

Task Behaviour

We analyzed the effect of cue valence (Win/Avoid) and required action (Go/NoGo) on the probability of making a Go response, using a generalized logistic mixed-effects model (R package lme4, v1.1-21). Here, the *Required Action (ReqAct)* factor quantifies learning (i.e. Go when Go is optimal). The *Valence* factor quantifies motivational bias (i.e. more Go to Win cues relative to Avoid cues). Their interaction term captures the degree to which performance varies with valence. Age and ICAR covariates were included to control for potential effects of age and cognitive performance. All continuous variables were z-standardized and factors sum-to-zero coded. All models included a full random effects structure. We computed p-values using likelihood ratio tests (LRT, R: afex package). Statistical

significance was defined as p-values<0.05. We excluded trials with RT<200 ms., where cues are unlikely processed meaningfully. As secondary analysis, we assessed the same model for RT data using linear mixed-level regressions. Here, we expected faster correct than incorrect Go responses, and faster responding to Win than Avoid cues.

Relation between psychiatric symptoms and task behaviour

As pre-registered, we assessed how psychiatric symptom dimensions related to behaviour by adding z-standardized factor scores as between-subject predictors to the mixed-effects models for choice and RT. As factors could be correlated, i.e. competing for variance, we examined models with i) each factor entered separately and ii) all factor scores in a combined model. The latter analysis allowed examination of the unique variance each dimension explained (see SI for RT and extended factor analysis results). We post-hoc characterized the contribution of each questionnaire on observed effects.

Computational modelling

We employed reinforcement learning models to more precisely dissect motivational bias and learning processes underlying interindividual differences in behaviour associated with symptom dimensions. We initially fitted five RL models of increasing complexity to capture learning and bias effects, following previous work but going beyond what was originally specified in our pre-registration (see below) (Swart et al., 2017). For details on model specification, fitting and comparison, see SI. The SI also contains a summary of the results for the pre-registered model-space and the rationale for diverting.

Having established the winning model in this model space, we added a final exploratory model (M6). This model is inspired by the proposal that exerting cognitive control is not so much dependent on the *ability* to exert control, but rather results from evaluating the future net benefits of exerting cognitive control (72). The key idea here is that making a bias-congruent response is computationally cheap, while making a bias-incongruent response requires suppression of (i.e. control over) the Pavlovian system. In M6, the 'value' of bias 'control' was learnt on a trial-by-trial basis for each stimulus through simple RL, governed by a 'control' learning rate. See SI for full model space equations, comparison, validation and parameter transformations.

We analysed how individual symptom scores on each dimension were linked to i) the model responsibility scores for the winning model, and ii) the individual parameter estimates from the winning computational model, using linear regression models. Model responsibility refers to the likelihood of each model within a model space to have generated the observed data, thus providing a relative measure of evidence (70). Linear regressions included individual dimension scores as independent variables, and parameter estimates or model responsibility as DV.

Results

Motivational biases and learning

Participants learnt the task, i.e. made more Go to Go than NoGo cues (ReqAct:$\chi^2(1)=638.7$, $p< .001$; Fig.1C,D). They also showed a motivational bias, i.e. more Go to Win than Avoid cues (Valence: $\chi^2(1)=276.1$, $p<0.001$). Their bias was stronger for Go cues (ReqActxValence: $\chi^2(1)=27.36$; $p<0.001$; Valence (Go): $\chi^2(1)=258.41$, $p<0.001$; Valence (NoGo): $\chi^2(1)=196.68$, $p<0.001$). Older participants performed better (AgexReqAct: $\chi^2(1)=50.8$, $p<0.001$), but did not significantly differ in motivational bias (AgexValence: $\chi^2(1)=2.8$, $p=0.09$). Cognitive ability positively predicted performance and negatively predicted motivational bias (CognAbxReqAct: $\chi^2(1)=90.4$, $p<0.001$; CognAbxValence: $\chi^2(1)=34.4$, $p<0.001$). Remaining effects were non-significant (p-values>0.4) (for details see SI).

Participants were faster for correct Go responses (ReqAct: $\chi^2(1)=274.8$, $p<0.001$, Fig.1F). This likely reflects that participants speed up responding when they are certain what to do, in line with

observations reduced choice certainty is associated with slowing (73). RTs also reflected motivational bias, as faster responses to Win cues (Valence: $\chi^2(1)=456.00$, $p<0.001$) and a significant interaction for ReqActxValence ($\chi^2(1)=18.5$, $p<0.001$).

----------INSERT FIG2 HERE----------

Figure 2. Clinical questionnaires and symptom dimensions A) Distribution of scores on individual questionnaires and demographic variables. **B)** Covariance matrix showing the cross-correlation of all questionnaire items included in the factor analysis **C)** Eigenvalue plot indicating a 3-factor solution for the factor analysis. **D)** Questionnaire item loadings across the identified clinical symptom factors.

Transdiagnostic factors and prediction of choice and reaction times

We replicated the previously described three-factor solution with dimensions Anxiety and Depression (AD), Compulsions and Intrusive Thoughts (CIT), and Social Withdrawal (SW) (Fig.2) (75). Factor weights of previous (49) and current work correlated highly ($r_{min}=0.85$; $r_{max}=0.95$). Below, we examine the association between clinical dimensions, motivational biases and learning. Full statistics are reported in the SI (Table S1-2)

Anxiety-Depression factor

AD significantly interacted with valence and required action (ReqActxValencexAD; $\chi^2(1)=7.31$, $p=0.007$). Breaking down this interaction showed that higher AD symptoms predicted better performance on bias-incongruent trials (NoGo2Win: $\chi^2(1)=10.4$, $p=0.001$; Go2Avoid: $\chi^2(1)=3.7$, $p=0.054$; Go2Win: $\chi^2(1)=1.3$, $p=0.3$; NoGo2Avoid: $\chi^2(1)=0.8$, $p=0.4$). To characterize directly this effect in terms of performance on congruent versus incongruent trials, we analysed accuracy as DV, with cue congruency and AD dimension scores. There was a significant (negative) association of accuracy and AD for bias-incongruent cues ($\chi^2(1)=9.1$, $p= .003$), but not congruent cues ($\chi^2(1)=0.6$, $p= .4$) (Fig 3A-B).

Increased AD symptoms were associated with a larger RT difference for correct versus incorrect responses (ReqActxAD: $\chi^2(1)=6.7$, $p=0.001$), driven by stronger slowing for incorrect responses with higher symptoms (AD (NoGo): $\chi^2(1)=4.5$, $p=0.03$; AD(Go): $\chi^2(1)=0.03$, $p=0.9$;) suggesting increased uncertainty about incorrect responses.

Compulsions and Intrusive Thought factor

Participants with higher CIT scores were less likely to perform the required action (ReqActxCIT: $\chi^2(1)=15.3$, $p<0.001$, Fig.3A,B), but symptom severity was not associated with motivational biases (ValencexCIT: $\chi^2(1)=0.1$, $p=0.7$) nor the interaction (ReqActxValencexCIT: $\chi^2(1)=2.69$, $p=0.1$; Fig.3B). In contrast to AD symptoms, higher CIT symptoms were associated with a *smaller* difference in RT for correct versus incorrect responses (ReqActxCIT: $\chi^2(1)=4.9$, $p=0.02$), driven by slower responding for correct responses with increasing symptoms (CIT (Go): $\chi^2(1)=4.2$, $p=0.04$; CIT (NoGo): $\chi^2(1)=0.03$, $p=0.9$). This corroborates findings in the choice domain (i.e. worse performance with increasing symptoms), as higher CIT individuals appear more uncertain of correct choices.

Social Withdrawal factor

Paralleling the finding for AD, there was a significant 3-way interaction for SW (ReqActxValencexSW: $\chi^2(1)=6.3$, $p=0.01$), again following a pattern of slightly better performance for incongruent relative to congruent cues (Fig.3A). This effect is numerically strongest for Win cues, though no individual conditions reached significance (p-values >.2: Go2Win: $\chi^2(1)=1.7$, $p=0.2$; Go2Avoid: $\chi^2(1)=0.1$, $p=0.7$; NoGo2Win: $\chi^2(1)=1.7$, $p=0.2$; NoGo2Avoid: $\chi^2(1)=0.31$, $p=0.6$) (Fig.3). There were no other significant interactions with SW (all $p>0.3$). There were also no significant RT effects for SW.

Remaining effects of dimensions were non-significant (p > .2) (Fig.3). Note that excluding ICAR scores from the models did not change our key findings (ReqActxValencexAD: $\chi^2(1)=7.2$, p=0.008, ReqActxCIT: $\chi^2(1)=25.7$, p<0.001, ReqActxValencexSW: $\chi^2(1)=6.2$, p=0.013). Briefly, all effect that had been significant in the previous models remained significant, and non-significant effects remained non-significant.

Individual questionnaire loading

In the primary analyses, we observed that AD symptoms were (positively) predictive of performance on incongruent but not congruent trials, while CIT symptoms predicted both (negatively). As a follow-up analysis we asked which individual questionnaires were driving these effects. For this analysis, we simplified the ReqActxValence interaction into a simple congruent vs. incongruent metric, allowing for reduction of model complexity, improving statistical power and interpretability. This particularly helps these analyses at the level of individual questionnaires, where measurement noise is likely higher and effect sizes smaller compared to latent symptom dimensions.

Here, particularly higher apathy and trait anxiety scores were significantly linked to higher task performance on bias-incongruent trials, in absence of a concurrent increase for bias-congruent cues (Fig.3A, see Table S5 for detailed statistics). Meanwhile, worse task performance across congruent and incongruent cues was significantly linked to clinical symptoms from questionnaires encompassing eating disorder symptoms and OCD (Fig.3A). Others showed no significant effects.

---------- INSERT FIG 3 HERE ----------

Figure 3. Dimension and questionnaire effects A) Transdiagnostic factors predict congruency-dependent performance. Separate visualization of ß estimates from mixed-effects models with p(correct) as dependent variable and z-standardized questionnaire scores, age and cognitive score as independent variables for congruent and incongruent cues. Increased AD symptoms were linked to better task performance specifically on bias-incongruent cues. In contrast, more CIT symptoms were associated with poorer performance across bias-congruent and incongruent cues. Effects of the SW dimension were similar to the AD dimension, though weaker and non-significant. B) **Raw behavioural data for choices and reaction times (RT)**, as a function of symptom dimension (tertile split of Low, Medium, High symptom scores) for accuracy and mean corrected RT. RTs are split up by correct (Go cues) and incorrect (NoGo cues) responses. For the AD and SW dimension, there is a significant slowing on incorrect Go responses with more symptoms, leading to an increased difference between correct and incorrect Go responses. In contrast, increasing CIT symptoms was associated with slowing on correct Go responses, reducing the RT difference between correct and incorrect responses. **C) Association of individual questionnaires with task performance.** Asterisks indicate significance levels of the ß effect estimates. *** p< 0.001, ** p< 0.01, * p<0.05, ° > 0.05. For more statistics see table S2 and S3 in the SI.

Model-based analysis

The base model comparison [M1-M5] identified M5 as winning model (model frequency=40%, PXP=0.84, Fig.4), encompassing a Go bias (*b*), Pavlovian bias (π) two feedback sensitivity parameters (ρ_{win}, ρ_{avoid}). Extending M5 with a learning rate for learning the value of exerting control (i.e., suppressing the motivational bias, M6) improved the model, both when comparing within the full model space (M1-M6, Fig.4A), and when compared directly to M5 (M6: PXP=1.0, model frequency=62.1 %; Fig.4A, Table.S7; see SI and Fig.S1 for parameter recovery). In terms of absolute model performance, M6 predicted the observed responses 72% of the time (vs. 50% chance).

Using HBI-derived model responsibility as a continuous outcome, we examined whether symptom dimension scores predicted responsibility for models M6 and M5, based on responsibility extracted from the full model space. Model M6 allows for adaptively learning the value of modulating the impact of the Pavlovian bias on behavior, thus reducing the motivational bias for incongruent trials over trials (Fig.4B). This feature may enable M6 to better capture improved performance on incongruent trials for high-scoring AD individuals. Higher AD scores were associated with greater responsibility for model M6 (AD: ß=0.076, t(472)= 3.6, p<0.001) and lower responsibility for model M5 (AD: ß=-0.056, t(472)= -2.9, p=0.005). Meanwhile, CIT scores showed the opposite pattern, predicting lower responsibility for

M6 (CIT: ß=-0.062, t(472)= -3.1, p=0.002) and higher responsibility for M5 (CIT: ß=0.057, t(472)= 3.1, p=0.002). Effects for the SW dimension were non-significant but followed the same direction as AD (p>.3). Full correlations between all dimension scores with parameter estimates are reported in the SI.

Discussion

In this study, we adopted a dimensional approach using online-data collection (46) in a general-population sample to investigate how aberrant motivational biases in decision-making manifests across psychiatric symptom dimensions (27–29,32,75). Specifically, we asked whether alterations in motivational biases in psychiatric disorders reflect associations with distinct transdiagnostic symptom dimensions, or instead are correlates of a generalized illness factor that leads to non-specific impairments in adaptive responding across various cognitive dimensions. Under the latter account, motivational bias parameters would be expected to show associations that are uniform in direction across symptom dimensions, consistent with a single latent factor influencing behavior. In contrast, dimension-specific effects would be reflected in dissociable—and potentially opposing—associations across symptom domains.

Our data revealed a double dissociation consistent with a symptom domain-specific effect on motivational bias, such that (1) AD symptoms were associated with improved performance on trials requiring bias suppression, whereas (2) CIT was associated with overall performance deficits. This pattern of associations was dissociable across dimensions in terms of both the nature (condition-specific) and direction (increased versus decreased performance). Hence, it is difficult to reconcile this with a generalized illness factor explanation, and instead supports the interpretation that changes in motivational bias suppression are specific to the domain of anxiety-depression symptoms, and thus may reflect alterations in cognitive computations within this symptom domain.

Factor analysis of a broad set of psychiatric symptom questionnaires collected in this large population sample replicated previous findings, identifying three primary symptom dimensions: anxiety-depression (AD), compulsivity-intrusive thoughts (CIT), and social withdrawal (SW). Surprisingly, we found that participants reporting increased AD symptoms appeared to learn to weigh the motivational bias more efficiently so that they could appropriately maintain it in bias-congruent trials, while appropriately dampening it in bias-incongruent trials. This effect was also observed, albeit to a lesser extent, in individuals with higher SW symptoms. In contrast, CIT symptoms were linked to general performance deficits across all task conditions.

Decreased performance or ‘decision acuity’ in individuals with high psychiatric symptom severity is a common phenomenon found in many reward learning tasks (76). It might reflect both cognitive limitations (e.g. concentration difficulties) and motivational problems that preclude the recruitment of sophisticated strategies needed to solve these tasks. Here, we observed such generalized task impairment for the CIT dimension. In contrast, a *higher* AD symptom score was associated with *increased* performance on bias-incongruent trials, which demand suppression of Pavlovian response tendencies (2,34,35), but was unaltered on congruent trials. This dissociation of CIT and AD symptoms was present in both choice and RT data.

The fact that the relationship between AD symptoms and improved performance was specific to bias-incongruent trials suggests that AD individuals do not simply show global suppression of motivational bias. Such global suppression would have resulted in reduced performance on congruent trials, as we observed in previous work (50). Rather, our finding suggests that what distinguishes higher anxious-depressive individuals is the ability to suppress biases when they are maladaptive (77,78). This notion is supported by an exploratory computational model allowing for the trial-by-trial up/down regulation of the Pavlovian bias suppression for congruent/incongruent cues respectively. This model was more likely to be the best model in individuals with higher AD scores, but less likely in those with higher CIT scores.

The reduced biases observed with higher AD symptoms are consistent with prior findings of lower biases in depressed patients using a Pavlovian-to-instrumental transfer paradigm, where relatively intact biases predicted future remission (26), though see (79). Other previous research has linked depression to effort avoidance, i.e. preferential choice of low-effort options, which suggests that depressed individuals prefer to exert less cognitive control (23,38,80,81). In seeming contrast, other studies suggests that when tasks are relatively easy, depressed patients exert more control (39–42). These findings highlight the possibility that effort avoidance and increased effort exertion may reflect a shared cognitive attitude of perceiving tasks as overly difficult and requiring dedicated effort and diligence. In line with this, research has shown that depressed individuals are more sensitive to effort costs (37), and under certain circumstances, may perform better than healthy controls. For example, apathy has been linked to improved task performance in a vigilance and threat detection task (82). Likewise, higher scores for trait anxiety predicted better performance when making decisions under ambiguity during stress (83) and better inference of hidden states (84). In sum, these results suggest that individuals with higher AD scores may be better capable of up- and down-regulating motivational biases when these are maladaptive. Importantly, the present findings should not be taken to suggest that anxiety and depression are interchangeable. Rather, the AD dimension captures variance shared across these domains, with apathy and anxiety loadings being most strongly linked to the motivational effects we discovered. Future work will be needed to replicate our findings and disentangle their potentially distinct influences on motivational bias.

Improved regulation of Pavlovian biases in individuals scoring higher on the AD dimension might stem from heightened risk aversion and an overly cautious, deliberative style of responding. Overestimating the potential of failure, even for relatively easy tasks, might motivate these individuals to over-exert control to suppress their biases when realizing they might result in poor performance. As such, behavior may be less guided by stimuli signaling potential rewards, underlying loss of pleasure and interest, core aspects of anhedonia (24,85). Outsourcing behavior to simple default strategies such as motivational biases can be healthy and adaptive in everyday life (86), but anxious-depressive individuals might not benefit from this possibility. Similarly, less guidance by stimuli signaling threats of punishment might lead to unexpected negative experiences and an increased sense of worry and caution (87,88). Lastly, while cognitive control recruitment is typically conducive to solving problems in everyday life and thus to a positive mental health status, chronic overexertion of cognitive control can become maladaptive (89) and might be linked to long-term consequences such as fatigue (90), apathy (91) or increased risk of relapse in depression (91). Future studies should further explore the clinical relevance of inter-individual differences in controlling motivational biases for treatment outcome.

Interestingly, one ‘transdiagnostic’ clinical feature that depression and anxiety share is rumination (92). Rumination can be construed as extensive recruitment of cognitive effort to think through a decision strategy. It might reflect the inability to rely on simple heuristics—like motivational biases—to make decisions instead relying on more effortful, deliberate control mechanisms (87,93). In our data, the severity of anxious-depressive symptoms correlated with slower response times, particularly for incorrect responses, which further supports the idea that rumination and the suppression of motivational biases might reflect an abnormally high recruitment of cognitive effort. At the same time, slower RT might also represent a more cautious approach with reduced confidence regarding choice selection, as previously reported in other studies (94,95). Thus, improvement in adaptive Pavlovian bias suppression could speculatively be construed as cognitive “side-effects” of depressogenic processes such as rumination. Interestingly, a previous study also reported better task performance (decision acuity) with increasing levels of worrying (96). While this explanation is tempting, our study did not explicitly examine a direct association with rumination and future studies are needed including validated rumination measures to test whether rumination mediates the link between adaptive suppression and depressive symptoms. Furthermore, while this suggests potential clinical significance of our findings, it is important to note that we did not collect data on participants' medical histories,

medications, or prior diagnoses, meaning we cannot draw conclusions about how these factors might have influenced the results.

In summary, using FA, mixed-effects models, and computational modeling, we replicate and extend previous research on transdiagnostic symptom dimensions in psychiatric disorders. Specifically, we find a double dissociation for symptom dimensions, namely, that anxious-depressive individuals are better able to suppress Pavlovian biases when they are maladaptive, whereas individuals with compulsive/intrusive symptoms show more generalized performance deficits. Our results suggest that previously observed wide-spread changes in motivational biases in psychiatric disorders may reflect both a general performance deficit and symptom-specific bias modulation. We speculate that lower reliance on simple default strategies and chronic recruitment of cognitive control to suppress biases might contribute to symptoms such as apathy or fatigue. Importantly, as the GoNoGo task is not a cognitive effort task and thus not optimized to capture individual differences in cognitive effort exertion in the context of Pavlovian biases, our interpretation should be seen as a potential avenue for future research. Here, it could be explored whether individual differences in cognitive control over motivational biases predict the development or worsening of psychiatric symptoms and how they might relate to cognitive processes like rumination. Moreover, our findings highlight the importance of distinguishing between performance deficits and alterations in computational strategy. Interventions may therefore benefit from targeting not only the magnitude of motivational biases, but also the flexibility with which they are regulated across contexts.

Code and Data availability

Anonymized data and analysis code for this study will be shared publicly upon publication

Acknowledgements:

VS was supported by a postdoctoral research fellowship by the German Research Foundation (SCHO1815/1-1 & SCHO1815/2-1). HdO was supported by a Vidi Innovation Grant from the Dutch Research Council (NWO; 016.Vidi.175.450). CMG is supported by the European Research Council (ERC H2020 HABIT) and Science Foundation Ireland (19/FFP/6418; 21/RC/10294).

The funders have no role in study design, data collection and analysis, decision to publish or preparation of the manuscript.

We thank Wilbert van Ham for help with programming the online version of the Go/NoGo task.

An earlier version of this manuscript was made publicly available as a preprint on PsyArXiv (https://osf.io/preprints/psyarxiv/v3ndf_v1

Authors Contribution

HEMdO, CMG and VS discussed study design. HEMdO and VS designed the paradigm and were responsible for study set-up. VS collected the data. VS, JA and HEMdO were responsible for data analysis. VS, JA, MRK, CMG and HEMdO discussed statistical analyses and results. VS and HEMdO wrote the manuscript. All authors edited the manuscript.

Competing Interests

The authors report no biomedical financial interests or potential conflicts of interest.

Supplement Description:

Supplement Methods, Results, Figures S1-S6, Tables S1-S15

References

1. Swart JC, Froböse MI, Cook JL, Geurts DEM, Frank MJ, Cools R, den Ouden HEM (2017): Catecholaminergic challenge uncovers distinct Pavlovian and instrumental mechanisms of motivated (in)action. *eLife* 6: 1–36.
2. Swart JC, Frank MJ, Määttä JI, Jensen O, Cools R, den Ouden HEM (2018): Frontal network dynamics reflect neurocomputational mechanisms for reducing maladaptive biases in motivated action. *PLoS Biol* 16: e2005979.
3. Dayan P, Niv Y, Seymour B, Daw ND (2006): The misbehavior of value and the discipline of the will. *Neural Netw* 19. https://doi.org/10.1016/j.neunet.2006.03.002
4. Guitart-Masip M, Huys QJM, Fuentemilla L, Dayan P, Duzel E, Dolan RJ (2012): Go and no-go learning in reward and punishment: interactions between affect and effect. *NeuroImage* 62: 154–166.
5. Boureau YL, Sokol-Hessner P, Daw ND (2015): Deciding How To Decide: Self-Control and Meta-Decision Making. *Trends Cogn Sci* 19. https://doi.org/10.1016/j.tics.2015.08.013
6. Boureau Y-L, Dayan P (2011): Opponency revisited: competition and cooperation between dopamine and serotonin. *Neuropsychopharmacol Off Publ Am Coll Neuropsychopharmacol* 36: 74–97.
7. Estes W (1943): Discriminative conditioning. I. A discriminative property of conditioned anticipation. *J Exp Psychol* 32: 150–155.
8. Estes WK (1948): Discriminative conditioning. II. Effects of a Pavlovian conditioned stimulus upon a subsequently established operant response. *J Exp Psychol* 38: 173–177.
9. Hershberger WA (1986): An approach through the looking-glass. *Anim Learn Behav* 14: 443–451.
10. Breland K, Breland M (1966): The misbehavior of organisms. *Am Psychol* 16. https://doi.org/10.1037/h0040090
11. Garbusow M, Schad DJ, Sebold M, Friedel E, Bernhardt N, Koch SP, *et al.* (2016): Pavlovian-to-instrumental transfer effects in the nucleus accumbens relate to relapse in alcohol dependence. *Addict Biol* 21: 719–731.
12. Hallquist MN, Hall NT, Schreiber AM, Dombrovski AY (2018): Interpersonal dysfunction in borderline personality: a decision neuroscience perspective. *Curr Opin Psychol* 21. https://doi.org/10.1016/j.copsyc.2017.09.011
13. Heinz A, Schlagenhauf F, Beck A, Wackerhagen C (2016): Dimensional psychiatry: mental disorders as dysfunctions of basic learning mechanisms. *J Neural Transm Vienna Austria 1996* 123: 809–821.
14. Krypotos AM, Engelhard IM (2020): Pavlovian-to-instrumental transfer in subclinical obsessive–compulsive disorder. *J Exp Psychopathol* 11. https://doi.org/10.1177/2043808720925244
15. Huys QJM, Schad D, Deserno L, Sebold M, Garbusow M, Smolka M, *et al.* (2015): Neurobehavioural predictors of relapse in alcohol use disorders. *Biol Psychiatry* 77.
16. Colaizzi JM, Flagel SB, Joyner MA, Gearhardt AN, Stewart JL, Paulus MP (2020): Mapping sign-tracking and goal-tracking onto human behaviors. *Neurosci Biobehav Rev* 111: 84–94.
17. Robinson TE, Yager LM, Cogan ES, Saunders BT (2014): On the motivational properties of reward cues: Individual differences. *Neuropharmacology* 76: 450–459.
18. Flagel SB, Akil H, Robinson TE (2009): Individual differences in the attribution of incentive salience to reward-related cues: Implications for addiction. *Neuropharmacology* 56: 139–148.
19. Flagel SB, Robinson TE (2017): Neurobiological basis of individual variation in stimulus-reward learning. *Curr Opin Behav Sci* 13: 178–185.
20. Tomie A, Grimes KL, Pohorecky LA (2008): Behavioral Characteristics and Neurobiological Substrates Shared by Pavlovian Sign-Tracking and Drug Abuse. *Brain Res Rev* 58: 121–135.
21. Schad DJ, Rapp MA, Garbusow M, Nebe S, Sebold M, Obst E, *et al.* (2020): Dissociating neural learning signals in human sign- and goal-trackers. *Nat Hum Behav* 4. https://doi.org/10.1038/s41562-019-0765-5

22. Schettino M, Ceccarelli I, Tarvainen M, Martelli M, Orsini C, Ottaviani C (2022): From skinner box to daily life: Sign-tracker phenotype co-segregates with impulsivity, compulsivity, and addiction tendencies in humans. *Cogn Affect Behav Neurosci* 22: 1358–1369.
23. Husain M, Roiser JP (2018): Neuroscience of apathy and anhedonia: a transdiagnostic approach. *Nat Rev Neurosci* 19: 470–484.
24. Treadway MT, Zald DH (2011): Reconsidering anhedonia in depression: Lessons from translational neuroscience. *Neurosci Biobehav Rev* 35: 537–555.
25. Ging-Jehli NR, Kuhn M, Blank JM, Chanthrakumar P, Steinberger DC, Yu Z, *et al.* (2024): Cognitive Signatures of Depressive and Anhedonic Symptoms and Affective States Using Computational Modeling and Neurocognitive Testing. *Biol Psychiatry Cogn Neurosci Neuroimaging* 9: 726–736.
26. Huys QJM, Gölzer M, Friedel E, Heinz A, Cools R, Dayan P, Dolan RJ (2016): The specificity of Pavlovian regulation is associated with recovery from depression. *Psychol Med* 46: 1027–1035.
27. Mkrtchian A, Aylward J, Dayan P, Roiser JP, Robinson OJ (2017): Modeling Avoidance in Mood and Anxiety Disorders Using Reinforcement Learning. *Biol Psychiatry* 82: 532–539.
28. Peterburs J, Albrecht C, Bellebaum C (2022): The impact of social anxiety on feedback-based go and nogo learning. *Psychol Res* 86: 110–124.
29. Nord CL, Lawson RP, Huys QJM, Pilling S, Roiser JP (2018): Depression is associated with enhanced aversive Pavlovian control over instrumental behaviour. *Sci Rep* 8: 1–10.
30. Blanchard DC (2017): Translating dynamic defense patterns from rodents to people. *Neurosci Biobehav Rev* 76: 22–28.
31. Roelofs K, Dayan P (2022): Freezing revisited: coordinated autonomic and central optimization of threat coping [no. 9]. *Nat Rev Neurosci* 23: 568–580.
32. Ousdal OT, Huys QJ, Milde AM, Craven AR, Ersland L, Endestad T, *et al.* (2018): The impact of traumatic stress on Pavlovian biases. *Psychol Med* 48: 327–336.
33. Millner AJ, den Ouden HEM, Gershman SJ, Glenn CR, Kearns JC, Bornstein AM, *et al.* (2019): Suicidal thoughts and behaviors are associated with an increased decision-making bias for active responses to escape aversive states. *J Abnorm Psychol* 128: 106–118.
34. Algermissen J, Swart JC, Scheeringa R, Cools R, Den Ouden HEM (2022): Striatal BOLD and midfrontal theta power express motivation for action. *Cereb Cortex* 32: 2924–2942.
35. Cavanagh JF, Eisenberg I, Guitart-Masip M, Huys Q, Frank MJ (2013): Frontal theta overrides pavlovian learning biases. *J Neurosci* 33: 8541–8548.
36. Treadway MT, Bossaller NA, Shelton RC, Zald DH (2012): Effort-based decision-making in major depressive disorder: A translational model of motivational anhedonia. *J Abnorm Psychol* 121: 553–558.
37. Vinckier F, Jaffre C, Gauthier C, Smajda S, Abdel-Ahad P, Le Bouc R, *et al.* (2022): Elevated Effort Cost Identified by Computational Modeling as a Distinctive Feature Explaining Multiple Behaviors in Patients With Depression. *Biol Psychiatry Cogn Neurosci Neuroimaging* 7: 1158–1169.
38. Valton V, Mkrtchian A, Moses-Payne M, Gray A, Kieslich K, VanUrk S, *et al.* (2025): A computational approach to understanding effort-based decision-making in depression. *Psychol Med* 55: e302.
39. Brinkmann K, Gendolla GHE (2008): Does depression interfere with effort mobilization? Effects of dysphoria and task difficulty on cardiovascular response. *J Pers Soc Psychol* 94: 146–157.
40. Bustamante LA, Barch DM, Solis J, Oshinowo T, Grahek I, Konova AB, *et al.* (2024, February 20): Major depression symptom severity associations with willingness to exert effort and patch foraging strategy. https://doi.org/10.1101/2024.02.18.24302985
41. Cléry-Melin M-L, Schmidt L, Lafargue G, Baup N, Fossati P, Pessiglione M (2011): Why Don't You Try Harder? An Investigation of Effort Production in Major Depression ((J. Laks, editor)). *PLoS ONE* 6: e23178.
42. Silvia PJ, Mironovová Z, McHone AN, Sperry SH, Harper KL, Kwapil TR, Eddington KM (2016): Do depressive symptoms "blunt" effort? An analysis of cardiac engagement and withdrawal for an increasingly difficult task. *Biol Psychol* 118: 52–60.

43. Otto AR, Raio CM, Chiang A, Phelps EA, Daw ND (2013): Working-memory capacity protects model-based learning from stress. *Proc Natl Acad Sci U S A* 110. https://doi.org/10.1073/pnas.1312011110
44. Caspi A, Houts RM, Belsky DW, Goldman-Mellor SJ, Harrington H, Israel S, *et al.* (2014): The p Factor: One General Psychopathology Factor in the Structure of Psychiatric Disorders? *Clin Psychol Sci J Assoc Psychol Sci* 2: 119–137.
45. Caspi A, Moffitt TE (2018): All for one and one for all: Mental disorders in one dimension. *Am J Psychiatry* 175: 831–844.
46. Wise T, Robinson O, Gillan C (2022): Identifying transdiagnostic mechanisms in mental health using computational factor modeling. *Biol Psychiatry*. https://doi.org/10.1016/j.biopsych.2022.09.034
47. American Psychiatric Association (2013): *Diagnostic and Statistical Manual of Mental Disorders (5th Ed.)*. Washington, DC: Author.
48. World Health Organisation (1993): *The ICD-10 Classification of Mental and Behavioural Disorders.*
49. Gillan CM, Kosinski M, Whelan R, Phelps EA, Daw ND (2016): Characterizing a psychiatric symptom dimension related to deficits in goal-directed control. *eLife* 5. https://doi.org/10.7554/eLife.11305%2520PM%2520%2520-%252026928075%2520M4%2520%2520-%2520Citavi
50. Scholz V, Hook RW, Kandroodi MR, Algermissen J, Ioannidis K, Christmas D, *et al.* (2022): Cortical dopamine reduces the impact of motivational biases governing automated behaviour. *Neuropsychopharmacology* 47. https://doi.org/10.1038/s41386-022-01291-8
51. van Nuland AJ, Helmich RC, Dirkx MF, Zach H, Toni I, Cools R, den Ouden HEM (2020): Effects of dopamine on reinforcement learning in Parkinson's disease depend on motor phenotype. *Brain* 143. https://doi.org/10.1093/brain/awaa335
52. Condon DM, Revelle W (2014): The international cognitive ability resource: Development and initial validation of a public-domain measure. *Intelligence* 43. https://doi.org/10.1016/j.intell.2014.01.004
53. Marin RS, Biedrzycki RC, Firinciogullari S (1991): Reliability and validity of the Apathy Evaluation Scale. *Psychiatry Res* 38: 143–162.
54. Slaney RB, Rice KG, Mobley M, Trippi J, Ashby JS (2001): The revised almost perfect scale. *Meas Eval Couns Dev* 34: 130–145.
55. Saunders JB, Aasland OG, Babor TF, de la Fuente JR, Grant M (1993): Development of the Alcohol Use Disorders Identification Test (AUDIT): WHO Collaborative Project on Early Detection of Persons with Harmful Alcohol Consumption--II. *Addict Abingdon Engl* 88: 791–804.
56. Patton JH, Stanford MS, Barratt ES (1995): Factor structure of the Barratt impulsiveness scale. *J Clin Psychol* 51: 768–774.
57. Garner DM, Olmsted MP, Bohr Y, Garfinkel PE (1982): The eating attitudes test: psychometric features and clinical correlates. *Psychol Med* 12: 871–878.
58. Liebowitz MR (1947): Social Phobia. pp 141–173.
59. Burns GL, Keortge SG, Formea GM, Sternberger LG (1996): Revision of the Padua Inventory of obsessive compulsive disorder symptoms: Distinctions between worry, obsessions, and compulsions. *Behav Res Ther* 34: 163–173.
60. ZUNG WWK (1965): A Self-Rating Depression Scale. *Arch Gen Psychiatry* 12: 63.
61. Mason O, Linney Y, Claridge G (2005): Short scales for measuring schizotypy. *Schizophr Res* 78: 293–296.
62. Spielberger CD, Gorsuch R, Lushene R, Vagg PR, Jacobs GA (1983): *Manual for the State-Trait Anxiety Inventory.* Consulting Psychology Press M4 - Citavi.
63. Rouault M, Seow T, Gillan CM, Fleming SM (2018): Psychiatric symptom dimensions are associated with dissociable shifts in metacognition but not task performance. *Biol Psychiatry*. https://doi.org/10.1016/j.biopsych.2017.12.017

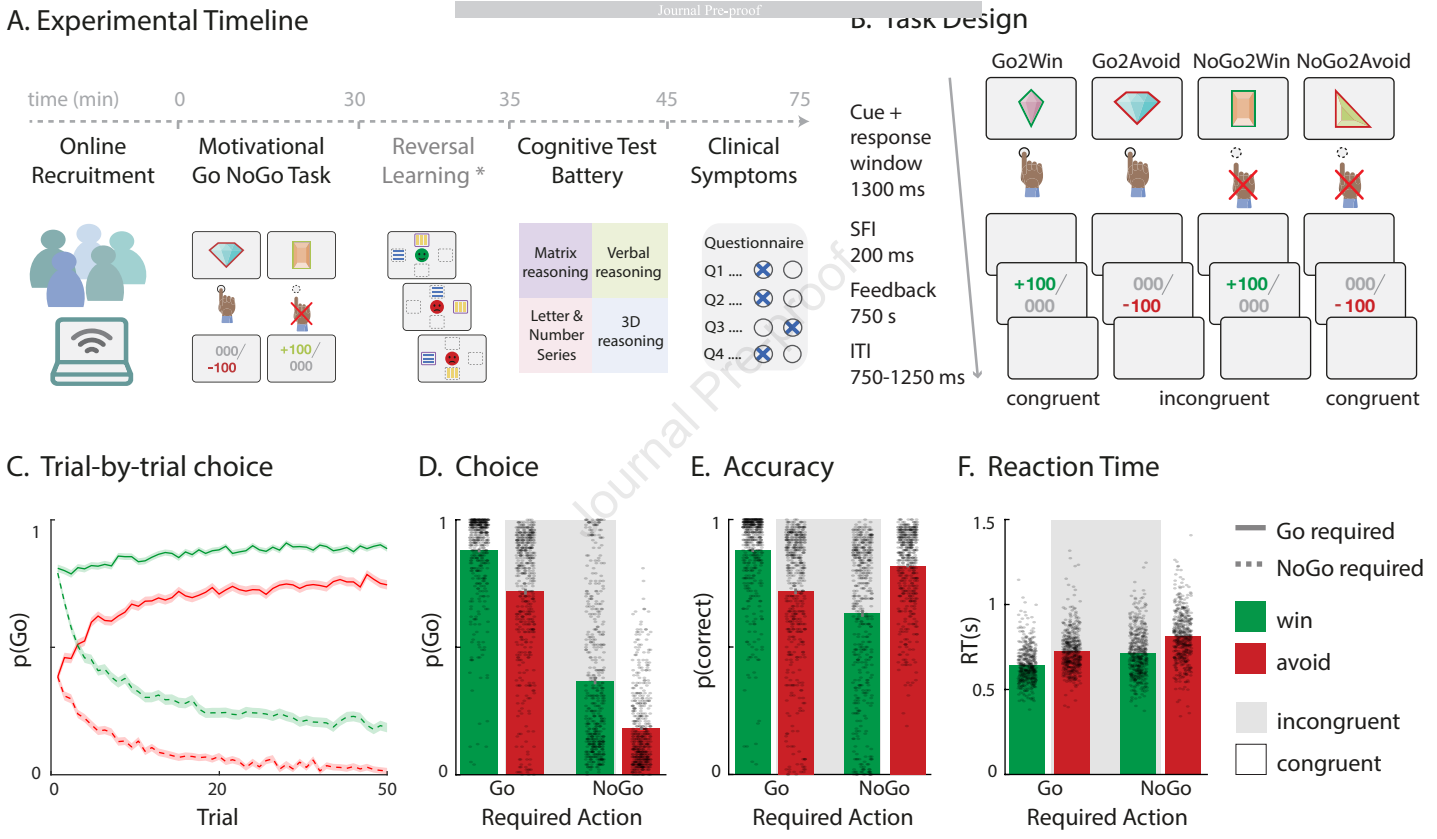
64. Gillan CM, Kalanthroff E, Evans M, Weingarden HM, Jacoby RJ, Gershkovich M, *et al.* (2020): Comparison of the Association between Goal-Directed Planning and Self-reported Compulsivity vs Obsessive-Compulsive Disorder Diagnosis. *JAMA Psychiatry* 77: 77–85.
65. Patzelt EH, Kool W, Millner AJ, Gershman SJ (2019): Incentives Boost Model-Based Control Across a Range of Severity on Several Psychiatric Constructs. *Biol Psychiatry* 85: 425–433.
66. Revelle W (2018): psych: Procedures for Personality and Psychological Research,Northwestern University, Evanston, Illinois, USA,https://cran.r-project.org/package=psych Version = 1.8.12. Northwestern University, Evanston, Illinois, USA. Retrieved from https://cran.r-project.org/package=psych Version = 1.8.12
67. Rice KG, Pence SL (2006): Perfectionism and Obsessive-Compulsive Symptoms. *J Psychopathol Behav Assess* 28: 103–111.
68. Rhéaume J, Ladouceur R, Freeston MH (2000): The prediction of obsessive–compulsive tendencies: does perfectionism play a significant role? *Personal Individ Differ* 28: 583–592.
69. Oltmanns JR, Widiger TA (2018): Maladaptive Variants of Adaptive Traits and Bloated Specific Factors. *J Res Personal* 76: 177.
70. Piray P, Dezfouli A, Heskes T, Frank MJ, Daw ND (2019): Hierarchical Bayesian inference for concurrent model fitting and comparison for group studies. *PLoS Comput Biol* 15: e1007043.
71. Rescorla RA, Wagner AR (1972): A Theory of Pavlovian Conditioning: Variations in the Effectiveness of Reinforcement and Nonreinforcement. *Clasical Cond II Curr Res Theory*.
72. Lieder F, Shenhav A, Musslick S, Griffiths TL (2018): Rational metareasoning and the plasticity of cognitive control. *PLoS Comput Biol* 14: 1–27.
73. Kiani R, Corthell L, Shadlen MN (2014): Choice Certainty Is Informed by Both Evidence and Decision Time. *Neuron* 84: 1329–1342.
74. de Boer L, Axelsson J, Chowdhury R, Riklund K, Dolan RJ, Nyberg L, *et al.* (2019): Dorsal striatal dopamine D1 receptor availability predicts an instrumental bias in action learning. *Proc Natl Acad Sci U S A* 116: 261–270.
75. Albrecht MA, Waltz JA, Cavanagh JF, Frank MJ, Gold JM (2016): Reduction of Pavlovian bias in schizophrenia: Enhanced effects in clozapine-administered patients. *PLoS ONE* 11: 1–23.
76. Eckstein MK, Master SL, Dahl RE, Wilbrecht L, Collins AGE (2022): Reinforcement learning and Bayesian inference provide complementary models for the unique advantage of adolescents in stochastic reversal. *Dev Cogn Neurosci* 55. https://doi.org/10.1016/j.dcn.2022.101106
77. Guitart-Masip M, Duzel E, Dolan R, Dayan P (2014): Action versus valence in decision making. *Trends Cogn Sci* 18: 194–202.
78. McNaughton N, Gray JA (2000): Anxiolytic action on the behavioural inhibition system implies multiple types of arousal contribute to anxiety. *J Affect Disord* 61: 161–176.
79. Moutoussis M, Rutledge RB, Prabhu G, Hrynkiewicz L, Lam J, Ousdal OT, *et al.* (2018): Neural activity and fundamental learning, motivated by monetary loss and reward, are intact in mild to moderate major depressive disorder. *PLoS ONE* 13. https://doi.org/10.1371/journal.pone.0201451
80. Bonnelle V, Manohar S, Behrens T, Husain M (2016): Individual Differences in Premotor Brain Systems Underlie Behavioral Apathy. *Cereb Cortex N Y NY* 26: 807–819.
81. Treadway MT, Buckholtz JW, Schwartzman AN, Lambert WE, Zald DH (2009): Worth the 'EEfRT'? The Effort Expenditure for Rewards Task as an Objective Measure of Motivation and Anhedonia. *PLOS ONE* 4: e6598.
82. Trier H, O'Reilly JX, Spiering L, Ma S, Kolling N, Rushworth M, Scholl J (2023, May 4): Emotions and individual differences shape foraging under threat. PsyArXiv. https://doi.org/10.31234/osf.io/v6u3y
83. Yang Y, Yan B, Sun K, Wu D, Wang C, Xiao W (2024): Trait Anxiety Leads to "Better" Performance? A Study on Acute Stress and Uncertain Decision-Making. *Behav Sci* 14: 1186.
84. Zika O, Wiech K, Reinecke A, Browning M, Schuck NW, De Z-BM (2022): Trait anxiety is associated with hidden state inference during aversive reversal learning. *bioRxiv* 5700: 2022.04.01.483303.
85. Hall AF, Browning M, Huys QJM (2024): The computational structure of consummatory anhedonia. *Trends Cogn Sci* 28: 541–553.

86. Algermissen J, den Ouden HEM (2023): Goal-directed recruitment of Pavlovian biases through selective visual attention. *J Exp Psychol Gen* 152: 2941–2956.
87. Huys QJM, Daw ND, Dayan P (2015): Depression: a decision-theoretic analysis. *Annu Rev Neurosci* 38: 1–23.
88. Huys QJM, Eshel N, Nions EO, Sheridan L, Dayan P, Jonathan P (2012): Bonsai Trees in Your Head : How the Pavlovian System Sculpts Goal-Directed Choices by Pruning Decision Trees. 8. https://doi.org/10.1371/journal.pcbi.1002410
89. Bustamante L, Lieder F, Musslick S, Shenhav A, Cohen J (2021): Learning to Overexert Cognitive Control in a Stroop Task. *Cogn Affect Behav Neurosci* 21: 453–471.
90. Müller T, Apps MAJ (2019): Motivational fatigue: A neurocognitive framework for the impact of effortful exertion on subsequent motivation. *Neuropsychologia* 123: 141–151.
91. Berwian I, Wenzel J, Collins A, Seifritz E, Stephan K, Walter H, Huys Q (2020): Computational mechanisms of effort and reward decisions in depression and their relationship to relapse after antidepressant discontinuation. *JAMA Psychiatry* 513–522.
92. McLaughlin KA, Nolen-Hoeksema S (2011): Rumination as a transdiagnostic factor in depression and anxiety. *Behav Res Ther* 49: 186–193.
93. Dayan P, Huys QJM (2008): Serotonin, inhibition, and negative mood. *PLoS Comput Biol* 4: e4.
94. Zakay D, Tuvia R (1998): Choice latency times as determinants of post-decisional confidence. *Acta Psychol (Amst)* 98: 103–115.
95. Dildine TC, Necka EA, Atlas LY (2020): Confidence in subjective pain is predicted by reaction time during decision making. *Sci Rep* 10: 21373.
96. Moutoussis M, Garzón B, Neufeld S, Bach DR, Rigoli F, Goodyer I, *et al.* (2021): Decision-making ability, psychopathology, and brain connectivity. *Neuron* 109: 2025-2040.e7.

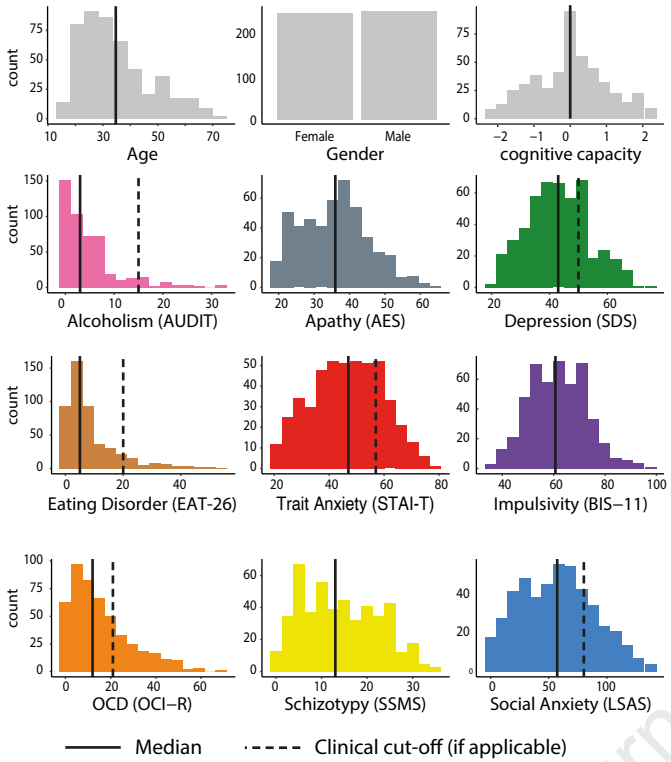
	Mean	SD	Range
Demographic Data (N=481)			
Age	34.7	12.7	18 - 75
Cognitive Score	8.5	3.6	0 - 16
Gender (% female)	50.3		
Educational degree completed (%)			
Primary school	3.1		
Secondary School	45.3		
Bachelor degree	36.4		
Master degree	11.2		
Advanced degree	3.9		
Clinical Questionnaires			
AES	35.8	9.6	18 - 63
APS	108.4	21.0	25 - 157
AUDIT	5.0	5.8	0 - 32
BIS-11	61.6	11.2	35 - 97
EAT [a]	8.4	9.6	0 - 54
LSAS [b]	57.2	32.0	0 - 137
OCI-R	15.7	13.2	0 - 69
SCZ	14.2	8.5	0 - 35
SDS	43.2	10.6	20 - 75
STAI-T	46.5	12.9	20 - 78
PI	26.3	24.5	0 - 115

696

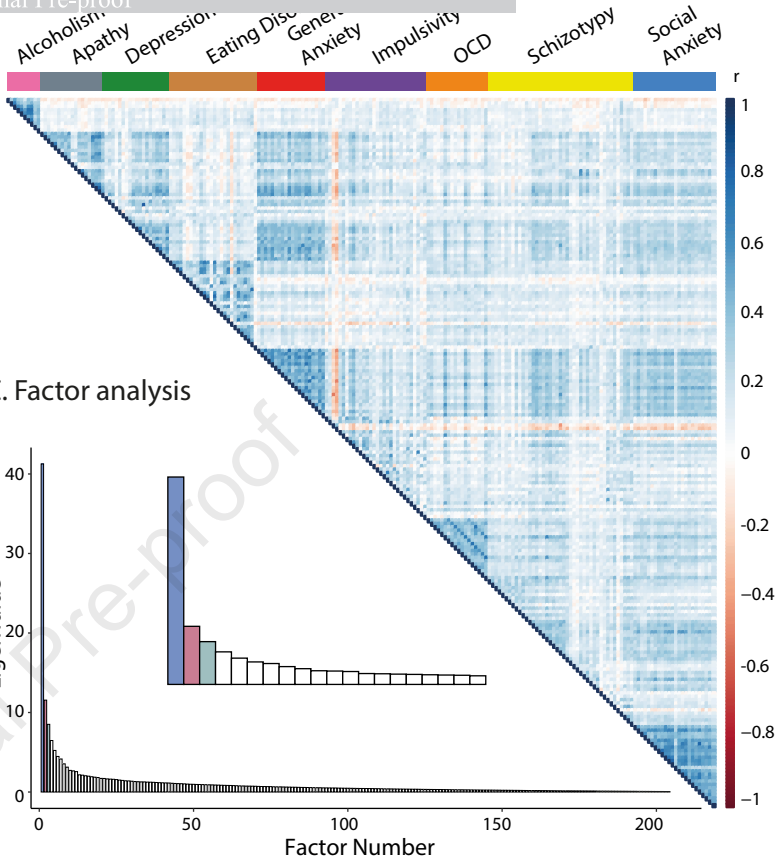
Table 1.



A. Distribution Questionnaire scores

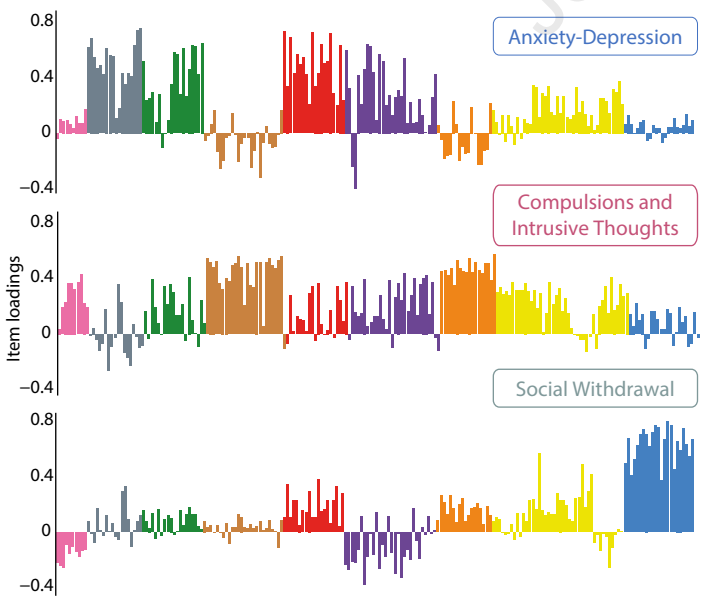


B. Item-level covariance matrix



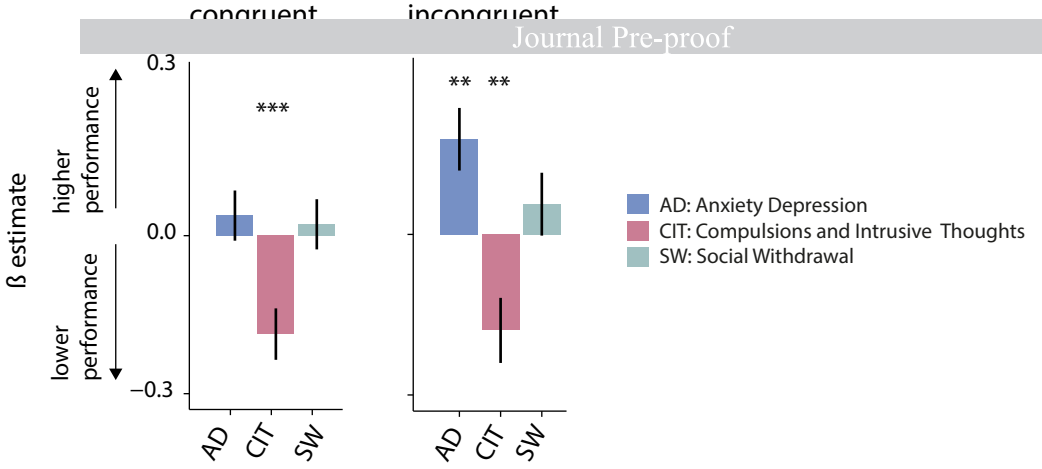
C. Factor analysis

D. Symptom Dimensions

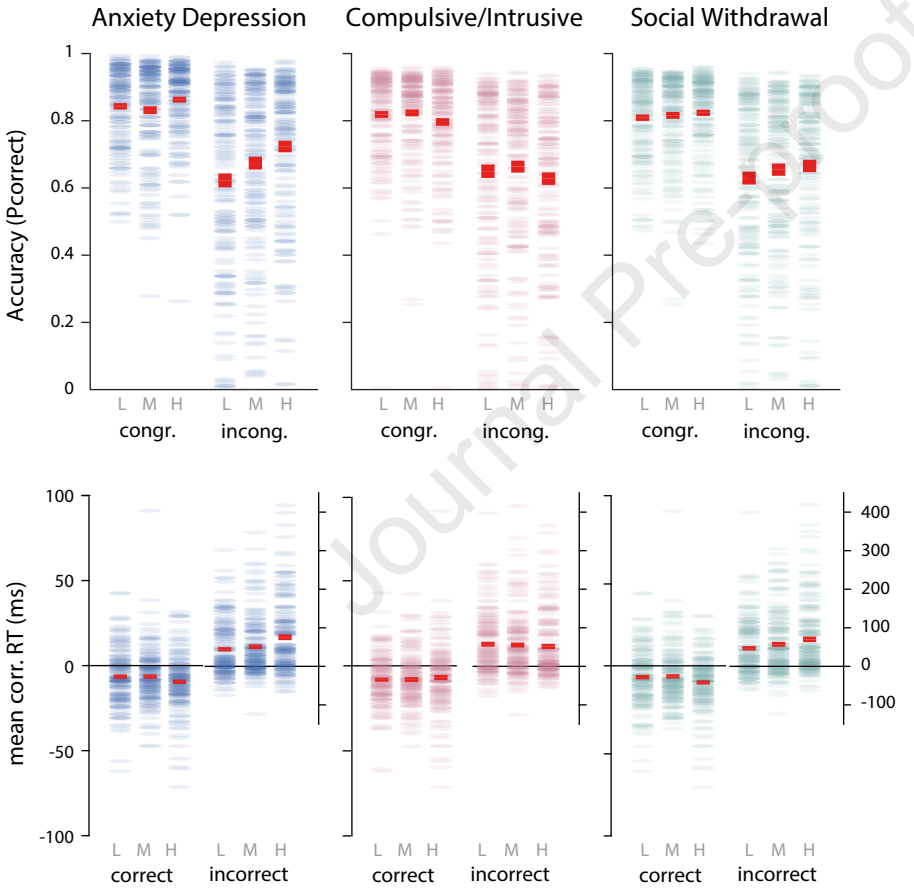


- Alcoholism (AUDIT)
- Apathy(AES)
- Depression (SDS)
- Eating Disorder (EAT-26)
- Trait Anxiety (STAI)
- Impulsivity (BIS-11)
- OCD (OCI-R)
- Schizotypy (SSMS)
- Social Anxiety (LSAS)

A. Factors predict congruency-dependent performance



B. Raw behavioural data by factor tertiles



C. Individual questionnaire loadings on task performance