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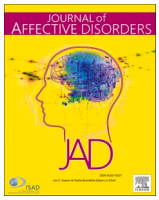

Research paper

Depression history and deficits in executive control following stress: A role for the expected value of control

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ABSTRACT

Deficits in executive control following stress have been proposed to drive depression-related dysfunction. Current theories of these executive control deficits in depression suggest these deficits reflect deficits in implementing control. However, aberrant computations of the value of said control can mimic a deficit in control, even in the absence of a deficit. The current study aimed to determine the mechanisms underlying depression-related deficits in executive control task performance following stress. To that end, individuals with a verified history of major depressive disorder (MDDh) and matched control participants without a history of psychopathology completed a modified Stroop task following an acute stressor. We took a multimethod, converging operations approach using fMRI and a novel cognitive model to assess how MDDh relates to both control-related (right inferior frontal gyrus; rIFG) and affective-related processes involved in the computation of control (dorsal anterior cingulate cortex; dACC) during executive control task performance following stress. We found depression-history-related differences in dACC but not rIFG responses to Stroop conflict. Further, computational modeling and control-related self-reported affect suggested that MDDh-related differences in task performance following stress were consistent with differences in computing the expected value of control. Our results suggest that depression-history-related deficits in executive control task performance following stress may reflect aberrant computations of the value of control.

1. Introduction

Poor executive function is a well-documented characteristic of depression (Snyder, 2013; Snyder et al., 2019), and the link between poor executive function and depression is even stronger when executive function is assessed under stressful conditions (Quinn and Shields, 2023). The current understanding of these findings is relatively intuitive: Executive control is thought to be critical to mitigating detrimental consequences of stress (Williams et al., 2009). To the extent that someone has poor executive control following stress, their poor control then confers less of an ability to effectively engage in stress-regulatory processes, leading to poorer stress recovery and eventual psychopathology (Quinn and Shields, 2023). The current model thus implies that a true deficit in control following stress exists in individuals at risk for depression (i.e., a diathesis), which then produces the link between poorer executive control following stress and depression. Such findings have led to the development of numerous interventions aiming to improve executive control following stress or similar negatively valenced states in hopes of ameliorating depression or its risk, with mixed results (Hoorelbeke et al., 2022; Jopling et al., 2020; Peckham et al., 2021; Zetsche et al., 2024). In this manuscript, we provide evidence for an alternative potential mechanism to explain the aforementioned findings.

Recent work on the recruitment of executive control provides an alternative explanation for the above-mentioned body of work. Perhaps the most prominent understanding of executive control recruitment is the expected value of control (EVC) theory (Musslick et al., 2015; Shenhav et al., 2021, Shenhav et al., 2013). According to this theory, control is recruited at its optimal expected value: We titrate our control exertion to the level that should provide the greatest value. Holding difficulty constant, affective processes—which are relatively central to depression (Borsboom and Cramer, 2013; Van Borkulo et al., 2015)—modulate the chosen control signal intensity (Grahek et al., 2020). Importantly, however, a lower chosen control signal intensity may appear behaviorally identical to a poorer ability to implement control (Musslick et al., 2015). This finding suggests that atypical affective

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processes could produce behavioral results that are identical to true control deficits in assessments of executive function.

Theoretical work has suggested that depression may be linked to differences in computing the expected value of control (Grahek et al., 2019; see also Cléry-Melin et al., 2011; Treadway et al., 2012), and recent empirical work has shown that stress influences the affective processes used in computing the expected value of control (Shields et al., 2024). Together, these findings provide for an alternative explanation of depression-related differences in executive control following stress: Stress influences affective dynamics used in computing the expected value of control, making it easier to detect latent control-related affective differences in depression following stress. According to this alternative, there may be no actual deficit in control, but a difference in the computation to use said control. To date, however, no study has examined whether differences in processes underlying computation of the expected value of control explain depression-related differences in executive control following stress.

Testing these competing hypotheses requires methods that can disentangle behavioral task performance beyond traditional measures. One such method is functional magnetic resonance imaging (fMRI). In particular, expected value of control theory was first proposed as an explanation not of behavior, but of dorsal anterior cingulate cortex (dACC) activity (Shenhav et al., 2013). Although the dACC has been implicated in a broad degree of cognitive processes, work has shown that dACC is, at least in part involved in computing the expected value of control (Bush et al., 2022; Shenhav et al., 2013). Considering control implementation, the right inferior frontal gyrus (rIFG) is thought to be in part responsible for the implementing specific forms of executive control which includes inhibition (Friedman and Miyake, 2017; Wessel and Anderson, 2024). Although both of these regions are part of larger intrinsic networks that underlie both executive control and reward processing, there is reason to believe the dACC and rIFG reflect the computation of the expected value of control and control implementation respectively (Bush et al., 2022; Wessel and Anderson, 2024). A second method, providing a converging operations approach, is parameter estimation within computational cognitive modeling. This approach permits quantification of latent cognitive processes of interest via mathematical modeling (Farrell and Lewandowsky, 2018; Shields et al., 2025; Shields et al., 2020; Shields et al., 2019b; Shields et al., 2019a). Some work has suggested that depression related differences in executive control performance are driven by affective processes (Stolicyn et al., 2017), response biases (Dillon et al., 2015), error processing (Steele et al., 2007), and reward learning (Huys et al., 2022). To date, however, no study has applied either of these approaches to determine the mechanisms underlying MDDh-related differences in executive control following stress.

1.1. Current research

Using a novel cognitive model and neuroimaging paradigm, we addressed the gaps described above within the present study. Participants, who either had a diagnosed history of major depressive disorder (MDDh) and met stringent inclusion criteria or were demographics-matched healthy control individuals without any history of psychopathology as determined by DSM-5 criteria, completed a Stroop task following a stressor (i.e., the Maastricht Acute Stress Test) while undergoing functional magnetic resonance imaging (fMRI). Saliva samples were collected and assayed for cortisol to validate the stressor. Modeling was conducted using drift diffusion model (DDM) architecture, which has been well validated for use in the Stroop (White et al., 2018). The DDM framework was chosen over others (e.g., parallel distributed processing; Cohen et al., 1990) because this was the architecture on which other EVC models have been based (Grahek et al., 2020).

Although the alternative hypothesis we have suggested is orthogonal to (and thus not inconsistent with) the current understanding of executive control following stress and depression, these frameworks make unique predictions, which we tested. In particular, according to the current understanding, MDDh individuals should both a) have less control-specific neural activity and b) have a poorer ability to implement or exert control. In contrast, according to the alternative hypothesis that we have put forward, MDDh individuals should a) differ in neural activity related to computing the expected value of control, and b) differ in estimating the expected value of control.

2. Method

2.1. Participants

Sixty participants were recruited from the greater area of Tulsa Oklahoma. Participants were recruited through radio ads, flyers, Facebook, and referrals from local mental health treatment facilities. This was the maximum number of participants we were able to recruit given budgetary constraints. Inclusion criteria are provided in the Supplement. All participants in the MDDh group had a diagnosis of major depressive disorder, and all participants in the healthy control group had no history of any psychopathology as determined by DSM-5 criteria. Diagnoses were determined by a trained professional who administered the Mini-International Neuropsychiatric Interview (MINI) (Sheehan et al., 1998). The MINI was administered prior to the current study and participants were recruited from a participant pool. As such, we refer to participants as having a history of major depressive disorder (i.e., MDDh). Missingness per variable is described in the respective sections below. Sample characteristics are provided in Table 1. Participants were compensated with $90 for completing this study.

2.2. Materials

2.2.1. Stress induction

The Maastricht Acute Stress Test (MAST) was administered to all participants to induce acute stress (Smeets et al., 2012). The MAST is a

Table 1
Sample characteristics.

	MDDh		HC	
	n / *M*	% / *SD*	*n* / *M*	% / *SD*
Group *n*	30	50%	30	50%
Depressive Sx (PHQ-9)	11.0	4.5	1.77	3.2
Age (years)	29.5	7.7	31.9	11.4
Sex				
Male	8	13.3%	10	16.6%
Female	22	36.7%	20	33.3%
Psychotropic Medication				
Yes	4	6.7%	0	0
No	26	43.3%	30	50%
Other M.H. Diagnoses				
None	17	28.3%	30	50%
GAD	7	11.7%	0	0%
Social Anxiety	1	1.7%	0	0%
PTSD	1	1.7%	0	0%
GAD & Social Anxiety	4	6.7%	0	0%
Stroop: Congruent-trial Mean Corr. RT	715.9	102.9	723.5	98.7
Stroop: Incongruent-trial Mean Corr. RT	867.5	165.3	838.4	130.6
Stroop: Congruent-trial Mean Accuracy	0.98	0.02	0.98	0.03
Stroop: Incongruent-trial Mean Accuracy	0.93	0.14	0.94	0.07
Stroop: Congruent-trial Prop. Omissions	0.01	0.01	0.01	0.03
Stroop: Incongruent-trial Prop. Omissions	0.01	0.01	0.02	0.03

Note. PHQ-9 = Patient Health Questionnaire-9, described below; Sx = Symptoms; MDDh = Major Depressive Disorder history; HC = Healthy Controls; M.H. = Mental Health; GAD = Generalized Anxiety Disorder; PTSD = Post Traumatic Stress Disorder; Social Anxiety = Social Anxiety Disorder; *M* = mean; *SD* = standard deviation; Corr = Correct; RT = Response Time; Prop = Proportion. Note that demographics variables within this table provide the demographics of the full sample, not the sample analyzed (i.e., participants with usable Stroop task data); no significant differences emerged in the sample used for analysis. For Stroop variables, the sample was 28 HC and 27 MDDh.

well validated stress induction that elicits a robust stress response (Shields et al., 2017; Smeets et al., 2012). Details are provided in the Supplement.

2.2.2. Manipulation check

Participants provided two saliva samples via passive drool, from which salivary cortisol was assayed. A pre- to post-stressor increase in salivary cortisol is considered the gold-standard stress biomarker (Shields, 2020; Shields et al., 2017). Assay details are provided in the Supplement.

2.2.3. Modified Stroop task

A modified color-word Stroop task that contained a probabilistic affect prompt was used to examine executive control and related affective processes (Shields et al., 2024). Task details are presented in the Supplement. Notably, the task included probabilistic affect prompts that asked participants how they were feeling at specific moments during the task. Affect prompts were anchored from 1 (Not good) to 4 (Very good) (see Fig. 2).

The Stroop RT interference effect (Stroop effect)—calculated by taking the difference in correct incongruent trial and correct congruent trial response times—is the primary behavioral outcome indicative of executive control in the Stroop task. According to the standard model of executive control, the Stroop effect indexes the general, or common, executive control process (Friedman et al., 2008; Karr et al., 2018; Shields and Yonelinas, 2025), though we note that many instead hold that the Stroop effect indexes interference control, which is a process within inhibitory control (e.g., Shields and Yonelinas, 2025). Given numerous meta-analyses (e.g., Snyder, 2013; Snyder et al., 2015) and our prior work (Quinn and Shields, 2023), we had a directional hypothesis regarding executive control: MDDh individuals would show worse executive control performance than healthy controls. We thus chose a one-tailed *t*-test for this test.

Three participants did not complete the Stroop due to technical issues. We excluded two participants: one because they reported in a subsequent task that their button box had been backwards during the scan until that point, and the other because they committed errors in 26% of congruent trials. The participants without usable Stroop data were equally divided between groups (MDDh $n = 27$, HC $n = 28$ with usable Stroop data).

2.3. Computational cognitive modeling

We fit a novel cognitive model that fits within the framework of the expected value of control. Although the expected value of control has been converted to a behavioral model previously (Grahek et al., 2020; Musslick et al., 2015), we chose to develop our own model within this theoretical framework for three reasons. First, the prior model selected the control signal prior to evidence accumulation, but it did so according to trial congruency (Grahek et al., 2020). This is inconsistent with the framework on which this behavioral implementation is based: In diffusion modeling, evidence accumulation includes perceptual evidence, toward a specific linked perceptual-motor decision (Ratcliff et al., 2016; White et al., 2011). Selecting the control signal based upon trial congruency but prior to evidence accumulation is thus somewhat inconsistent with theory behind diffusion modeling of response times. Second, and in part related to the first issue, the parameters underlying attention, computation of the expected value of control, and control implementation were not uniquely identifiable within the prior behavioral model implementation. In particular, because the control signal in the prior model implementation is chosen at the beginning of the trial and operates on a single parameter (i.e., drift rate), within the prior model, goal-directed and automatic attention had to be fixed rather than estimated, as did the reward rate for correct responding.

The above considerations are addressed by two small conceptual differences that changed our approach to implementing EVC within a framework for fitting Stroop task data. First, we permitted control to operate *intratrial*. In other words, control implementation occurred when computations during the evidence accumulation process suggested that the expected value of control was high. Second, drawing on literature suggesting that modulating decision boundary is a control process (Rahnev et al., 2016; Wenzlaff et al., 2011), we allowed control to operate on either drift rate or decision boundary, according to a differential conjunction of reward from fast/correct responses and aversion to making an error vs. exerting control. These time-dependent and differentially influential control valuation processes permit estimation of relevant processes, as they produce unique time- and location-dependent changes in drift diffusion rate and decision boundary parameters.

Because a full description of the model exceeds space limitations, we describe most of our model, including its structure, the fitting procedure, and selection process within the Supplement. In brief, at each step in evidence accumulation, the model estimates with some uncertainty the probability of making a correct response on that trial, and weighs that against the value of responding correctly quickly as well as the aversiveness of committing an error. Probabilistically, as a function of those odds ratios, the model exerts control over either the evidence accumulation drift rate or decision boundary as appropriate. The fitting procedure minimized trial-weighted discrepancies between predicted and actual response time distributions for error and correct trials, as well as discrepancies in predicted and observed trial counts. Our model selection procedure operated across all participants, rather than at the individual participant level. We felt we had only two degrees of freedom: We felt that we could alter the 1) the formulae determining the expected values of control over drift rate and decision boundary, and 2) which function the model used to estimate (with some uncertainty) the probability of making a correct response on that trial, given the current drift, location, etc. We ultimately selected the model with the most predictive validity from estimated parameters—the model where parameters estimating the expected value of control best predicted dACC activity. For complete model details, see Supplement.

This model has four parameters solely related to valuing or computing the expected value of control: *AE*, *RfF*, $\xi_{(0)}$, and κ. First, *AE*, which we conceptualized as aversion to committing an error. Second, *RfF* describes the expected reward from responding quickly and correctly. Third, $\xi_{(0)}$ describes the initial degree of uncertainty within a trial around estimating the amount of control needed for a correct response. Finally, κ describes the within-trial decay rate of that uncertainty.

The model recruits and adjusts control in real time during each trial, via three parameters: β, C^{max}, and θ_C^{scale}. First, β describes the rate of change in either control process when that control process is recruited. Second, C^{max} describes the maximum extent to which goal-directed attentional control can be deployed to bias information processing (i.e., control over drift rate). Finally, θ_C^{scale} is a multiplicative scaling factor for C^{max}, describing the extent to which one can dynamically increase one's own decision boundary (e.g., to avoid committing an error).

This model thus permits disentangling potential depression-history-related differences in control processes from potential differences in computing the expected value of control.

2.4. Functional magnetic resonance imaging

Acquisition details are presented within the Supplement.

2.4.1. Preprocessing and analysis

Functional MRI data used in subsequent analyses were preprocessed using only one deviation from the standard pipeline from fMRIPrep (Esteban et al., 2020, Esteban et al., 2019)—namely, functional file output in MNI152 2009 2 mm space. Quality assurance was conducted on the fMRIprep output as recommended (Esteban et al., 2020, Esteban et al., 2019).

Masks were selected or created for the regions of interest. The mask for the dACC was taken from the networks atlas provided within the CONN toolbox (Whitfield-Gabrieli and Nieto-Castanon, 2012) and resampled to our subject space using nearest neighbor interpolation. The mask for the rIFG was created by manual segmentation of the MNI152 2009 T1 1 mm brain template via ITK-SNAP. The manually segmented ROI included 474 voxels. The ROI definition was a priori, following the anatomical boundaries shown in The Allen Human Brain Neuroanatomic Reference Atlas for orIFG and LOrG, given that the orbital portions of the right inferior frontal cortex in particular seem to underpin inhibitory control task performance (Boen et al., 2022). Voxels were manually selected for inclusion into the mask by GSS and saved out of ITK-SNAP once the mask was complete. The mask was then resampled to our functional file space via cubic spline interpolation, and we then respected the probabilistic components of the ROI via a probability-weighted mean. See Fig. 1.

Our fMRI first-level analysis methods are provided in the Supplement. Finally, we extracted the regression coefficient *t* values (as these values represent the typical beta values divided by their standard error, thus penalizing variability within the effect in the ROI) for both the incongruent/correct and congruent/correct regressors and created a contrast (i.e., the fMRI equivalent of the Stroop effect) for use in analyses.

2.5. Procedure

Participants first provided informed consent. Following consent, participants completed questionnaires to acclimate, after which they provided the first (baseline) saliva sample. Next, participants completed the acute stress manipulation, which lasted 15 min. Following stress induction, participants then briefly practiced several cognitive tasks they would complete in the scanner, which included the modified Stroop. After approximately 15 min from the end of the stress manipulation, participants provided the second saliva sample. Participants were then brought to the scanner; they completed the modified Stroop as their first task once inside. This timeline is consistent with work finding that stress influences cognition up and through 90 min post-stress (Schwabe and Wolf, 2014). See Fig. 2. All procedures were reviewed and approved by the WCG IRB (protocol #20222670).

3. Results

Sex did not moderate any of the following analyses (see syntax/data), *ps* >.110.

3.1. Preliminary analyses

Analyses presented in this section are illustrated within Supplemental Material.

We first examined cortisol responses from pre- to post-stressor to determine whether our stress induction was successful. Participants showed the expected increase in salivary cortisol from pre- to post-stressor, $F(1, 49) = 18.67$, $p < .001$, $\eta_p^2 = 0.28$, with no differences between diagnostic groups at either timepoint (MDDh main effect, $p = .789$, $\eta_p^2 < 0.01$; MDDh×Time interaction, $p = .967$, $\eta_p^2 < 0.01$). We thus successfully induced stress in both groups.

We next examined behavioral performance on the Stroop task in its most common outcome measure: Stroop interference effects. The Stroop effect was present in individuals with MDDh, $t(26) = 8.51$, $p < .001$ and controls, $t(27) = 10.00$, $p < .001$. In comparison to the healthy control participants, participants with a history of MDD showed the expected difference in Stroop effects: MDDh participants ($M = 151.7$ ms, $SE = 15.0$) showed a greater Stroop effect than healthy control participants ($M = 114.8$ ms, $SE = 14.7$), $t(53) = 1.75$, $p = .043$ (one-tailed), $p = .086$ (two-tailed), $d = 0.46$, 95% CI_d: [−1.00, 0.08]. MDDh individuals thus showed a numerically larger Stroop interference effect in the expected direction.

As our final preliminary analysis, we examined whether BOLD signal congruency contrast differed from zero for each of our ROIs within the healthy control participants (i.e., whether our ROIs showed expected Stroop effects in "typical" participants within fMRI studies). As expected, both the dACC ($M_{diff.} = 0.38$, $p = .004$, $d = 0.595$, 95% CI_d: [0.19, 0.99]), and the rIFG ($M_{diff.} = 0.45$, $p = .001$, $d = 0.689$, 95% CI_d: [0.27, 1.10]).

3.2. Expected value of control analyses

3.2.1. fMRI

Drawing on work described above, if depression-history-related differences in executive control performance following stress were driven by differences in computing the expected value of control, we would expect depression-history-related differences in dACC activity to be larger than depression-history-related differences in rIFG activity; conversely, if MDDh differences in performance following stress were driven by differences in ability to implement or exert control, we would expect depression-history-related differences in rIFG activity to be larger than depression-history-related differences in dACC activity. Consistent with both hypotheses, we found a significant Group × ROI interaction for congruency effects, $F(1, 53) = 8.22$, $p = .006$, $\eta_p^2 = 0.13$ (Fig. 3).

Probing this interaction, we found support for the hypothesis that depression-history-related differences in executive control following stress were consistent with differences in computing the expected value

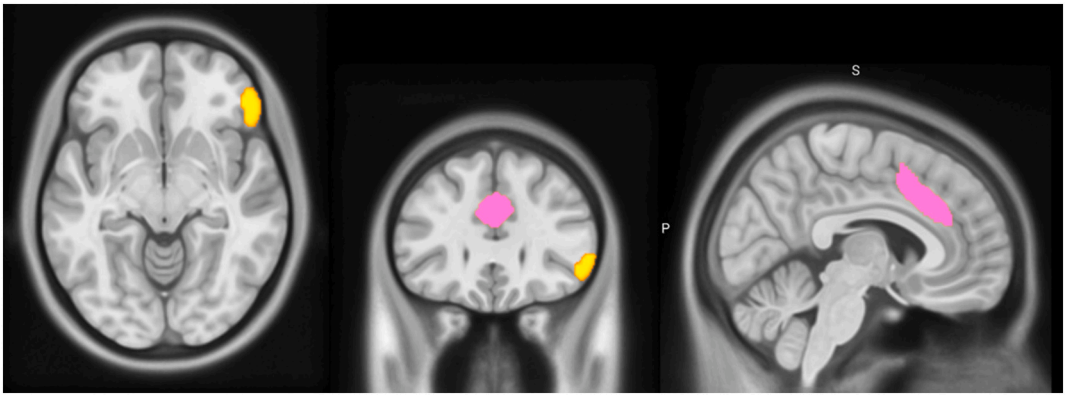

Fig. 1. Regions of interest (ROIs) in neuroimaging analyses
Note. The dACC is depicted in orchid, and the rIFG is depicted in gold. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

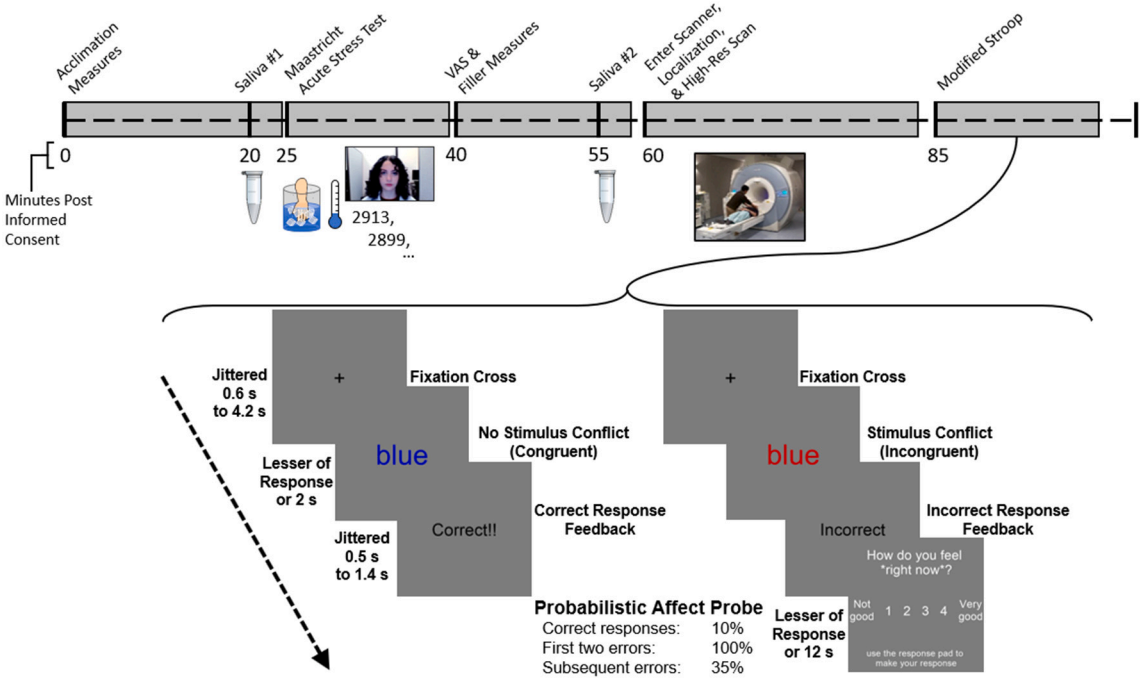

Fig. 2. Study Procedure and Stroop Task.

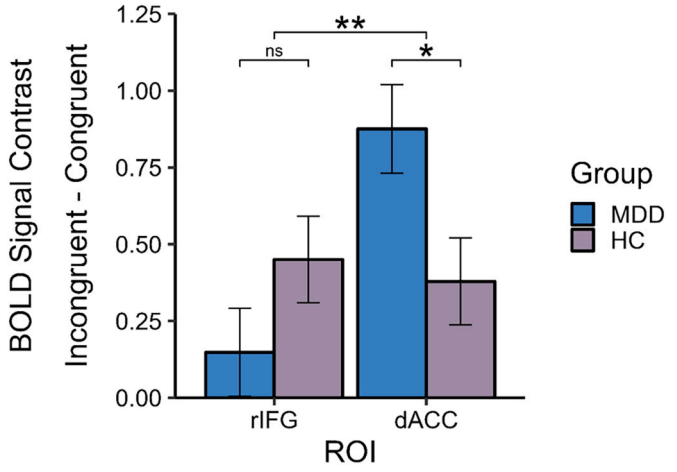

Fig. 3. *fMRI Results*

Note. ns = nonsignificant, *$p < .05$, **$p < .01$; BOLD = blood oxygen level dependent; rIFG = right inferior frontal gyrus; dACC = dorsal anterior cingulate cortex; MDD = history of major depressive disorder; HC = healthy control.

of control. In particular, we found a significant difference between groups in dACC activity, such that—relative to individuals without a history of psychiatric conditions ($M = 0.38$, $SE = 0.14$)—those with MDDh ($M = 0.88$, $SE = 0.14$) showed greater stimulus incongruency effects on dACC activity, $t(53) = 2.46$, $p = .017$, $d = 0.65$, 95% CI_d: [0.10, 1.19]. In contrast, the test for differences between groups in rIFG activity was nonsignificant, $p = .138$, $d = -0.39$, 95% CI_d: [−0.92, 0.14]; this test for differences in rIFG activity remained nonsignificant when outliers in congruent errors were excluded, $p = .301$, $d = -0.28$, 95% CI_d: [−0.83, 0.26], whereas the significant difference in dACC became numerically greater, $p = .014$, $d = 0.69$, 95% CI_d: [0.13, 1.25]. In short, fMRI results suggested that MDDh-related executive control abnormalities were stronger within the region that is thought to compute the expected value of control—the dACC—than they were within the region that is thought to implement that control—the rIFG.

3.2.2. *Computational modeling*

Prior to conducting the primary modeling analyses, we examined the model's fit to empirical data. The model was a good fit to 89.1% of participants' datasets (see Supplemental Material).

As the next part of our model selection procedure, we examined how expected value of control parameters related to dACC activity. The selected model had the strongest association with dACC activity. Although dACC activity was not involved in model parameter estimation, the conjunction of the expected value of control parameters was associated with dACC activity, model omnibus test, $p < .001$. Moreover, these EVC parameters explained approximately 30% of the variance in congruency effects on dACC activity (controlling for model fit, multiple $R^2 = .37$; without controlling for model fit, multiple $R^2 = .29$; see Supplemental Material for figure). In contrast, the EVC parameters did not conjunctively predict the rIFG congruency effect, $p = .292$.

Finally, we examined model parameters in relation to diagnostic group using a logistic regression with group as the outcome. To reduce the need for multiple comparisons, we fit the full model (Table 2) and

Table 2
Parameter differences by group, with controls as the reference group.

Parameter	Odds Ratio	95% CI of the Odds Ratio
AE	0.15	[0.02, 0.62]
RfF	3.69	[0.61, 26.66]
$\xi_{\kappa}^{(0)}$	0.91	[0.44, 1.86]
	3.24	[1.38, 9.75]
β	1.16	[0.49, 2.84]
C^{max}	1.79	[0.59, 6.21]
θ_C^{scale}	1.35	[0.49, 3.92]
μ_{word}	0.59	[0.25, 1.31]
μ_{color}	1.12	[0.44, 2.82]
$\theta_{(0)}$	0.57	[0.11, 2.79]
NDT	1.08	[0.39, 3.07]
Model Fit Statistic	1.11	[0.55, 2.21]

Note. Odds ratios with 95% confidence intervals. Each predictor was standardized, such that the odds ratio represents the increase in odds for being a member of the MDDh group with every one standard deviation increase in the predictor. Individual estimates should be interpreted as exploratory without correction for multiple comparisons. See both Materials and Method and Supplemental Material for explanations of these parameters.

compared the deviance explained by the full model with either 1) a model absent the four parameters that are mathematically selectively involved in computing the expected value of control, irrespective of control ability (i.e., *AE, RfF,* $\xi_{(0)}$, and κ); and 2) a model absent the three control-related parameters (i.e., β, C^{max}, and θ_C^{scale}). In brief, we found that adding the EVC-related-parameters to the model absent them improved the ability to distinguish between the two groups, $\chi^2(4) = 11.72$, $p = .0196$, whereas adding the control-implementation-parameters to the model absent them did not improve the model's ability to distinguish between the groups, $\chi^2(3) = 2.71$, $p = .438$. Therefore, the conjunction of parameters involved in computing the EVC better differentiated the diagnostic groups than did the conjunction of control-implementation parameters within these data. Although hypothesis-driven, this block-level analysis is exploratory and thus should not be interpreted as providing confirmatory evidence. Nonetheless, together, these results converge with neuroimaging findings to suggest that differences in estimating and computing the expected value of control are associated with MDDh-related deficits in executive control task performance following stress.

3.3. Self-reported task-related affect

We next considered whether control-related changes in self-reported affect within the Stroop task were consistent with our neuroimaging and modeling findings. Group differences in affect were consistent with expectations and are provided within Supplemental Material.

If MDDh differences in Stroop task performance are in part driven by differences in affective processes underlying control recruitment, we would expect to see a difference in the association between trial-conflict-induced changes in affect (hereafter Conflict-Induced ΔAffect) and Stroop performance. We analyzed this difference in two ways, and results were entirely consistent; we report the second in the Supplement. In our primary method, we measured Conflict-Induced ΔAffect as the true experimental effect of trial type: Across all trials (i.e., self-reported affect following incongruent trials minus self-reported affect following congruent). We standardized Conflict-Induced ΔAffect and Stroop effects and dummy-coded Group such that the reference group's Conflict-Induced ΔAffect to Stroop effect coefficient was interpretable as a β coefficient, and the interaction was the change in β by group.

In this analysis, we found a significant Group × Conflict-Induced ΔAffect interaction in predicting Stroop effects, $\beta = .763$, $p = .005$. The MDDh group showed a significant negative association between Conflict-Induced Δ Affect and Stroop effects, $\beta = -.611$, $p = .004$. In contrast, the healthy control group showed no significant association between Conflict-Induced Δ Affect and Stroop effects, $\beta = .152$, $p = .338$. See Fig. 4. The interaction persisted even when the one outlier (healthy control; no change to MDDh slope) in Conflict-Induced ΔAffect >0.6 (see Fig. 4) was excluded, $\beta = .579$, $p = .043$. These results are consistent with differences in computing the expected value of control.

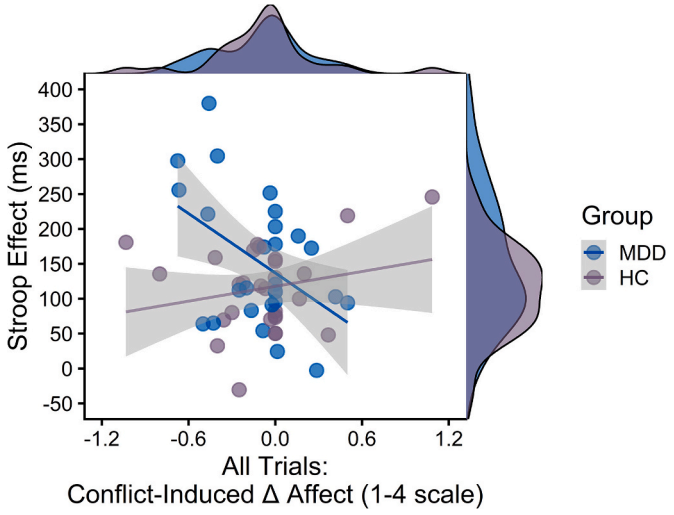

Fig. 4. Association between control-related changes in affect and behavioral task performance by group.

4. Discussion

Prior work had established that individuals with MDD show poorer executive control performance following stress than individuals without a history of psychopathology, but failures of interventional work to improve MDD by targeting executive control suggest that these findings may be driven by factors other than deficits in control per se. In this study, we examined whether processes involved in valuing and recruiting executive control might be associated with depression-history-related deficits in executive control following stress. Consistent with prior work, we found depression-history-related deficits in executive control task performance following stress (Quinn and Shields, 2023). To understand how these deficits emerge, we took a converging operations approach that incorporated both neuroimaging and computational modeling. The results converged to suggest that depression-history-related deficits in executive control task performance following stress were consistent with differences in estimating or computing the expected value of control.

At the level of neuroimaging, we observed a depression-history-related difference in dACC activity, but not rIFG activity. According to expected value of control theory (Shenhav et al., 2013), the dACC computes the expected value of control—calculating the costs and benefits expected from exerting control. Given this pattern of results, reverse inference considerations notwithstanding, these findings could be taken to suggest that atypical computations of the expected value of control may contribute to depression-history-related deficits in executive control behavioral performance.

The computational model results should be interpreted with caution, both because we have not yet conducted a full parameter recovery study (discussed below) and because we did not correct for multiple comparisons in our model predicting participant group from parameter values. We emphasize that we view the *pattern* of parameter results, irrespective of the parameters' significance, to be complementary—namely, that the exploratory parameter-block analysis suggested that EVC-related parameters may better differentiate the groups than control-implementation parameters. Nonetheless, this pattern, although exploratory, converges with our behavioral and imaging results to suggest that differences in the expected value of control may play a role in MDD-related differences in executive control following stress.

Given the expected value of control is thought to be computed in the dACC, it is possible these parameters capture processed reflected by the dACC. This idea is supported by our association between DDM parameters and dACC activity. However, this interpretation is speculative: We did not formally jointly model behavioral and neural data, and DDM parameters are presumably difficult to map onto specific regions or neural circuits without such an approach.

Consistent with the idea that MDDh-related differences in executive control task performance following stress reflect differences in processes involved in estimating the expected value of control, analyses of self-report data revealed that MDDh participants differed in the association between control-related self-reported affect and behavioral performance. Notably, MDDh-group participants showed an inverse association between changes in affect as a function of control exertion and Stroop effects, whereas no such association was observed in control participants. Considered in conjunction with our imaging and modeling results, we believe that these results suggest that larger Stroop effects in participants with a history of MDD may often reflect differences in affective processes underlying control recruitment. In short, our

behavioral, neuroimaging, and modeling findings converge to suggest that differential processing of the expected value of control for MDDh participants may, at least in part, be responsible for executive control deficits in MDDh.

4.1. Role of stress

We aimed to determine why poor executive control following stress is related to depression, given prior work (Quinn and Shields, 2023). Although we are not the first to speculate on the expected value of control in relation to depression (e.g., Grahek et al., 2019), we are the first to consider it as an explanation for performance following stress. Because we did not have a no-stress control condition in this study, we cannot determine why assessing executive control specifically following stress strengthens its link to stress-related psychopathology. However, we may speculate to some degree. In particular, prior work has suggested that stress may influence executive control task performance, at least to some degree, through reconfiguring the "goal": exerting control is more aversive following stress (Bogdanov et al., 2021; Shields et al., 2024). Such an approach to task difficulty is akin to an avoidance strategy in emotion regulation (Inzlicht et al., 2015; Shields et al., 2024). Stress may thus increase the likelihood that individuals predisposed to avoidance (e.g., those at risk for depression; Grant et al., 2013; Holahan et al., 2005) choose to do so within executive control tasks, and thus attenuate their own performance expectations and show poorer performance accordingly.

4.2. Strengths and limitations

This study had multiple strengths. First, MDDh participants were a community sample that had a diagnosis of either current or past major depressive disorder. Second, most participants in our sample were not taking psychotropic medications nor did most have comorbid diagnoses. Third, we used a well-validated stress induction, and confirmed its success using the gold-standard validator (i.e., a cortisol response) (Shields, 2020).

Despite the strengths, the current study is not without its limitations. First, the current sample was majority female, which limits generalizability. Second, our sample was nonetheless smaller than ideal. Because of our relatively small sample size, our ability to detect anything smaller than medium-sized effects was limited. A replication study that uses a larger sample with pre-registered hypotheses is warranted to examine our preliminary findings. Indeed, evidence that MDDh participants had behavioral deficits in executive control was limited by a one-tailed test. Our findings indicate that the MDD-related differences following stress are consistent with differences in computing the expected value of control, but our data cannot be taken to support the idea that there are no differences in control itself. It is certainly possible that we would have observed differences in control processes themselves if our sample size had been larger. We were further limited by our sample size to assess whether our results were resistant to the influence of medication and comorbid psychopathology. As such, we are unable to conclusively state whether our findings were specific to those with a history of MDD. However, other forms of psychopathology are frequently comorbid with MDD, meaning our sample reflects a presentation common in clinical practice (Kessler et al., 2005). Third, the processes that give rise to depression and related cognitive differences in our participants may differ in other cultures (Henrich et al., 2010). Fourth, the current study does not have a non-stress control group for those with and without a history of depression. As described above, our study assumed the findings of Quinn and Shields (2023) as a starting point, and aimed to testing mechanisms potentially underlying depression-history-related differences within an acute stress context. However, without a nonstress control condition, we were unable to directly test the extent to which executive control assessed following stress is a better predictor of psychopathology than assessing executive control in the absence of stress. Therefore, we cannot disentangle simple depression-history-related differences from stress-specific depression-history-related differences. It is quite possible that the depression-history-related differences that we observed may have been observed under nonstressful conditions, too. Nonetheless, these limitations do not preclude us from making inferences related to the aim of this study: Determining mechanisms underlying MDD-related deficits in executive control performance following stress.

Our model, although theoretically driven, is novel. It is not uncommon in novel modeling work to initially focus on elucidating mechanisms underlying known effects (e.g., Ulrich et al., 2015), conducting parameter recovery studies later to revise the model as appropriate (e.g., White et al., 2018). Lacking a parameter recovery study, it is difficult to know whether the parameters we estimated were reliable. Importantly, however, the parameters in which we found group differences were solely related to estimating the expected value of control. Additionally, the convergence of our modeling results with our behavioral and fMRI provides confidence in our modeling inferences. Nonetheless, in the absence of a parameter recovery procedure, our modeling results should be considered exploratory: It is unclear whether the pattern we observed would still emerge following a parameter-recovery-study-guided model revision, should any revisions be required.

We also note that our interpretation of our neuroimaging results is reverse inference, which can be problematic (Poldrack, 2006), though not always (Hutzler, 2014). Although there is strong evidence that the dACC is involved in the computation of control, and the rIFG is involved in the implementation of control, both regions are part of larger distributed networks (Shenhav et al., 2013, Shields et al., 2017, Bush et al., 2022, Friedman and Miyake, 2017; Wessel and Anderson, 2024). Because of the role of the dACC is involved larger distributed networks, it is possible that dACC activity could also be explained in part of processes related to conflict monitoring, error likelihood, or other cognitive processes. Similarly, a lack of a statistical difference in the rIFG activity does not necessarily rule out implementation differences in the MDDh group. Other regions, such as the DLPFC, may also be involved in control implementation. As such, there are limitations to which individual regions explain complex cognitive processes. Nonetheless, our converging operations approach—and the convergence of findings across neuroimaging, computational modeling, and self-reported affect analyses—lends some confidence to our interpretation: Differences in the computation of the expected value of control are consistent with depression-history-related differences in executive control following stress.

5. Conclusion

A large body of work has documented poorer executive control following stress in stress-related psychopathology, including MDDh. The current study examined the mechanisms underlying these known associations. We found that executive control deficits following stress in individuals with a history of MDD are consistent with atypical computations of the expected value of exerting control. These results hint that there may be a benefit to exploring interventions that target control valuation processes in depression.

CRediT authorship contribution statement

Ben Swanson: Writing – review & editing, Writing – original draft, Visualization, Validation, Formal analysis, Conceptualization. **Grant S. Shields:** Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Matt R. Judah:** Writing – review & editing, Supervision.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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All authors declare no conflicts of interest related to this work.

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No large language models were used in any way related to this project or manuscript (e.g., analyses, writing, etc.).

Data and syntax for analyses are available on the OSF: https://osf.io/eu8yh/overview?view_only=d72bb116bb184dbf93b3b49e99932e80

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jad.2026.122274.

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