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Effective connectivity underlying reward-based executive control

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Abstract

Motivational influences on cognitive control play an important role in shaping human behavior. Cognitive facilitation through motivators such as prospective reward or punishment is thought to depend on regions from the dopaminergic mesocortical network, primarily the ventral tegmental area (VTA), inferior frontal junction (IFJ), and anterior cingulate cortex (ACC). However, how interactions between these regions relate to motivated control remains elusive. In the present functional magnetic resonance imaging study, we used dynamic causal modeling (DCM) to investigate effective connectivity between left IFJ, ACC, and VTA in a task-switching paradigm comprising three distinct motivational conditions (prospective monetary reward or punishment and a control condition). We found that while prospective punishment significantly facilitated switching between tasks on a behavioral level, interactions between IFJ, ACC, and VTA were characterized by modulations through prospective reward but not punishment. Our DCM results show that IFJ and VTA modulate ACC activity in parallel rather than by interaction to serve task demands in reward-based cognitive control. Our findings further demonstrate that prospective reward and punishment differentially affect neural control mechanisms to initiate decision-making.

KEYWORDS
cognitive control, dynamic causal modeling, fMRI, motivation, punishment, reward

1 | INTRODUCTION

Every action we take in our daily lives has potential consequences, which we perceive as either positive or negative. These consequences shape our future behavior substantially and fuel our motivation to make decisions that lead toward desirable outcomes, such as the receipt of rewards, and also the avoidance of punishments. Importantly, heightened motivation facilitates executive functioning (for review, see Botvinick & Braver, 2015). An illustrative example is the enhancing effect of monetary incentives (i.e., potential gain or loss) on the performance in cognitive tasks (Boehler, Schevernels, Hopf, Stoppel, & Krebs, 2014; Engelmann & Pessoa, 2014; Etzel, Cole, Zacks, Kay, & Braver, 2015; Guitart-Masip et al., 2012; Padmala & Pessoa, 2011; Tricomi, Delgado, & Fiez, 2004; Wächter, Lungu, Liu, Willingham, & Ashe, 2009). Krebs, Boehler, and Woldorff (2010), for instance, could show that performance-contingent monetary reward improved cognitive control such that participants named the color of a Stroop stimulus faster and more accurately in prospective-reward compared to no-reward trials.

On a neural level, the beneficial effect of motivation on executive control seems to depend on communication between structures

within the dopaminergic system (Cools, 2008). Particularly, the anterior cingulate cortex (ACC), inferior frontal junction (IFJ), and ventral tegmental area (VTA) located in the midbrain have been suggested as instrumental in the integration of cognitive control and motivation (Aarts et al., 2010; Aarts, van Holstein, & Cools, 2011; Bahlmann, Aarts, & D'Esposito, 2015; Parro, Dixon, & Christoff, 2017; Westbrook & Braver, 2016).

The ACC takes the center stage in this setting (Shenhav, Cohen, & Botvinick, 2016) and has consistently been associated with interactions between motivation and control. In particular, it is thought to serve as an integrative node, where input from other areas signaling the prospect of reward or punishment on the one hand and the need for enhanced control on the other hand is combined (Fujiwara, Tobler, Taira, Iijima, & Tsutsui, 2009; Kahnt, Grueschow, Speck, & Haynes, 2011; Mansouri, Egner, & Buckley, 2017; Vassena et al., 2014; Wallis & Kennerley, 2011). According to the "expected value of control" theory (Shenhav, Botvinick, & Cohen, 2013), the ACC assesses the estimated benefit and cost to expend cognitive effort and appropriates control correspondingly. This account is substantiated by studies linking ACC activity with both anticipated effort and anticipated reward (Croxson, Walton, O'Reilly, Behrens, & Rushworth, 2009; Prévost, Pessiglione, Météreau, Cléry-Melin, & Dreher, 2010).

Besides the ACC, the VTA as a source of dopaminergic neuromodulation in goal-directed behaviors is assumed to play a pivotal role in the interaction between cognitive control and motivation (Arias-Carrión, Stamelou, Murillo-Rodríguez, Menéndez-González, & Pöppel, 2010; Beier et al., 2015; Zellner & Ranaldi, 2010). For instance, dopamine (DA) is key in implementing the influence of incentives on executive control (Aarts et al., 2010; Adcock, Thangavel, Whitfield-Gabrieli, Knutson, & Gabrieli, 2006). Activations of the VTA have been associated with cognitive flexibility as well as both reward and punishment motivation (Carter, MacInnes, Huettel, & Adcock, 2009; D'Ardenne, McClure, Nystrom, & Cohen, 2008; Guitart-Masip et al., 2012). Of particular interest in this context are DA projections from VTA to the lateral prefrontal cortex (lPFC) via the mesocortical pathway, which are thought to be essential in constituting cognitive function (Durstewitz & Seamans, 2008; Goldman-Rakic, 1992). Likewise, VTA projects to the ACC and is assumed to thereby contribute to reward-based cognitive processing (Haber & Knutson, 2010; Hauser, Eldar, & Dolan, 2017; Köhler, Bär, & Wagner, 2016).

Alongside ACC and VTA, the IFJ, a subregion of the lPFC, recently has come into focus as a critical neural substrate in the integration of control demands and potential outcomes. Numerous studies have demonstrated IFJ activation during reward-based executive control (see Parro et al., 2017 for a meta-analysis). Modulations in effective connectivity from IFJ to ACC have been associated with changes in cognitive demand (Harding, Yücel, Harrison, Pantelis, & Breakspear, 2015; Hinault, Larcher, Zazubovits, Gotman, & Dagher, 2019). Evidence further suggests that the IFJ is closely linked to the dopaminergic system (Stelzel, Basten, Montag, Reuter, & Fiebach, 2010; Stelzel, Fiebach, Cools, Tafazoli, & D'Esposito, 2013) and serves task demands by selectively engaging brain regions related to control (Asplund, Todd, Snyder, & Marois, 2010; Baldauf & Desimone, 2014; Kim, 2014) and motivation (Paschke et al., 2015). In addition, functional connectivity between IFJ and midbrain has been found to correlate with the individual enhancing effect of reward on cognitive performance (Bahlmann et al., 2015).

All three regions—IFJ, VTA, and ACC—therefore appear to be interconnected and involved in the interplay between motivation and cognitive control. While the body of evidence suggests that both IFJ and VTA direct ACC activity in motivation-based executive functioning, the exact pathways and in particular the direction of connectivity between IFJ and VTA remains unclear. In an initial study using transcranial magnetic stimulation, we could show that the left IFJ is causally involved in reward-related cognitive facilitation and suggest that this effect may be realized via modulation of the dopaminergic network (Hippmann et al., 2019). Likewise, previous evidence suggests that the lPFC initiates motivated behavior by influencing VTA activity in anticipation of reward (Ballard et al., 2011). These findings, therefore, support the notion that the lPFC, and particularly the IFJ, exerts control over VTA and ACC to shape motivated cognitive control. However, an alternative possibility is that interactions between motivation and control are rooted in DA projections from VTA to the lPFC, essential positing an influence in the opposite direction. For instance, DA levels in the lPFC have been associated with cognitive control and attention (Durstewitz & Seamans, 2008; Vijayraghavan, Wang, Birnbaum, Williams, & Arnsten, 2007).

Another unsolved issue concerns potential differences in neural processing between two main motivational goals: pursuing reward and avoiding punishment. Even though activations in the aforementioned regions have been associated with both processes, it is unclear whether they act via the same neural mechanism to produce cognitive performance increments. Some researchers argue in favor of a common mechanism (Xue et al., 2013), while others support the idea of opponent systems for reward and punishment (Palminteri & Pessiglione, 2017; Wächter et al., 2009).

The goal of the present study, therefore, was to discern these possibilities by means of dynamic causal modeling (DCM) within a proposed network of ACC, IFJ, and VTA considering both, reward- and punishment-driven influences on cognitive control. Even though DCM has been criticized in terms of biophysical realism (Daunizeau, David, & Stephan, 2011), it serves as a valuable tool to observe information flow between critical structures as it allows to make inferences about the strength and directionality of connections between regions of interest (Friston, Harrison, & Penny, 2003).

We designed a functional magnetic resonance imaging (fMRI) study using a task-switching paradigm that included motivational manipulations through monetary incentives. Participants either switched between or repeated two competing tasks (i.e., numbers were judged either by parity or magnitude). These tasks were embedded in two motivational conditions, in which participants received performance-contingent monetary reward or punishment, and a neutral condition without monetary incentives. We expected that switching (compared to repeating) tasks would be facilitated through both prospective reward and prospective punishment. Based on the evidence outlined above, we hypothesized that both IFJ and VTA

would exert control over ACC activity and that these modulations would relate to interactions between motivation and cognitive control. We also suspected that neural interactions between IFJ and VTA would be modulated by cognitive control and motivation and considered modulations in either direction to be possible scenarios. Furthermore, we were interested in whether prospective reward and punishment recruited these structures in the same manner.

2 | METHODS

2.1 | Participants

Twenty-nine right-handed participants took part in the present study after giving informed consent. All reported normal or corrected-to-normal vision and were screened for common fMRI exclusion criteria as well as their personal and family history of neurological abnormalities. They received compensation (7€/hr) for participation as well as monetary rewards in accordance with their individual performance on the task. Data from two participants had to be discarded due to structural abnormalities on the basis of a T1-weighted structural scan evaluated by a neuroradiologist. Four additional data sets were excluded because of technical issues during measurement (i.e., scanner failure, no recording of responses, incorrect display of stimuli). The remaining 23 participants (13 females, mean age = 23.0, ranging between 19 and 29 years) were included in the behavioral and fMRI analysis. The study was approved by the local ethics committee and conducted in conformance with the principles of the Declaration of Helsinki.

2.2 | Experimental paradigm

Participants performed a task-switching paradigm including different motivational conditions while being comfortably placed in an MRI scanner. Stimuli were numbers between 1 and 50 presented on a black screen in random sequence (see Figure 1a) and viewed over a mirror mounted on the MR head coil. The color of these numbers

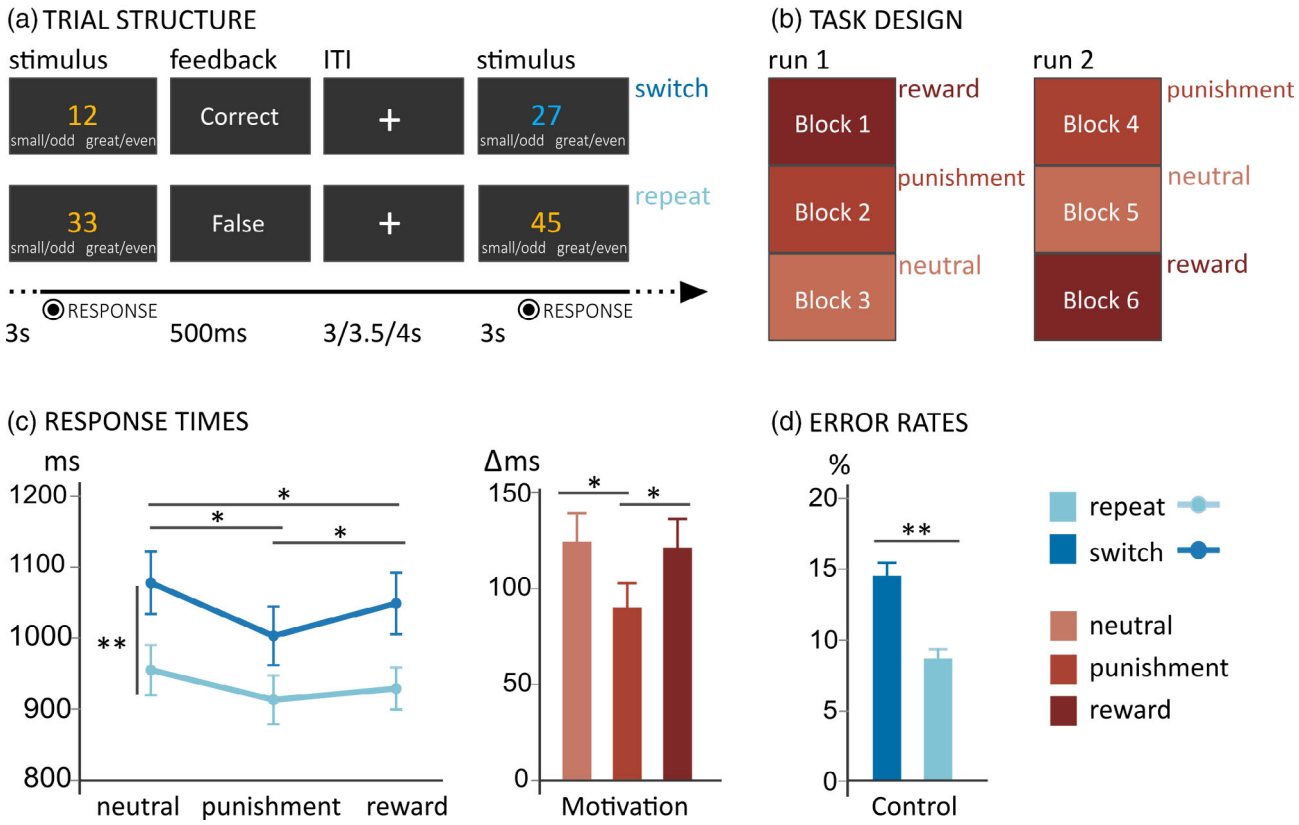

FIGURE 1 Design and behavioral results. (a) Participants judged numbers according to either magnitude (blue) or parity (yellow). Switch and repeat trials, defining the factor Control, were pseudo-randomized (not more than three in a row). (b) Three levels of motivation were linked to monetary incentives. Reward = participants earned 7 Cent for correct responses. Punishment: Participants lost 7 Cent for incorrect responses. Neutral: No money could be gained or lost. The factor Motivation was blocked and randomized within two fMRI runs. Each block was composed of 50 trials and 10 null trials (white fixation cross). (c) Response times (mean ± SE) per task condition and switch costs (switch minus repeat) per motivational condition in milliseconds. Switch trials were followed by slower responses compared to repeat trials. This effect was reduced in the punishment condition. (d) Error rates (mean ± SE) in percent. Across motivational conditions, participants made more errors on switch compared to repeat trials. *$p < .05$, **$p < .001$, FDR-corrected; $n = 23$. fMRI, functional magnetic resonance imaging; ITI, inter-trial interval; FDR, false discovery rate

established which of two tasks to perform. Blue required participants to judge a number regarding its magnitude (smaller or greater than 25), while a yellow number constituted the parity task (even or odd). If two subsequent trials obeyed the same task rule (parity following parity or magnitude following magnitude) this sequence was defined as a repeat. A switch was defined as a sequence of two trials with diverging task rules (parity following magnitude or vice versa). Participants gave responses on a fiber optic response pad with the index (smaller/odd) or middle (greater/even) finger of their right hand. A response map was displayed below the stimuli. Numbers were chosen to create contrasting response patterns and thus avoid indistinct responses. To that end, only even numbers smaller than 25 and odd numbers greater than 25 were included (Hippmann et al., 2019). The task was divided into blocks with different motivational conditions: In the punishment condition, participants lost 7 Cents when they gave an incorrect response but did not gain any money for correct responses. In the reward condition, participants earned 7 Cents for correct responses but did not lose any money for incorrect responses. In the neutral condition no money was gained or lost. Participants were instructed to respond as fast as possible. Each response was followed by feedback displayed on the screen (“Plus 7 Cent”, “Minus 7 Cent”, “Correct,” or “False”).

The total of six blocks was divided by a short break into two scan runs. Each run was composed of a reward, a punishment, and a neutral block (order counterbalanced across participants, see Figure 1b). An instruction screen (10 s) before each block indicated which motivational condition would follow. After completing a block, participants received feedback about the amount of money they gained or lost (10 s). A block contained 50 trials and 10 null trials (white fixation cross, randomized) and was composed of an equal number of switches and repeats, presented in pseudo-randomized order (not more than three in a row). Null trials were introduced for jittering purposes in order to increase detectability of task-related responses (Burock et al., 1998). A fixation cross presented for 3, 3.5, or 4 s initiated each trial followed by the presentation of a stimulus (3 s) and feedback (500 ms). Each stimulus was displayed for 3 s regardless of response time (RT). A depiction of the trial structure can be found in Figure 1a.

Prior to the MR session, participants familiarized themselves with the task in a brief preparatory training outside the scanner, which consisted of 25 trials without motivational cues. If participants had more than 10 misses or errors, the training was repeated. The individual mean RT from the training served as initial response window duration (ranging between 900 and 1,400 ms) in the task. Over the time course of the experiment the width of this window changed depending on the individual performance in order to continuously tailor task difficulty to each participant's ability. For each correct response, 15 ms were subtracted from the duration. For each false response, 85 ms were added. Missed responses had no influence on the duration.

In the debriefing, we asked participants which strategies they used to make judgments during the task. All participants reported using parity and magnitude as criteria and neither participant described strategies that were in conflict with our intended task requirements. The whole experiment lasted approximately 60 min.

2.3 | Behavioral analyses

Reaction time (RT) and error rate (ER) were subjected to separate repeated-measures analyses of variance (ANOVAs) with the factors Motivation (neutral, punishment, reward) and Control (repeat, switch). First trials of each block and those following null trials were excluded from the analysis, since they could not be categorized as switch or repeat trials. RT was analyzed on correct trials only. Post-hoc *t*-tests were carried out on significant main effects and interactions and corrected for multiple comparison using the false discovery rate (FDR) method. Effect sizes for ANOVAs are reported as partial eta-squared. Data were analyzed using R v3.3.2 (R Core Team, 2016).

2.4 | fMRI data acquisition

Structural and functional MR imaging was performed at the Center of Brain, Behavior and Metabolism (CBBM, University of Lübeck) using a 3-T Siemens Magnetom Skyra scanner equipped with a 64-channel head coil. Functional MRI data were acquired in two runs, each containing three experimental blocks (reward, punishment, neutral in randomized order). A gradient echo-planar imaging sequence sensitive to blood oxygen level-dependent (BOLD) contrast was used with the following specifications: TR = 1,690 ms, TE = 25 ms, flip angle = 80°, parallel imaging acceleration factor 2 (GRAPPA), 3×3 mm^2 in-plane resolution, 192×192 mm^2 field of view, 34 transversal ascending slices of 2.5 mm thickness and 25% gap coplanar to AC-PC. Structural images were collected using a 3D T1-weighted MPRAGE sequence (TR = 1900 ms, TE = 2.44 ms, TI = 900 ms, flip angle = 9°, $1 \times 1 \times 1$ mm^3 resolution, $192 \times 256 \times 256$ mm^3 field of view).

2.5 | fMRI data analysis

Functional data were analyzed using the Statistical Parametric Mapping software package SPM12 (available at http://www.fil.ion.ucl.ac.uk/spm) implemented in MATLAB 2015a (Mathworks, Sherborn, MA). Preprocessing included slice-timing correction to the first slice, realignment to the first functional volume, co-registration to T1 structural image, segmentation, normalization to Montreal Neurological Institute (MNI) space in native voxel space, smoothing with an 8 mm full width half maximum Gaussian kernel and high-pass temporal filtering ($T = 128$ ms). Autocorrelation in fMRI time series has been accounted for by applying an autoregressive AR(1) model during parameter estimation.

For each participant, event-related responses were estimated using a general linear model approach. The model included separate regressors for correct trials for the six conditions (punishment switch, punishment repeat, neutral switch, neutral repeat, reward switch,

reward repeat) convolved with the hemodynamic response function. Events were time-locked to the onset of each trial (appearance of colored number) and were modeled as having a duration of 0 s. Additionally, for each run the model comprised six regressors for movement and three regressors of no interest for error trials, null-trials, and feedback onsets.

Statistical analyses on the group level were specified as flexible factorial design and implemented in SPM12 with each subject treated as a random effects variable. The main effects of cognitive control (switch > repeat) and motivation (punishment > neutral and reward > neutral) were tested using t-contrasts, the interaction between both factors was assessed via F-contrasts. Contrast weights were assigned according to Gläscher and Gitelman (2008). An uncorrected voxel-level threshold of $p < .001$ was selected for all analyses, with a family-wise error (FWE) correction threshold of $p < .05$ at cluster level.

2.6 | Dynamic causal modeling

We used DCM (Friston et al., 2003) as implemented in SPM12 version DCM12, to disentangle the directional interactions between brain regions underlying the enhancing effect of motivation on cognitive control.

DCM models describe changes in regional activity as:

$$\frac{d\vec{x}}{dt} = \left(A + \sum_{j=1}^{m} u_j B^{(j)}\right)\vec{x} + C\vec{u}$$

with $\vec{x}$ representing a neuronal state vector and $\vec{u}$ representing an input vector. A describes latent connectivity between brain regions irrespective of experimental conditions, B describes the modulatory influence of experimental conditions on the intrinsic connections, and C describes the extrinsic effects of input $\vec{u}$ on activity. Since DCM is considered a hypothesis-driven rather than data-driven approach (Daunizeau et al., 2011), prior knowledge of, or hypotheses on network connections and modulations are essential for inferring an optimal, thus most plausible, model of effective connectivity out of an a priori defined model space. For each model, the state equation is transformed into a predicted BOLD signal by a biophysical forward model of hemodynamic responses (Friston, Mechelli, Turner, & Price, 2000; Stephan, Weiskopf, Drysdale, Robinson, & Friston, 2007), which is then fitted to the actual BOLD signal through a gradient ascent on the free-energy bound. Note that while interactions between nodes in the network may occur on a millisecond level, the predicted BOLD signal as the actual measured signal represents changes on a second level. A "winning model" is subsequently selected based on the posterior probability associated with each model's evidence using the Bayesian model selection procedure (BMS, Penny, Stephan, Mechelli, & Friston, 2004). We extracted connectivity parameters from the winning model and used random-effects parametric analysis across participants to estimate changes in modulatory effects on connectivity.

2.7 | Time series extraction

We specified left IFJ, left ACC, and left VTA as volumes of interest (VOIs). We limited the DCM analysis to the left hemisphere since previous studies report hemispheric specialization in cognitive control (e.g., Badre & Wagner, 2007; Serrien & Sovijärvi-Spapé, 2013; Stephan et al., 2003). Regarding the research questions addressed in the present study, previous work finds control and motivation effects related to the IFJ in the left hemisphere (Bahlmann et al., 2015; Harding et al., 2015; Hippmann et al., 2019). As there is limited knowledge about interhemispheric interactions between the selected VOIs, we wanted to avoid making unsupported assumptions about network connections in order to narrow down the model space. Coordinates of the VOIs for IFJ ($x = -45$ mm, $y = 5$ mm, $z = 29$ mm) and VTA ($x = -5$ mm, $y = -25$ mm, $z = -10$ mm) were selected based on the group level maxima in the task > baseline contrast ($p < .05$, FWE-corrected, see Table 2B) in order to account for changes in BOLD signal related to all six task conditions. VOI coordinates for the ACC ($x = -10$ mm, $y = 8$ mm, $z = 41$ mm) were isolated from the task > baseline contrast with an anatomical mask (see Figure 2 for visualization of VOIs). Time series for each VOI were extracted from significant voxels ($p < .01$, uncorrected) in the individual task > baseline contrasts. For each participant, the sphere center of each VOI was moved to the closest suprathreshold voxel, which was kept within a 10 mm radius from the group peak coordinates. Xjview toolbox (http://www.alivelearn.net/xjview) and AAL brain atlas were applied to verify that the individual sphere centers were located within the regions of interest. This individualized peak approach allows targeting those regions on the single-subject level that are most likely to drive ongoing neural processes in the group level while ensuring that individual regions remain comparable. The approach is in line with prior comparable studies examining task-related DCM effects (Heim et al., 2009; Kleineberg et al., 2018; Roswandowitz, Swanborough, & Frühholz, 2021).

Using a singular value decomposition procedure implemented in SPM12, we computed the first eigen variate across all suprathreshold voxels within a 6 mm radius from the sphere center for each participant. We chose a larger radius for the spheres to avoid including signals in the DCM analysis, which are derived from VOIs with very few voxels and thus are more susceptible to noise. Time series were then adjusted for effects of interest and sharp improbably temporal artifacts were smoothed by an iterative procedure implementing a six-pint cubic-spline interpolation. We could not extract time series in three participants for all VOIs (one IFJ, two ACC) based on these criteria. Since DCM requires time series from the full network, we repeated the procedure for the respective participants and VOIs with a more tolerant threshold of $p < .05$ (uncorrected), which allowed the inclusion of all 23 participants in the analysis.

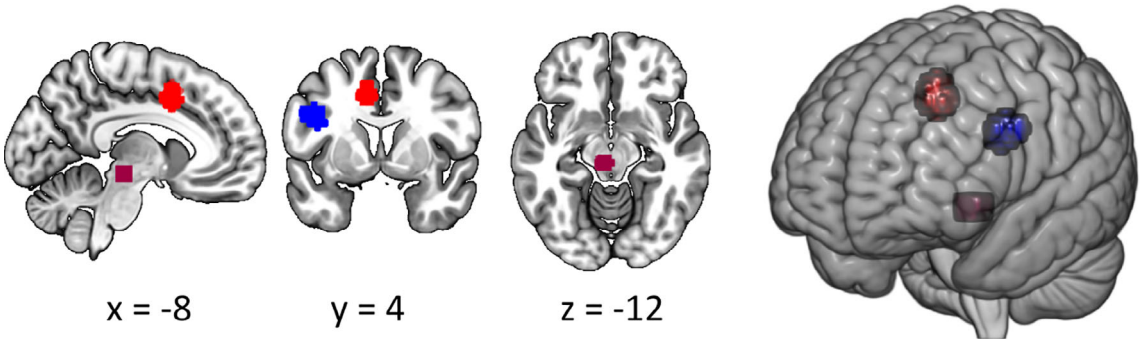

FIGURE 2 Clusters entering DCM analysis. Pooled participants' individual VOIs for anterior cingulate cortex (ACC, red), interior frontal junction (IFJ, blue), and ventral tegmental area (VTA, violet); $n = 23$. DCM, dynamic causal modeling; VOIs, volumes of interest

We conducted a power analysis using G*Power v3.1.9.2 to estimate the power that could be achieved on a one sample *t* test (as applied to test the strength of intrinsic and modulatory connections in DCM models). Analysis revealed that 23 participants suffice for 96% power on strong effects ($d = .8$), 63% power on medium effects ($d = .5$), and 15% power on small effects ($d = .2$) with an alpha of .05.

2.8 | DCM specification

Input vector $\vec{u}$ was constructed as a stick function of the single events of stimulus presentation. Given that distinct motivational states (i.e., neural processes related to reward and punishment) may differentially interact with task processing in the selected regions of interest, we built separate model families for the three motivational conditions (neutral, punishment, reward). These model families were based on the same task, comprised the same number of trials and consisted of models with identical architecture between the same set of regions. They only differed in terms of the modulatory effect (B-matrix) meaning, for instance, the modulatory input for the reward family derived from "reward switch" and "reward repeat" trails. The DCM framework allows to compare plausible models that are constructed based on a set of predefined regions of interest and a priori knowledge on network connections between those regions. We, therefore, made a few assumptions regarding the model space in order to restrict the amount of comparisons. First, we allowed bidirectional intrinsic connections between the VOIs in all models. Second, we considered both IFJ and midbrain to be plausible input regions, based on the evidence outlined in the introduction. In order to avoid inflating the model space and thereby decreasing the stability of results, we opted against establishing the input region computationally by building separate models for different input nodes. We decided to admit task input on both IFJ and midbrain in all models. This allowed us to identify each region's contribution to the neural processes on the parametric level. Note that it is plausible that an unmodeled region such as the visual cortex could serve as better input node to the model. Still, the summed influence of the input on the chosen input regions should be evident. Third, the model space comprised all possible permutations of modulatory connections between the VOIs, except for the connection from IFJ to ACC. Since previous work on effective connectivity during cognitive tasks showed that the IFJ directs activity in the ACC (Harding et al., 2015; Hinault et al., 2019), the connection was operative in all models. This amounted to 32 models per family (see Supplementary Figure) and 96 models in total. Assuming variability across participants, we compared models and model families using RFX BMS [Penny et al., 2010]. After selecting a winning model based on the highest exceedance probability, we extracted the estimated model parameters in each participant and calculated an average parameter estimate for each connection, which was then tested for strength using one-sample *t*-tests. Multiple comparisons were corrected using FDR.

3 | RESULTS

3.1 | Behavioral results

3.1.1 | Response time

We found significant effects on RT for Control ($F_{(1,22)} = 67.34$, $p < .001$, $\eta_p^2 = 0.75$, repeat = 933 ms, switch = 1,044 ms) and Motivation ($F_{(2,44)} = 5.71$, $p < .01$, $\eta_p^2 = 0.21$). Participants were significantly faster in the punishment condition (957 ms) compared to the reward (988 ms, $p = .02$) and neutral conditions (1,017 ms, $p < .01$). RT in the neutral and reward blocks differed significantly ($p = .04$). Moreover, we found a significant interaction between Control × Motivation ($F_{(2,44)} = 3.74$, $p = .03$, $\eta_p^2 = 0.15$). Post-hoc analyses revealed significant differences between switch and repeat trials for all three motivational conditions with $p < .001$. Switch costs were significantly lower in punishment (90 ms) compared to neutral (123 ms, $p = .04$) as well as reward blocks (121 ms, $p = .04$, see Figure 1c). Neutral and reward blocks did not differ significantly in regard to switch costs ($p = .83$). See Table 1 for RT values of each task condition.

3.1.2 | Error rates

The significant main effect of Control ($F_{(1,22)} = 29.16$, $p < .001$, $\eta_p^2 = 0.57$) revealed that participants showed lower ERs on repeat

TABLE 1 Summary of behavioral measures

	Neutral		Punishment		Reward	
	Switch	Repeat	Switch	Repeat	Switch	Repeat
RT in ms (*SD*)	1,079 (210)	956 (169)	1,004 (198)	914 (165)	1,050 (207)	929 (141)
ER in % (*SD*)	15.3 (6.8)	9.1 (5.2)	14.8 (5.9)	9.2 (5.3)	13.6 (5.6)	7.8 (4.6)

Note: Means of error rates in percent and response times in milliseconds separately for each condition; $n = 23$.

TABLE 2 Whole brain imaging results

	MNI coordinates					
	x	*y*	*z*	Cluster size	*T*-value	p_{FWE}
Contrast/brain region		(mm)		(voxel)	(peak)	(cluster)
A switch > repeat						
R insula	31	23	5	187	6.94	.000
L midbrain	−2	−25	−13	157	6.55	.000
L SMA	−5	2	56	144	4.68	.000
L IPS	−30	−52	53	72	4.37	.022
L precuneus	−7	−70	50	64	4.15	.036
Repeat > switch						
R SFG	6	50	38	72	4.87	.022
B task > baseline						
L precentral gyrus	−30	−22	56	1,598	14.70	.000
L midbrain	−5	−25	−10	443	11.83	.000
R insula	31	23	5	118	10.08	.000
R cerebellum	11	−52	−16	89	9.24	.000
R precentral gyrus	36	−7	50	77	8.73	.001
L insula	−30	20	5	63	9.14	.000
R IPS	33	−46	47	27	8.22	.002
R middle occipital gyrus	31	−91	17	24	8.44	.001
L IFJ	−45	5	29	24	7.66	.005

Note: Results are reported with a cluster defining threshold of $p = .001$ and FWE correction at $p < .05$. L, left; R, right; SMA, supplementary motor area; IPS, inferior parietal sulcus; SFG, superior frontal gyrus; IFJ, inferior frontal junction; $n = 23$.

trials (8.7%) than on switch trials (14.5%, see Figure 1d). The main effect of Motivation ($F_{(2,44)} = 1.17$, $p = .32$, $\eta_p^2 = 0.05$) and the interaction between Control × Motivation ($F_{(2,44)} = 0.04$, $p = .96$, $\eta_p^2 = 0.002$) were not statistically significant. ERs for each task condition can be found in Table 1.

3.2 | fMRI results

Higher cognitive demand (switch > repeat, see Table 2A) was associated with significantly increased neural activity in the right insula, left midbrain, supplementary motor area (SMA), precuneus, and inferior parietal sulcus (IPS). For the reverse contrast (repeat > switch) a significant cluster was found in the right superior frontal gyrus. We found no significant clusters associated with the main effect of Motivation or the interaction between Control and Motivation.

3.3 | DCM results

Figure 3a depicts exceedance probabilities derived from the Bayesian model comparison. Family level inference results showed that the reward family had a higher exceedance probability ($p\_ex = .73$) compared to the punishment ($p\_ex = .10$) or neutral family ($p\_ex = .17$ see Figure 3a) suggesting that interactions between IFJ, ACC, and VTA are more prominent during reward blocks. The Bayesian omnibus risk (BOR) indicator measuring the probability that all model frequencies are distinguishable was 0.9 indicating that the models were not well distinguishable (Rigoux, Stephan, Friston, & Daunizeau, 2014). Note, however, that this is to be expected with a large number of models of similar structure. BOR calculation further cannot consider family grouping of the models. We thus continue to report the results from the winning model and compare its modulatory parameters to those of the models from the other families with the same architecture.

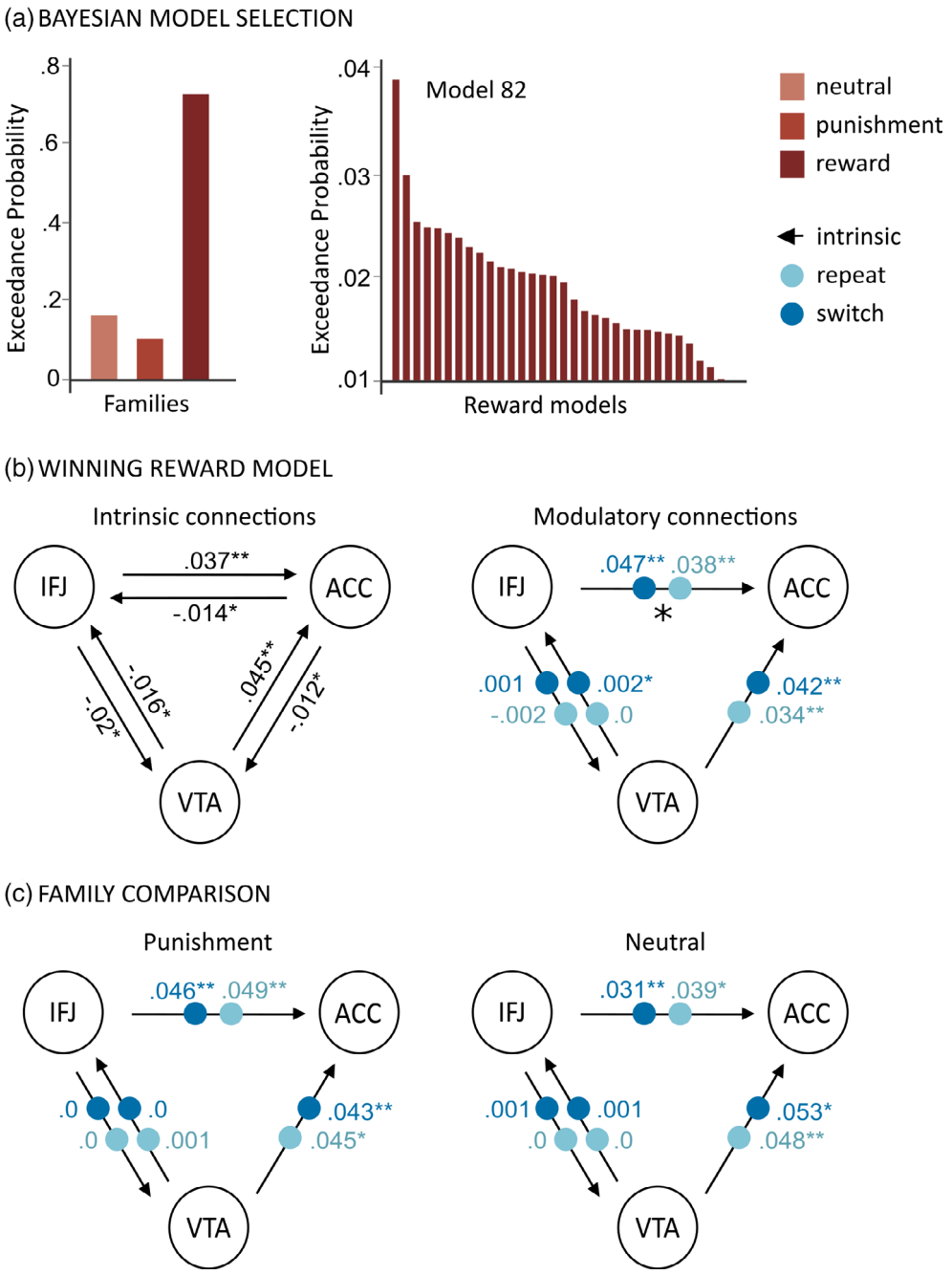

FIGURE 3 DCM results. (a) RFX family-wise inference. Models were grouped into families by motivational input (neutral, punishment, reward, 32 models each). The reward family attained the highest posterior probability with .73. Exceedance probability for the winning reward model was .039 among all 96 models. No neutral or punishment model exceeded a posterior probability of .010. (b) Architecture of winning model. Under the prospect of reward, cognitive control modulates the connections from IFJ to ACC and VTA as well as the connection from VTA to IFJ and ACC. (c) Modulatory parameters of models from the punishment and neutral families with same architecture as winning model. $^{*}p < .05$, $^{**}p < .001$, FDR-corrected; $n = 23$. DCM, dynamic causal modeling; IFJ, inferior frontal junction; ACC, anterior cingulate cortex; VTA, ventral tegmental area

Accordingly, the winning model (Model 82) belonged to the reward family with $p\_ex = .039$ followed by the next best reward model with $p\_ex = .031$ (see Figure 3a, right panel). No neutral or punishment model exceeded a posterior probability of .010. We found significant positive intrinsic connections, independent of cognitive control or motivation, from IFJ and VTA to ACC and significant negative intrinsic connections from ACC to IFJ and VTA as well as from IFJ to VTA and vice versa (Figure 3b, left panel). Moreover, we observed four modulatory connections in the winning reward model: both switch and repeat significantly enhanced connectivity from IFJ and VTA to ACC. Switch trials also significantly increased connectivity from VTA to IFJ, while repeat trials did not. Modulations from IFJ to VTA were not significant (see Figure 3b). Parameter estimates, *T*- and *p*-values of intrinsic and modulatory connections of the winning reward model are listed in Table 3. We compared connectivity strength of switch and repeat input on these modulatory connections. We found significant modulation differences in the connection from IFJ to ACC with stronger positive modulation by switch trials ($t = 2.32$, $p = .030$). All other modulatory connections showed no significant differences between switch and repeat trials (all $t < 1.8$ and $p > .09$). We neither found significant differences in the strength of driving input between IFJ and VTA (all $t < .73$, $p > .47$) nor between switch and repeat trials (all $t < 1.5$, $p > .20$). We further explored modulatory parameters of the model from the punishment and neutral families with the same

TABLE 3 Average parameter estimates of intrinsic connections and input modulations for the winning reward model

	Strength (Hz)	SD	*T*-value	*p*
Intrinsic connections				
IFJ to ACC	0.037	0.040	4.39	.001
IFJ to VTA	−0.020	0.027	−3.41	.007
ACC to IFJ	−0.014	0.023	−3.01	.005
ACC to VTA	−.012	0.018	−3.18	.008
VTA to IFJ	−.016	0.033	−2.31	.030
VTA to ACC	0.045	0.046	4.69	.001
Modulation by switch				
IFJ to ACC	0.047	0.051	4.38	.001
IFJ to VTA	0.001	0.002	0.75	.610
VTA to IFJ	0.002	0.003	2.93	.012
VTA to ACC	0.042	0.034	5.92	.000
Modulation by repeat				
IFJ to ACC	0.038	0.046	3.95	.001
IFJ to VTA	−0.002	0.002	−0.51	.610
VTA to IFJ	0.000	0.004	0.55	.610
VTA to ACC	0.034	0.036	4.49	.001
Switch input				
IFJ	0.327	0.257	6.10	.000
VTA	0.358	0.193	8.88	.000
Repeat input				
IFJ	0.326	0.232	6.74	.000
VTA	0.336	0.197	8.20	.000

Note: SD, standard deviation; IFJ, inferior frontal junction; ACC, anterior cingulate cortex; VTA, ventral tegmental area. *p*-Values were corrected using FDR; $n = 23$.

architecture as our winning reward model (see Figure 3c). Parameter estimates, *T*- and *p*-values of both models can be found in Supplementary Table. We observed that all three models display significant modulation with same valence on the same connections, except for significant positive modulation from VTA to IFJ for switch trials, which was exclusively found in the reward condition. Another unique characteristic of the reward condition is a significant difference between switch and repeat trials in modulation from IFJ to ACC.

4 | DISCUSSION

We aimed to disentangle the interplay between IFJ, ACC, and VTA in motivated cognitive control. Using DCM, we characterized dynamic interactions between these brain regions during task switching under different motivational states. Our results reveal fundamental differences in the behavioral and neural effects of prospective reward and punishment on decision-making.

Participants performed a switch/repeat task embedded in three distinct motivational conditions, namely, prospective monetary reward or punishment, and a neutral condition with no external motivators. For all motivational conditions, switching between tasks led to slower, more error-prone responses compared to repeating tasks, indicating a successful manipulation of cognitive control through our paradigm. Such a disruption of performance is thought to represent the higher cognitive demand inherent to shifts in attention from one competing task to another (Monsell, 2003). This was also reflected on a neural level. Switch trials elicited heightened activity in a number of cortical and subcortical areas that are notably related to cognitive control, such as the SMA, IPS, insula, and midbrain (Bahlmann et al., 2015; Dosenbach et al., 2007; Köhler et al., 2016). Unlike evidenced in prior studies (Derrfuss, Brass, Neumann, & von Cramon, 2005), the IFJ was not differentially activated by switch and repeat trials in our paradigm. Since it did, however, show robust activation across all trials, the area was likely strategically involved in both conditions. Several explanations are possible: First, due to the slow pace of our fMRI design, participants may have treated each trial as a distinct event instead of establishing continual task sets. Thus, the IFJ may have activated task rules anew for every trial. This, however, seems arguable in light of the robust behavioral switch costs observed in the experiment. Second, as prior studies failing to demonstrate switch-related activity (Brass & von Cramon, 2004; Crone, Donohue, Honomichl, Wendelken, & Bunge, 2006), our paradigm may have diminished neural differences between switch and repeat trials by including approximately 50% trials of each type. This composition has been suggested to induce different preparatory processes for task switches than classical task switching designs due to their high frequency (for relevant discussion see Richter & Yeung, 2014).

Task performance differences between switch and repeat trials were modulated differentially by prospective punishment and reward. While a main effect of Motivation revealed that across Control tasks (switch and repeat trials) response speed was enhanced when participants were motivated to avoid punishments as well as when pursuing rewards (compared to the neutral condition), switch costs were reduced exclusively in the punishment condition. This observation concurs with studies showing that potential losses attract more focus of attention and thereby affect decision-making more strongly than equivalent gains (for review, see Yechiam & Hochman, 2013). Given the large body of literature stressing the importance of reward in executive functioning (Botvinick & Braver, 2015; Chiew & Braver, 2014; Jimura, Locke, & Braver, 2010), it nevertheless is remarkable that we found no effect of reward on switch costs. One explanation for this finding might be the notion that the subjective value of money changes depending on its perception as a loss or gain (Tversky & Kahneman, 1979). Thus, in our study, unlike punishment magnitude, reward magnitude (both 7 Cents) might have been too small to effectively reduce switch costs.

Our DCM further suggests that reward and punishment rely on different neural mechanisms to influence executive functioning. We tested whether and how cognitive enhancements through prospective punishment and reward relate to interactions between IFJ, ACC, and VTA, and found that reward is more likely to modulate connectivity between these structures during cognitive processing. Using RFX

family level inference (Penny et al., 2010), we show that changes in causal connections within this network are best explained by models that included modulations through reward-based decision-making.

While it is surprising that the obtained hemodynamic responses were not reflected in behavioral results and vice versa, they support accounts postulating separate motivational systems for reward and punishment with distinguishable behavioral and neural signatures (Camara, Rodriguez-Fornells, & Münte, 2009; Cubillo, Makwana, & Hare, 2019; Galea, Mallia, Rothwell, & Diedrichsen, 2015; Palminteri, Khamassi, Joffily, & Coricelli, 2015; Palminteri & Pessiglione, 2017). Wächter et al. (2009), for instance, showed that reward improved implicit learning and retention mediated through the dorsal striatum, while punishment facilitated online motor performance by modulating insula activity. Others have reported increased amygdala activity in response to prospective punishment, while prospective reward was associated with activity in the ventral striatum (Murty, LaBar, & Adcock, 2012; Yacubian et al., 2006). Relatedly, it has been suggested that unlike reward, punishment does not operate through the dopaminergic but serotonergic neurotransmitter system to enhance executive control and that these systems act as mutual opponents (Daw, Kakade, & Dayan, 2002; den Ouden et al., 2013; Guitart-Masip et al., 2014). However, since we found no neural activations associated with punishment, it is unclear whether this rationale can be applied to our findings. Future studies could shed light on this issue by probing the influence of serotonin and DA antagonists on the effects of punishment and reward on task switching and its neural substrates.

In our winning reward model, intrinsic connections were significantly positive from IFJ and VTA to ACC and significantly negative from ACC to VTA and IFJ as well as between VTA and IFJ. While both switch and repeat trials modulated the connection from IFJ to ACC positively, the modulation was significantly stronger for switch trials. This supports previous studies associating signaling from IFJ to ACC with increased cognitive effort (Harding et al., 2015; Hinault et al., 2019). Importantly, this difference in cognitive modulation was exclusively found in the reward condition suggesting that distinct motivational settings differentially affect neural interactions between IFJ and ACC. The connection from VTA to ACC was positively modulated by switch and repeat trials alike, potentially due to dopaminergic projections between the two areas playing a fundamental role irrespective of task demands (Haber & Knutson, 2010; Hauser et al., 2017; Köhler et al., 2016). Our findings thereby concur with the notion that the ACC receives and integrates signals carrying information about prospective reward and the need to expend control from other brain regions (Shenhav et al., 2013; Shenhav et al., 2016). Complementing prior research (Bahlmann et al., 2015; Hippmann et al., 2019), we found a positive modulatory connection from VTA to IFJ exclusively for switch trials, suggesting that the VTA exerts causal influence on the IFJ when cognitive demand is high. This was observed exclusively in the reward condition emphasizing the importance of projections from the VTA as a source of dopaminergic neuromodulation to the prefrontal cortex in the integration of prospective reward and control demands. However, note that the modulation is comparably weak, and we did not find differential IFJ activation with respect to switch versus repeat trials. As discussed above, our design encompassed a high frequency of switch trials, which may also have decreased a potential interaction between IFJ and VTA. Future studies using revised study designs (i.e., less frequent switches, cued task-switching) are clearly warranted to further corroborate our results.

Taken together, our DCM results show that the IFJ and VTA modulate ACC activity independently to serve task demands in reward-based cognitive control. They further emphasize differences in neural processing of reward and punishment related to executive functions.

5 | LIMITATIONS

A few limitations of the current findings should be noted. First, the paradigm used in our study consisted of incongruent stimuli only (i.e., numbers smaller than 25 were even, numbers greater than 25 were odd). Given the time constraints of fMRI experiments, we wanted to ensure a reasonable number of trials to include in the analysis. Therefore, we chose this task to obtain responses that could unmistakably be classified as correct or incorrect and thus avoid interpretational problems of congruent stimuli. This, however, allows participants to use stimulus features to predict responses in both task sets. Even though none was reported in the debriefing, we cannot rule out that participants applied such strategies, and commend future studies to implicate both congruent and incongruent stimuli to rule out a potential use of alternative strategies.

Second, motivated by specific assumptions about neural interactions between motivation and cognitive control and limited by technical constraints of DCM, we only included a small number of brain regions in the DCM analysis thereby neglecting other areas presumably involved in motivated control, for instance, the ventral striatum (Aarts et al., 2011; Asci, Braem, Park, Boehler, & Krebs, 2019; Cools, 2008). Note, however, that effective connectivity does not reflect anatomical connectivity. Rather, DCM allows to reveal a net effect between relay regions and is intended to answer hypothesis-driven questions. We further recognize that neither of our candidate regions was associated with the main effect of cognitive control. Nevertheless, DCM can detect changes in effective connectivity between VOIs even when void of significant clusters regarding specific contrasts and thus does not depend on activation analysis. In the present study we were particularly interested in disentangling neural interactions with respect to the interface between cognitive control and motivation. We therefore aimed at including areas in the DCM analysis that would relate to all aspects of the task. The results of this study could allow future studies to explore a more extended network.

6 | CONCLUSION

The current study explored the neural interactions between executive functions and distinct qualities of motivation by means of DCM. We

found that structures from the dopaminergic system—specifically the IFJ, VTA, and ACC—contribute to the integration of cognitive control and reward but not punishment. Our findings, therefore, point to different neural mechanisms underlying the influence of reward and punishment on cognitive control.

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CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are openly available at: https://osf.io/j695z/?view_only=26c3d2ae19c2414b80a4b9d0b1ff0943

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of this article.

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