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Research Report

The causal neural substrates underpinning prospective and retrospective sense of agency



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ARTICLE INFO

Article history:

Received 21 August 2024

Revised 8 April 2025

Accepted 14 April 2025

Action editor Katherine Dyke

Published online 20 May 2025

Keywords:

Sense of agency

Neurostimulation

tDCS

Dorsolateral prefrontal cortex

Temporoparietal junction

Libet clock

Intentional binding

ABSTRACT

Sense of agency (SoA) is the subjective feeling of authorship experienced over one's actions and their subsequent sensory outcomes. Despite extensive theoretical accounts of this experience, there remains a dearth of literature investigating its causal neural substrates. To address this, we modulated neural activity of two regions thought to be critical to the agentic experience, namely the left dorsolateral prefrontal cortex (l-dLPFC), and the left temporoparietal junction (l-TPJ). In a sham-controlled, double-blinded, crossover study 104 healthy young adults were stratified to receive anodal stimulation to either the l-dLPFC or the l-TPJ whilst completing an implicit SoA task. Participants performed either an action or outcome binding paradigm in which both prospective (action choice) and retrospective (outcome valence) agency cues were manipulated. Stimulation to the l-TPJ and l-dLPFC produced divergent effects on intentional binding. In the outcome binding condition specifically, anodal stimulation to either target region increased the difference in intentional binding scores between rewarding and punishing action outcomes. We also observed a dissociable, causal role for both the l-dLPFC and l-TPJ on intentional binding, identifying binding specific, but not site specific, effects related to outcome valence. We propose that excitatory stimulation may upregulate the attentional processes relevant to intentional binding.

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1. Introduction

The Sense of Agency (SoA) refers to the experience of oneself as the originator of our actions and their subsequent sensory effects (Gallagher, 2000; Haggard & Tsakiris, 2009). Enabling agents to distinguish one's own actions from those of another agent (Gallagher, 2000; Newen & Voguely, 2003), SoA is

foundational to our experience of, and interaction with, the external world. As a result, this experience is understood to occupy a formative role in healthy cognitive development (Bandura, 2018; Haggard, 2017; Synofzik et al., 2013), with disturbances in the SoA forming a central characteristic of several major psychiatric and neurological conditions (Di Plinio et al., 2019; Saito et al., 2017; Scott et al., 2022; Vogel

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<https://doi.org/10.1016/j.cortex.2025.04.014>

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et al., 2024). Despite this, there remains relatively little evidence mapping the causal neural substrates underpinning the agentic experience (Haggard, 2017).

Cognitive interrogations have resulted in a demarcation in the heterogeneous phenomenology of the SoA, positing that there exists two separable levels at which it is experienced (Synofzik et al., 2008). The first level, the *judgement of agency* (JoA), relies on higher-order cognitive processes to infer causality between one's prior intentions and the generated action itself (Synofzik et al., 2008). However, some components of an action representation, such as the precise details of a motoric plan, may not always be accessible at the level of conscious cognition (Balconi, 2010; Dennett, 1991; Jeannerod, 2006). It is this non-conceptual content of an action that is central to experiences of Synofzik et al.'s (2008) second level of agency, the *feeling of agency* (FoA). The FoA can be understood as the low-level pre-reflective process that emerges during daily actions as an implicit registration within the sensorimotor system as 'self-produced' (David et al., 2008; Synofzik et al., 2008). As a result, while the FoA is *sine qua non* for experiences of the JoA, the inverse does not stand true. Specifically, where an attributive JoA necessitates a reflective processing of the FoA, the FoA can be experienced during instances in which sensorimotor representations of volitional action do not reach conscious awareness (Synofzik et al., 2008). Thus, the two levels of agency are understood as independent agentic experiences underpinned by distinctly dissociable cognitive processes (Dewey & Knoblich, 2014; Moore et al., 2012; Saito et al., 2015; Schwarz et al., 2019).

Given the disparate mechanisms underpinning the two experiences of agency, it follows then that investigations of the FoA necessitate an alternative experimental approach to that employed in studies of the JoA. The FoA is thought to be most robustly measured (Siebertz & Jansen, 2022; Tanaka et al., 2019) using a Libet clock paradigm (Libet et al., 1983) in which SoA is implicitly inferred from the temporal compression between a performed action and its succeeding sensory outcome (e.g., Haggard et al., 2002). This paradigm is grounded in a multi-sensory model of *intentional binding* which proposes that otherwise parcellated sensory components become transiently integrated within the perceptual system to form a spatially and temporally coherent experience of agency (Crick & Koch, 1990; Damasio, 1989). That is, in order to form a fluid experience of the external world, it is not enough to perceive sensory and motor inputs as fragmented representations alone but instead as a continuous perceptual event (Damasio, 1989).

This temporal binding can, however, occur in one of two directions, according to the constituent mechanism under investigation (Tanaka et al., 2019). Where *action binding* denotes the temporal shift of an action towards its succeeding outcome (e.g., Cao et al., 2021; Christensen et al., 2016; Di Plinio et al., 2019), *outcome binding* instead reflects an inverted perceptual pattern, with a shift in the outcome towards its preceding action (e.g., Suzuki et al., 2019; Tanaka et al., 2019). Recent examinations of these two components found that temporal shifts in one were wholly unrelated to shifts in the other, suggesting that the two mechanisms represent independent aspects of the agentic experience (Tonn et al., 2021). While there is yet a theoretical framework conclusively outlining the specific processes for which each of these

binding components govern, there is some evidence to suggest that action binding reflects learned action-outcome contingencies and outcome binding arises from the ability to temporally predict the onset of an action's outcome (Tanaka et al., 2019).

In such that the SoA can be seen to vary according to binding measurement, it is also determined by characteristics intrinsic to the performed action itself. According to the theory of *optimal cue integration*, for one to construct an experience of agency that is valid and robust, the perceptual system must assimilate prospective and retrospective action information proportionately according to its reliability, availability, and salience within a given context (Ernst & Bühlhoff, 2004; Moore & Haggard, 2008; Synofzik et al., 2009, 2013). When informed primarily by *prospective* cues, SoA can be understood as an experience grounded in the perceptual and cognitive processes that precede the performance of a volitional action. For instance, action authorship may be prospectively determined by way of a predictive comparison between efferent signals transmitted during the premotor stage of an action operation and re-afferent signals produced from the executed action itself (Frith, Rees, & Friston, 1998; Frith et al., 2000; Blakemore et al., 2002). It is only when the predicted action representation and executed action feedback are congruent with one another that a SoA is then experienced. Alternatively, SoA may also emerge *retrospectively*, according to an action's sensory outcome. For authorship to be ascribed retrospectively, an action's outcome must occur with no plausible alternative source, be preceded by a corresponding intention to act, and is consistent with the action's predicted outcome (Wegner, 2003; Wegner & Wheatly, 1999).

While internal efferent signals typically offer access to information about one's actions with the least noise (Wolpert & Flanagan, 2001), there are contexts in which such signals might be outweighed or even replaced by information derived from external sensory feedback. As such, the malleable weight with which prospective and retrospective cues contribute to one's SoA serves to create an experience of authorship that is flexible across variable contexts (Moore & Fletcher, 2012; Synofzik et al., 2009, 2013; Synofzik & Voss, 2010).

In light of this unified framework, research has sought to better understand the specific conditions under which retrospective cues might outweigh prospective sensorimotor signals in informing the SoA. One such factor is the affective valence of an action's outcome. Extant research suggests that implicit SoA is strengthened when an action elicits a positive outcome and weakened when the succeeding outcome is negative (Christensen et al., 2016; Yoshie & Haggard, 2013, 2017). Authors argue that this affective pattern of agency reflects a self-serving bias, that is, the general propensity for individuals to distance themselves from negative events and claim authorship for positive ones (Bradley, 1978; Greenberg et al., 1992; although see conflicting evidence when learning from negative outcomes, Majchrowicz et al., 2020).

Yet, existing research suggests that implicit SoA is informed not only by retrospective inferences about the valence of an action's outcome but also by prospective information pertaining to the number of action choices available

(Barlas & Kopp, 2018; Barlas & Obhi, 2013; Tanaka & Kawabata, 2021). Accordingly, Barlas and Kopp (2018) found that implicit SoA is at its greatest when an action is both freely chosen and succeeded by a positive outcome. Such findings are consistent with Pettit's (2001) theoretical notion of freedom which posits that executing an action without interference is alone not enough to inform the agentic experience. Instead, the feeling of being the agent of one's own actions is best brought about in an "environment of opportunity" within which an individual is able to freely choose which action to make.

This instrumental role of choice in experiences of agency has also been reflected at the level of neural activation. In particular, previous research reports that the dorsolateral prefrontal cortex (dlPFC), a region understood to be a response space in which available action alternatives are represented (Fletcher et al., 2000; Rowe et al., 2010), shows elevated activation during the execution of freely chosen, but not instructed, voluntary actions (Filavich et al., 2013). As such, the dlPFC is thought to play a critical role in processing prospective components of SoA, particularly during the stage of action selection (Chambon et al., 2013). This notion is consistent with research using transcranial direct current stimulation (tDCS) which maintains this region's function as one of prospective action monitoring. TDCS is a non-invasive brain stimulation technique which delivers a weak electrical current to a target region of the brain either increasing (anodal) or decreasing (cathodal) neuronal excitability. Using tDCS, Khalighinejad et al. (2016) found that anodal l-dlPFC stimulation produced a significantly stronger intentional binding effect, but only during trials in which participants' actions were freely selected. These findings suggest that upregulating the action selection processes governed by the l-dlPFC strengthens the perception of oneself as the generator of freely performed actions, and in turn, SoA.

Non-agency (or reduced agency) refers to the experience of feeling that one's actions or decisions are not fully under their own control, often leading to a sense of detachment or lack of ownership over one's behaviour. Experiences of non-agency are thought to involve the posterior temporoparietal region (Hughes, 2018; Khalighinejad & Haggard, 2015). Existing neuroimaging evidence suggests that this region is responsible for registering error signals transmitted about discrepancies between expected and observed action consequences (Sperduti et al., 2011; Uhlmann et al., 2020; Zito et al., 2020a, 2020b). As such, the TPJ has been functionally conceptualised as a centre in which violations to the SoA are retrospectively tracked by detecting mismatches between internal sensorimotor predictions and external sensory feedback (Farrer et al., 2003; Farrer et al., 2008). Indeed, in one of the few studies to have examined this region's role at a causal level, Khalighinejad and Haggard (2015) found that anodal tDCS targeting the left TPJ produced a significantly reduced implicit SoA over voluntary actions.

The current preregistered study will extend on this existing evidence base to provide the first high-powered investigation into the causal role that the left dlPFC and TPJ play in processing prospective, retrospective, and temporal components of implicit SoA. Building on the previous work of Khalighinejad and colleagues (2015; 2016), we employed two intentional binding paradigms (action and outcome binding)

in which both the prospective factor of action choice and retrospective factor of outcome valence were manipulated while participants received anodal stimulation to one of two target brain regions. In an effort to provide a robust and valid account of the SoA's neural substrates, we employed a more spatially accurate neurostimulation technique (Bortolotto et al., 2016; Gbadeyan et al., 2016) than that reported in previous studies; namely, focal tDCS (f-tDCS). In light of these methodological changes and the extant literature, we expected anodal stimulation to have dissociable effects on intentional binding according to the target brain region. Specifically, we propose that intentional binding will be weakened following anodal l-TPJ stimulation, and that this effect will be greatest when participants' actions are followed by a punishing outcome. We also predicted that anodal stimulation targeting the l-dlPFC, but not l-TPJ, would produce a stronger intentional binding effect during trials in which participants were given a choice over their performed action.

2. Methods

The current study was preregistered on the Open Science Framework (<https://doi.org/10.17605/OSF.IO/UK9SF>).

2.1. Participants

A priori power analyses using G*Power 3.1 (Faul et al., 2007) indicated that a sample size of 26 for each study arm (total $N = 104$) was necessary to detect a Cohen's f score of .33, with 80% power, and an alpha level of $< .05$. The Cohen's f value was guided by previous SoA research using tDCS which reported a medium-to-large effect size (Khalighinejad & Haggard, 2015; Khalighinejad et al., 2016). Participants aged 18–35, with normal or corrected-to-normal vision, and no history of psychiatric or neurological conditions were eligible for inclusion. Individuals who disclosed irremovable electrical medical equipment in the head or reported a history of migraines or seizures were precluded from participation. One-hundred-and-thirteen participants who met the above criteria were recruited via a Research Participation Scheme at the University of Kent ($n = 95$) and Durham University ($n = 18$). Following post-hoc attention checks, nine participants were excluded for failing to follow task instructions on at least 80% of trials, resulting in a final sample of 104. All participant procedures were implemented in compliance with the ethical standards outlined in the Declaration of Helsinki and ethical approval was granted by the Psychology Ethics Committee at the University of Kent and Durham University.

2.2. Design

Participants were stratified to receive either l-dlPFC or l-TPJ stimulation while completing either an action or outcome binding paradigm. A $2 \times 2 \times 2 \times 2$ mixed-factorial design was used with brain region (l-TPJ or l-dlPFC) and binding mechanism (action or outcome) as between-subjects factors and stimulation condition (anodal or sham), action choice (free or forced), and outcome valence (rewarding or punishing) as within-subjects factors. The presentation of all within-

subjects factors was randomised across trials and stimulation sessions.

2.3. Materials and measures

2.3.1. Libet Clock task

Intentional binding was measured using a Libet clock task in which the prospective factor of action choice and retrospective factor of outcome valence were manipulated. Participants were informed that the task entailed a trial-by-trial point loss/gain structure indicated by one of two tones (cash machine or klaxon) and would receive a small reward if they were to score within the top 25% of the study cohort. However, outcome tones were non-contingent to their preceding action and all participants received a reward following participation. In each trial, participants were presented with a clock face around which a black dot rotated, completing a revolution every 2000 msec (see Fig. 1). The centre of the clock face was labelled with a fixation cross for a random interval of 750–2000 msec, after which it was replaced by an arrow cue (“<”, “>”, or “<>”) for 500 msec. In baseline action binding blocks, participants performed a key press in correspondence with the presented arrow cue but did not receive an outcome (tone), while in baseline outcome binding blocks, participants perceived one of two tones without performing an action (key press). In operant blocks for both binding conditions, participants performed an action (freely chosen or instructed key press) succeeded by an outcome tone (rewarding or punishing). Following each trial, participants were instructed to record, according to the clock face, either the time at which

they performed the key press (action binding) or heard the tone (outcome binding). Responses in all blocks were reported as the minute value on the clock (0–60) and were recorded on a standard keyboard.

The task comprised of two baseline blocks and seven operant blocks. The first baseline block contained 32 trials, the first four of which were excluded to account for familiarisation effects. All subsequent operant blocks contained 24 trials, followed by a final baseline block of 28 trials.

2.3.2. Data processing

Following the exclusion of trials outside 3 standard deviations of participants' mean, judgement error scores were calculated according to binding condition by subtracting a) the reported time of key press from the actual time of key press (action binding) or b) the reported outcome onset from the actual outcome onset (outcome binding) in each trial. Judgement error scores were then averaged across all within-subjects conditions, resulting in 16 outcome binding and 12 action binding mean judgement error scores (mJE) for each factorial combination within baseline and operant blocks. Operant mJE scores were subtracted from each baseline mJE score to produce eight outcome binding and six action binding perceptual shift scores for each factorial condition, reflecting intentional binding. Positive action binding and negative outcome binding scores are indicative of a greater perceptual shift and, in turn, intentional binding effect. In order to account for these directional differences and compute statistical comparisons between the two binding conditions, raw outcome binding scores were inverted prior to analyses.

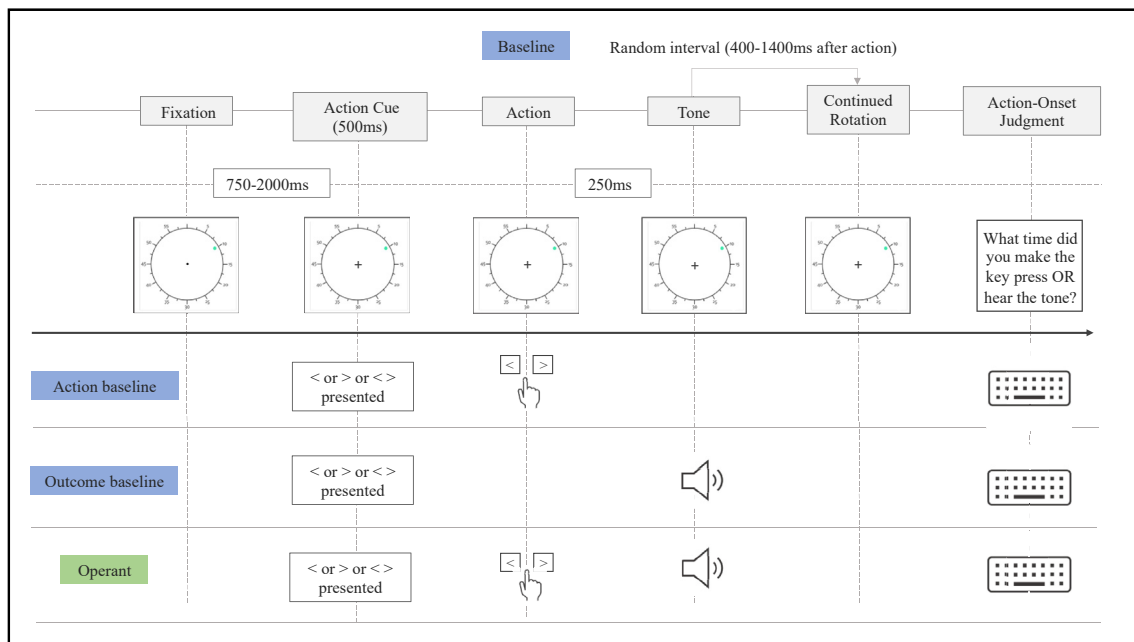


Fig. 1 – Schematic illustration of SoA Libet Clock Task.

Note. Schematic illustrates baseline and operant blocks across both action and outcome binding tasks. Participants stratified to the action binding group were instructed to record the time they performed the keypress at the end of each trial. Those in the outcome binding group were instructed to record the time they heard the outcome tone. Participant responses were reported as the minute value on the clock at the time of the event (keypress or tone).

2.3.3. Psychometric questionnaires

To identify and exclude those at an elevated risk of experiencing adverse stimulation effects, all participants completed a tDCS safety screening questionnaire. Differences in mood and anxiety between participant groups (l-TPJ and l-dlPFC; action and outcome binding) were assessed using total scores on the Beck Depression Inventory (BDI; Beck, Steer & Brown, 1966) and Beck Anxiety Inventory (BAI; Beck & Steer, 1990). In addition, the Visual Analogue of Mood Scale (VAMS; Folstein & Luria, 1973) was employed to assess intraindividual changes in mood within and across stimulation sessions. The difference between pre- and post-stimulation scores was calculated to assess positive and negative mood change for sham and anodal sessions. Finally, the presence and extent of adverse stimulation effects was assessed by comparing participants' total scores on the Adverse Effects Questionnaire between sham and anodal sessions (adapted from Brunoni et al., 2011).

2.3.4. Equipment

Questionnaire data was collected via Qualtrics (2019) and the cognitive task was completed on Psychopy (Peirce et al., 2019) using a standard computer monitor and keyboard.

2.3.5. Focal tDCS

Neural activity was modulated using a single-channel direct current stimulator (DC-Stimulator Plus, NeuroConn), with a central electrode (2.5 cm diameter) and return electrode (inner/outer diameter of 7.5 cm and 9.8 cm respectively) attached using adhesive electroconductive gel and an EEG cap. The central electrode determines stimulation polarity, in this case anodal, and the return electrode limits electrical current to the target region, preserving spatial focality (Martin, et al., 2019a; Martin et al., 2019b; Villamar, Wivatvongvana, et al., 2013; Villamar, Volz, et al., 2013). A stimulation current of 1 mA was delivered during anodal sessions for 20-min with a 5-s ramp-up and down period. In the sham condition, stimulation was delivered for 40-s between the ramp-up and ramp-down period to simulate a cutaneous sensation similar to that experienced during the anodal condition, but with no neurophysiological effect. Current modelling was completed using SimNIBS version 4.1 (Thielscher et al., 2015). Parameters were selected to estimate current delivery according to EEG 10–20 guided electrode positions, F3 for the l-dlPFC and CP5 for the l-TPJ, with the return electrode placed equidistantly around the centre electrode. Electrode and gel thickness were modelled as 2 and 1 mm respectively. Standard conductivity values as provided by SimNIBS were used. We present the theoretical cortical electrical field (magnitude E) for anodal focal-tDCS to both the l-dlPFC and the l-TPJ (see Fig. 2).

2.4. Procedure

Participants attended two 90-min sessions separated by a minimum of 48 h to mitigate against potential carryover stimulation effects. In the first session, participants completed the BAI, BDI, VAMS, and tDCS safety screening form. Electrodes were attached and stimulation commenced, followed immediately by Libet Clock task onset. The delivery

of anodal and sham stimulation was randomised and counterbalanced across participants. Double blinding was achieved using study mode, whereby anonymised codes were input to set stimulation parameters for each session. Following task completion, participants completed a second VAMS and the Adverse Effects Questionnaire. This procedure was repeated during the second session (with exception of the tDCS safety screening, BDI, and BAI), followed by a question requiring the participant to guess which session they received the real stimulation, to assess blinding efficacy.

3. Results

3.1. Adverse effects, demographics, Mood Scales, and blinding

Between-subjects ANOVAs indicated that there were no significant differences in self-reported anxiety or depression between brain region and [BDI: $F(1,100) = .00, p = .960, \eta^2_p < .001$; BAI: $F(1,100) = .06, p = .800, \eta^2_p .001$; See Table 1] and binding mechanism [BDI: $F(1,100) = .19, p = .667, \eta^2_p .002$; BAI: $F(1,100) = .23, p = .635, \eta^2_p .002$] groups. Adverse stimulation effects were not significantly different between sham ($M = 12.1, SD = 4.7$) and anodal ($M = 12.5, SD = 4.7$) sessions, $t = -1.34, p = .183, d = -.13$. Changes in negative mood were not significantly different between sham and anodal stimulation sessions, $t = -1.27, p = .206, d = -.13$. We observed a reduction in positive mood following both sham and anodal stimulation. This change, however, was significantly greater following sham than anodal stimulation, $t = -2.18, p = .032, d = -.21$. A chi-square test of difference indicated that significantly more participants in the l-TPJ group guessed the anodal stimulation session correctly ($N = 39$) than incorrectly ($N = 13$), $X^2(1, 102) = 13.00, p < .001$. In the l-dlPFC group, however, participants did not correctly guess the anodal session above that of chance, $X^2(1, 102) = .72, p = .396$.

3.2. Libet Clock task

A $2 \times 2 \times 2 \times 2$ mixed effects ANOVA¹ revealed a significant main effect of outcome valence, $F(1,100) = 44.49, p < .001, \eta^2_p .31$, with rewarding outcomes ($M = 86.6, SD = 87.1$) producing a greater intentional binding effect than punishing outcomes ($M = 62.0, SD = 66.6$). However, the main effect of action choice was not significant, $F(1,100) = 2.46, p = .120, \eta^2_p .02$, indicating that intentional binding scores did not meaningfully differ between trials in which participants had free choice over the performed key press ($M = 77.1, SD = 77.8$) and those in which key presses were instructed ($M = 71.5, SD = 76.3$). Neither stimulation type $F(1,100) = .30, p = .584, \eta^2_p .003$ nor brain region yielded a significant main effect $F(1,100) = 2.68, p = .105, \eta^2_p .03$, indicating that intentional binding was not substantially different between anodal ($M = 72.4, SD = 79.2$) and sham conditions ($M = 76.2, SD = 87.0$).

¹ Note: Two multivariate outliers were identified prior to analysis. However, the conclusions were not affected with or without their exclusion, so we present all data.

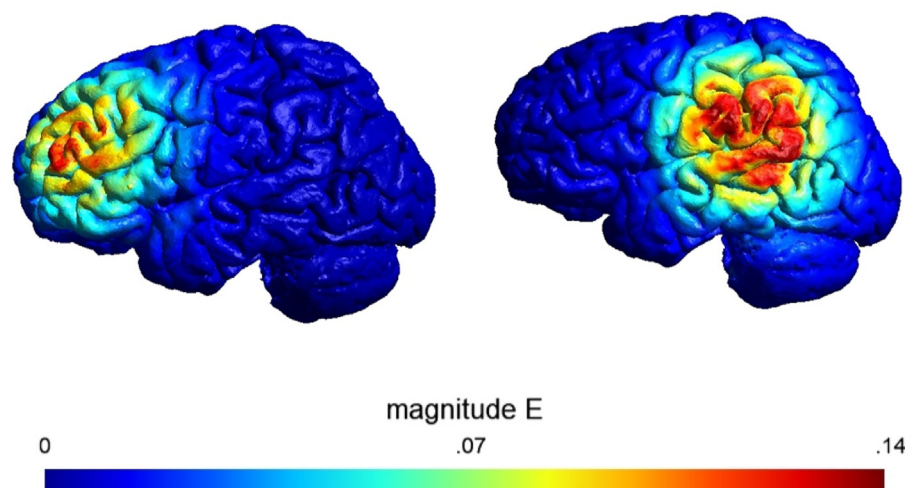


Fig. 2 – F-tDCS Current Modelling Across l-dlPFC and l-TPJ regions.

Note. Figures depict the flow and spread of electrical current when delivered at 1ma to the l-dlPFC and l-TPJ respectively.

Table 1 – Demographic characteristics of participants across experimental arms.

		N	Age	BDI	BAI
l-dlPFC	AB	26 (19 F)	20.0 (2.3)	8.6 (8.4)	11.5 (11.8)
	OB	26 (19 F)	20.3 (2.4)	8.8 (6.9)	8.2 (5.9)
l-TPJ	AB	26 (22 F)	20.4 (3.0)	9.3 (9.6)	9.6 (10.5)
	OB	26 (21 F)	19.4 (1.2)	7.8 (5.4)	11.0 (9.6)

Note: AB: action binding; OB: outcome binding; BDI: Beck Depression Inventory; BAI: Beck Anxiety Inventory. Table presents the means and standard deviations in brackets for participant age, BDI, and BAI scores. Gender is presented in brackets under the N column. No participants identified as non-binary.

or between l-dlPFC ($M = 86.8$, $SE = 8.7$) and l-TPJ ($M = 63.8$, $SE = 8.7$) participant groups. We did observe a significant main effect of binding condition $F(1,100) = 34.29$, $p < .001$, $\eta^2_p = .26$, with a greater intentional binding effect in the outcome ($M = 114.3$, $SE = 8.7$), relative to action ($M = 36.2$, $SE = 8.7$), binding condition. See Table 2 for full breakdown of intentional binding scores.

The two-way interaction between brain region and stimulation type yielded a significant effect $F(1,100) = 4.24$, $p = .042$, $\eta^2_p = .04$. Post-hoc simple effects analyses indicated that intentional binding was significantly greater following anodal stimulation to the l-dlPFC ($M = 91.2$, $SD = 86.7$) compared to when anodal stimulation was delivered to the l-TPJ ($M = 55.0$, $SD = 67.3$; see Fig. 3), $t(102) = 2.29$, $p = .024$, $d = .45$. Conversely, sham stimulation yielded no significant differences in intentional binding scores between l-dlPFC and l-TPJ regions, $t(102) = .37$, $p = .713$, $d = .07$. This reduction in intentional binding was driven primarily by anodal stimulation administered at the l-TPJ site, although this just failed to reach significance, $t(51) = 1.98$, $p = .053$, $d = .28$. The increase in intentional binding at the l-dlPFC site was not significant, $t(51) = -.98$, $p = .331$, $d = -.14$.

We also observed a significant two-way interaction between outcome valence and binding mechanism $F(1,100) = 13.11$, $p < .001$, $\eta^2_p = .12$. Post-hoc t-tests revealed that although outcome valence produced a significant effect on action binding scores, $t(51) = 2.14$, $p = .03$, $d = .17$, the effect on outcome binding was greater, $t(51) = 7.23$, $p < .001$, $d = .56$ (see Fig. 4). However, this two-way interaction was subsumed under a three-way interaction with stimulation, $F(1,100) = 4.16$, $p = .044$, $\eta^2_p = .04$. Mean intentional binding scores across factorial conditions indicated that anodal stimulation increased the difference between rewarding and punishing outcomes, but only when outcome binding was the

Table 2 – Mean intentional binding scores across all factorial levels.

				Free	Forced
Action binding	l-dlPFC	Sham	R	46.3 (61.4)	40.6 (54.7)
			P	42.4 (63.9)	31.1 (51.3)
		Anodal	R	45.3 (61.1)	31.3 (45.3)
			P	29.7 (35.5)	16.7 (74.2)
	l-TPJ	Sham	R	68.9 (87.4)	136.0 (117.0)
			P	51.2 (81.6)	35.9 (63.1)
		Anodal	R	26.3 (48.8)	35.7 (56.2)
			P	16.1 (45.1)	31.8 (54.8)
Outcome binding	l-dlPFC	Sham	R	136.5 (124.8)	50.9 (73.1)
			P	106.4 (109.8)	95.4 (112.9)
		Anodal	R	178.9 (107.3)	178.3 (94.2)
			P	119.7 (78.9)	118.6 (85.8)
	l-TPJ	Sham	R	113.6 (114.2)	93.2 (105.0)
			P	87.4 (81.4)	83.0 (97.7)
		Anodal	R	102.6 (89.4)	101.6 (95.3)
			P	61.8 (91.9)	64.1 (82.0)

Note. Data represents the means and standard deviations (presented in parentheses) of intentional binding scores in milliseconds (ms) across all factorial levels. Notation R and P represent rewarding and punishing outcome conditions respectively. Higher values represent greater binding. Outcome binding scores were reversed for ease of interpretation and comparison.

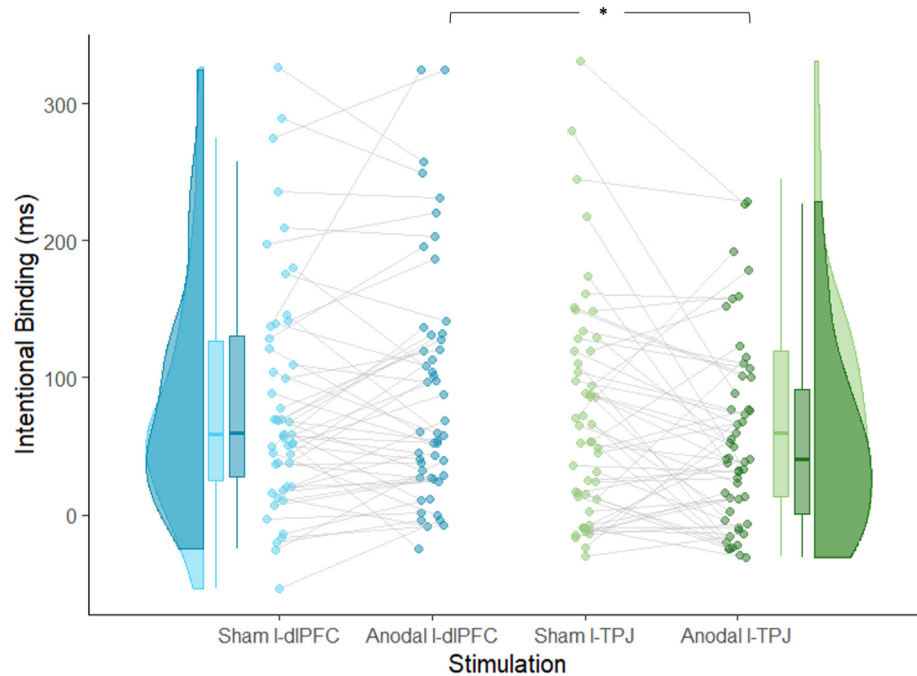


Fig. 3 – The Difference in Participants' Mean Intentional Binding Scores Across Stimulation Conditions and Brain Region.

Note. Graph presents the mean intentional binding scores in milliseconds for anodal and sham conditions across l-dlPFC and l-TPJ participants groups. Anodal stimulation significantly increased the difference in intentional binding scores between l-dlPFC and l-TPJ groups. Boxplots present the median, interquartile range, and the distribution of outcome binding scores within each stimulation condition.

mechanism of measurement. To examine this effect statistically, two 2×2 RM-ANOVAs were computed between stimulation type and outcome valence, for both action and outcome binding separately. The interaction between stimulation and outcome valence was significant only for outcome binding, $F(1,51) = 5.02$, $p = .030$, $\eta_p^2 = .09$, but not action binding, $F(1,51) = .01$, $p = .923$, $\eta_p^2 < .001$, task. Post-hoc t -tests showed that stimulation did not significantly shift rewarding, $t(51) = 1.30$, $p = .396$, $d = .18$, or punishing outcomes specifically, $t(51) = -.86$, $p = .396$, $d = -.12$, but rather increased the difference between the two valence conditions (see Fig. 5). No other interaction effects were significant (see Supplementary Table A for full statistical model).

Due to the complex nature of the planned analysis, we also present the results separately for each study arm in the supplementary materials section (see supplementary Table B).

4. Discussion

The aim of the current study was to present causal evidence for the role of the l-dlPFC and l-TPJ in SoA. We also assessed specific effects relevant to the processing of prospective (action choice) and retrospective (outcome valence) factors on intentional binding as measured by both action and outcome binding within a Libet Clock paradigm. Partially supporting our hypotheses, we identified reduced intentional binding following l-TPJ stimulation compared to stimulation delivered to the l-dlPFC. However, contrary to our hypotheses, the

reduction in intentional binding due to excitation of the l-TPJ was not specific to punishing outcomes and the increase in intentional binding due to excitation of the l-dlPFC was not specific to freely chosen actions. In addition, a common effect was associated with excitation of both regions. Specifically, in the outcome binding condition only, anodal stimulation polarised the effect of valence on SoA, with increased intentional binding for rewarding outcomes and decreased intentional binding for punishing outcomes.

Findings from the present study do not support the notion that the SoA is prospectively informed by the ability to freely select amongst action alternatives. Rather, we observed that participants' SoA over freely chosen actions was not substantially different to that experienced over actions performed under instruction. This absent effect of choice likely reflects an insufficient “environment of opportunities” presented within the task design of the current study (Pettit, 2001). Particularly, while participants were able to select between action alternatives, this was arguably substantially constrained by the availability of only two choices (Barlas & Obhi, 2013). Further compounding this effect, neither action choice presented was meaningfully different from the other and both were unpredictable in the outcome that they produced, likely weakening participants' perception of ‘free choice’ (Lukitsch, 2020).

Another possible explanation for the null effect of choice is grounded in the premise of the What-Whether-When model of intentional action (Brass & Haggard, 2008; Haggard, 2008); namely that intentional action comprises of three core

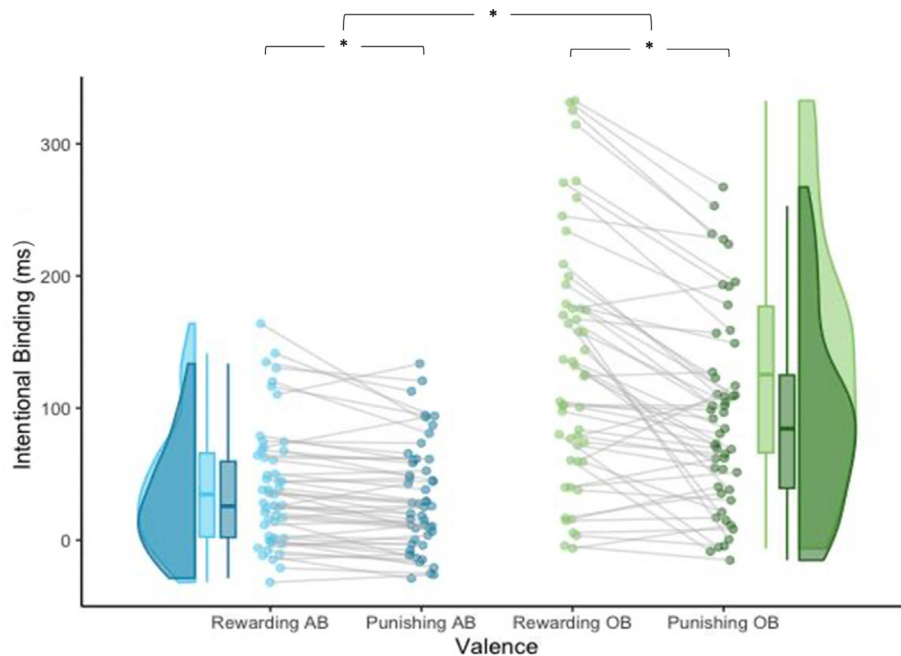


Fig. 4 – The Effect of Outcome Valence Across Action and Outcome Binding Conditions.

Note. AB: action binding; OB: outcome binding. Figure presents individual mean binding scores for the effect of outcome valence across both action binding and outcome binding participant groups. Relative to action binding, participants in the outcome binding group showed a significantly greater difference in intentional binding scores between rewarding and punishing outcomes. Boxplots present the median, interquartile range, and the distribution of outcome binding scores within each stimulation condition.

decisional components, that is, selecting *what* action to perform, *whether* or not to perform it, and *when* in time it will be performed. While the free choice condition in the current study arguably fulfilled both what and when components, participants were prohibited from not acting at all should they have wished. Recent research by Zanutto et al. (2023) suggests that constraining this *whether* component may have attenuated the effect of freely chosen actions on SoA. Here authors found that even when participants were able to freely choose between key presses but the decision of whether or not to execute that action was externally determined, SoA was significantly and detrimentally impacted (Zanutto et al., 2023). We therefore propose that existing conceptualisations surrounding the prospective registration of choice during action selection has a perhaps more nuanced effect on SoA than has been previously considered. As such, our findings do not necessarily discount a prospective account of agency, but instead highlight that the conditions under which freedom over action selection is experienced may not always be met by existing SoA paradigms.

In light of a null effect for action choice on SoA at a cognitive level, it is perhaps unsurprising then that upregulating neural activity within the l-dlPFC also did not significantly strengthen participants' SoA over freely selected actions. Indeed, despite a number of studies detailing the critical role that this region plays in monitoring the action selection processes informing one's SoA (Chambon et al., 2013; Khalighinejad & Haggard, 2015; 2016; Rowe et al., 2010), we failed to find any evidence

to this effect. In addition to the cognitive considerations detailed above, these contradictory findings may also reflect critical differences in neurostimulation techniques employed between Khalighinejad et al.'s (2016) study and our own. Rather than conventional tDCS, as used in previous research (e.g., Khalighinejad et al., 2015; 2016), we present findings using focal tDCS, a method which has been shown to yield up to 80% greater spatial focality (Dmochowski et al., 2011) and 7–12% higher current dosage to target regions (Niemann et al., 2024) than traditional techniques. The distribution of electrical current in standard montages of conventional tDCS is diffuse and in many instances extends beyond that of the delimited target region (DaSilva et al., 2015). It is therefore possible that in targeting the l-dlPFC, other regions, outside of those under investigation, may have also undergone an upregulation in neural activity. One particular region which may account for these disparate stimulation effects is the pre-supplementary motor cortex (pre-SMA; Cavazzana et al., 2015; Seghezzi et al., 2019). The pre-SMA is not only proximal to the l-dlPFC but has also been intimately implicated in prospective SoA processes such as motor planning and action initiation (Fried et al., 2011; Hoffstaedter et al., 2013; Jahanshahi et al., 1995; Moore et al., 2010; Nachev et al., 2008; Van Gaal et al., 2011; Zapparoli et al., 2018).

However, it should be also noted that the current dose of focal tDCS may be reduced by electrode positioning errors related to the approximate nature of using EEG coordinates to locate the l-dlPFC (Niemann et al., 2024). As such, the null

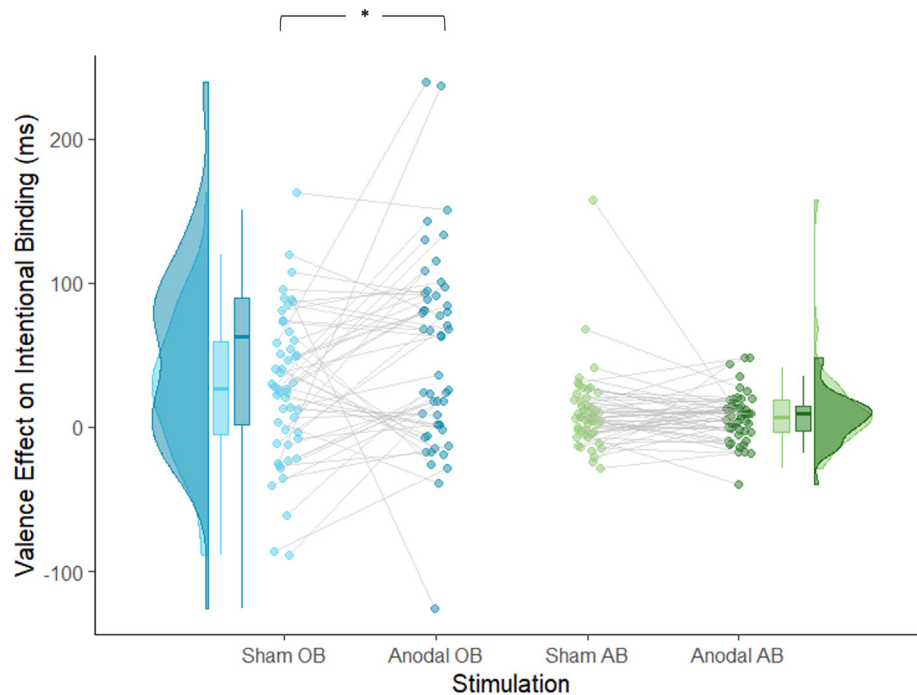


Fig. 5 – The Effect of Stimulation on Outcome Valence Across Binding Conditions.

Note. Figure presents individual intentional binding scores in milliseconds for the effect of outcome valence across stimulation and binding factorial levels. The effect of valence is presented here as the mean difference in intentional binding scores between rewarding and punishing action outcomes. Relative to sham stimulation, anodal stimulation increased the difference in intentional binding scores between rewarding and punishing outcomes, in the outcome binding condition only. Boxplots present the median, interquartile range, and the distribution of outcome binding scores within each stimulation condition.

interaction between action choice and anodal l-dlPFC stimulation observed in the current study could reflect either a failure to sufficiently engage the l-dlPFC when choices are meaningless and limited in scope, or a methodological limitation of using focal tDCS to target specific brain regions without precise neuronavigation. As such, further research interrogating cognitive, neural, and methodological explanations for the inconsistent effects of neurostimulation to the dlPFC is required.

Despite failing to observe a significant effect of prospective cues, our analyses did yield a large effect of retrospective cues on implicit SoA. Specifically, in line with existing research, we found that rewarding outcomes elicited a stronger SoA than was observed for actions followed by a punishing outcome (Barlas & Kopp, 2018; Christensen et al., 2016; Scott et al., 2022; Tanaka & Kawabata, 2021; Yoshie & Haggard, 2013). As a result, our findings are consistent with the notion that SoA is inferred retrospectively according to the specific nature of an action's sensory consequence. Extant conceptualisations of the SoA suggest that this valence effect serves as a psychological mechanism through which individuals are able to preserve self-esteem and encourage future goal-directed behaviour (Yoshie & Haggard, 2017). This apparent positivity bias is thus understood as an adaptive mechanism of self-protection which is critical to upholding both mental and physical wellbeing (Taylor & Brown, 1994).

Examining how the effect of outcome valence was modulated by excitatory stimulation, we observed a novel effect. Namely, our findings indicate that in the outcome binding condition only, anodal stimulation exaggerated the difference in SoA experienced between rewarding and punishing action outcomes. However, despite our expectation that a weakened SoA over punishing outcomes would be related to elevated l-TPJ activity exclusively, we found that this effect was not specific to either target brain region.

The differential effects observed for action and outcome binding in the current study align with existing literature attesting to the divergent cognitive processes with which each of the two mechanisms govern (Tonn et al., 2021). Though there is scant research delineating the specific facets of agency to which action and outcome binding shifts relate, recent work by Tanaka et al. (2019) suggests that action binding might best reflect an ability to predict the content of an action's outcome, while outcome binding instead occurs when the temporal onset of an outcome can be accurately predicted. The consistent 250 millisecond onset of auditory outcomes in all trials of the current study may have therefore served to strengthen outcome shifts and, in turn SoA, by means of temporal prediction. Moreover, as rewarding and punishing tones were distributed randomly across trials irrespective of the action performed, participants were unable to learn which actions were more likely to produce a desirable outcome, thus potentially weakening succeeding action shifts.

We propose that it is these mechanistic differences between binding components that are central to the effect of valence observed in the outcome binding condition of our study specifically. In line with literature on action expectancy effects, we advance that our findings reflect the anticipation of a particular, in our case rewarding, outcome when an action is performed. This prediction may take form as a preactivated neural representation of the expected outcome (Hon, 2023). In accordance with the self-positivity framework outlined previously (Yoshie & Haggard, 2013), an action may preactivate a network associated with the prediction of a positive outcome. Excitation to either the l-TPJ or l-dlPFC may have therefore facilitated this preactivation, increasing the resulting prediction, and, in turn, too the difference in intentional binding between rewarding and punishing outcomes. Reward expectation has been associated with activity and connectivity within the prefrontal cortex, including the l-dlPFC (Rowe et al., 2010). The expectation of a rewarding outcome could therefore have served as a motivating factor, driving the maintenance of cognitive control and effort. As increased mental effort has been associated with attenuated intentional binding (Howard et al., 2016), stimulation of the left frontoparietal network (TPJ and dlPFC) may have strengthened cognitive processes related to maintaining the reward expectancy as well as the mental effort and cognitive control required to complete the task.

Finally, we also observed a global effect of stimulation on SoA according to target brain region. While SoA did not significantly differ across brain region groups during sham stimulation, we observed reduced intentional binding following anodal l-TPJ stimulation when compared stimulation delivered to the l-dlPFC. This finding fits within existing conceptualisations of the two regions and the respective roles that each play in monitoring perceptual experiences of agency. In particular, although we found only a slight increase in binding following l-dlPFC stimulation, this effect is somewhat consistent with research claiming a role for the dlPFC in registering actions for which individuals feel a sense of authorship (Chambon et al., 2013). This notion is supported by studies examining disturbances in the SoA amongst those with functional movement disorders. Functional movement disorders refer to a group of conditions, including Tourette's, Parkinson's, and some symptom profiles of schizophrenia, in which individuals report an experience of involuntary movement despite the physiological markers of voluntary action. Neuroimaging research within this population suggests that the diminished SoA experienced over self-produced actions is associated with a reduced activation of the dlPFC (Nahab et al., 2017), thus underscoring the importance of this region's role in informing experiences of willed action.

Similarly, the finding that anodal stimulation to the l-TPJ reduced implicit SoA over voluntary actions, when compared against stimulation to the l-dlPFC, is consistent with the role of this region in registering experiences of reduced agency (Farrer et al., 2003; Hughes, 2018; Sperduti, et al., 2011; Uhlmann et al., 2020; Zito, Wiest, & Aybek, 2020). Previous neurostimulation research examining the role of the TPJ in the agentic experiences suggests that this effect may not necessarily be lateralised to the left hemisphere. A recent study by Zito, Andereg, et al. (2020) found that transcranial magnetic stimulation (TMS) delivered over the right TPJ

produced similar effects, with participants experiencing a diminished SoA as a function of neural excitation. These findings, together with those of the current study, underscore the TPJ as a region responsible for detecting errors in action authorship. As such, the TPJ can be understood as critical to one's ability to distinguish self-produced actions from those of others. Broader socio-cognitive literature supports this notion, placing the TPJ at the core of self-other representations, and may be key for maintaining a sense of self during social interactions or tasks involving joint actions (Eddy, 2016; Quesque & Brass, 2019; Van Veluw & Chance, 2014).

It is the neural substrates of SoA which are important to informing our understanding of clinical conditions. As disturbances in SoA are characteristic of several neurological and psychiatric conditions (e.g., Gentsch et al., 2012; Saito et al., 2017; Scott et al., 2022; Seghezzi et al., 2021; Zapparoli et al., 2020), a better understanding of the neural architecture underpinning this experience may give way to the development of novel treatments. For example, treatments targeting the TPJ may be better suited to psychiatric conditions characterised by impairments in registering violations to the SoA for actions affected by, or originating from, external sources.

The current study presents novel causal evidence for dissociable effects of stimulation to the l-dlPFC and l-TPJ on intentional binding. The results should encourage further research focusing on more intricate manipulations known to affect intentional binding. For example, the SoA task used in the present study featured only non-contingent action-outcomes, such that both rewarding and punishing outcome tones were allocated randomly across trials irrespective of the key press that participants chose to perform. It could therefore be argued that action selection processes in the current study did not accurately reflect that of typical goal-directed behaviour. Indeed, traditional conceptualisations of volitional action outline the anticipation of a specific goal or outcome that precedes the intention to act as a core feature informing the SoA (Haggard, 2008). As this ability to prospectively predict the outcomes of our actions has been shown to have a significant effect on implicit SoA (Christensen et al., 2016; Majchrowicz & Wierchoń, 2018; Schwarz et al., 2022; Tanaka and Kawabata, 2021; Yoshie & Haggard, 2017), future research should aim to embed probabilistic learning within its design. Such paradigms would enable participants to effectively learn the statistical environment relevant to their decision-making process and thus, better harness prospective components of the SoA.

Although intentional binding paradigms have been proposed as the most robust method of measuring the implicit agentic experience (Siebertz & Jansen, 2022; Tanaka et al., 2019), there remains outstanding questions surrounding their internal validity (e.g., Kong et al., 2024; Suzuki et al., 2019). Namely, recent research by Kong et al. (2024) found that both active and passive actions produced comparable intentional binding effects, suggesting that inferences of causality rather than action intentionality may better define observed temporal shifts (however see Ciaunica et al., 2024, Borhani et al., 2017 and Caspar et al., 2016 for contrasting evidence). We favour a unified approach to temporal binding, in accordance with that outlined by Hoerl et al. (2020). Under

this framework, temporal binding is reconceptualised to encompass causality and intentionality as two cues to the same agentic experience. More specifically, while the SoA may arise within the expected context of voluntary action (“I caused x”), it may also do so in the case of distal events (“I caused y by causing x”). This latter experience can be defined as one informed by the presence of internal proprioceptive signals in conjunction with causal beliefs about one’s proximal movements producing a distal event. While this model does not stand to entirely resolve the existing issues around intentional binding, our findings positioned within this framework can be better understood to encompass an experience of action authorship that is informed from a number of different cognitive sources.

Further to the broader concerns of intentional binding as an adequate proxy for SoA, the interpretation of the current study’s findings must also consider finer limitations grounded in the design of Libet clock paradigms themselves. Despite their wide use in SoA research and evidence to suggest that they are a more reliable alternative to interval estimation methods (Siebertz & Jansen, 2022), there remains a somewhat heterogeneous evidence base surrounding their use (Braun, Wessler, & Friese, 2021). Research examining the source of this variability suggests that intentional binding is particularly sensitive to alterations in Libet clock parameters. Of particular interest, Ivanof and colleagues (2022a; 2022b) found that across nine experiments the magnitude of outcome binding effects increased proportionately with the speed of each Libet clock revolution (Ivanof et al., 2022a). As the clock speed selected in the current study (2000 msec) fell between the medium (2560 msec) and fast (1280 msec) conditions outlined by Ivanof (2022a), there is a possibility that the reported outcome binding effects were marginally exaggerated by task artefacts. It should be noted however that while authors observed a trend towards increased outcome binding at faster clock speeds, the difference between medium and fast speed conditions specifically was not significant. It is therefore unlikely that the ~500 msec discrepancy between Ivanof and colleagues’ (2022a) procedure and our own significantly skewed the reported findings. Nonetheless, these issues constitute an important concern for future research to address. In order for meaningful comparisons to be drawn across the SoA literature, we strongly encourage future research to consider the development of a standardised protocol for Libet clock task parameters. Moreover, the complexity of the statistical model used in the current study may have reduced our ability to detect smaller effects. In future research, it would be beneficial to develop more focused hypotheses based on the current findings and test them in larger samples or with more targeted statistical designs. This approach could help improve the power to detect subtle effects. Finally, as SoA differences are associated with several mental health conditions (Moore, 2016), assessing stimulation effects in different clinical populations may provide valuable insights into novel treatment options.

5. Conclusions

The present study provides causal evidence for the respective roles of the l-dlPFC and l-TPJ in processing prospective and retrospective components of agency, using both action and outcome binding. We find opposing effects of excitatory stimulation to the l-TPJ and the l-dlPFC on intentional binding, irrespective of prospective or retrospective manipulations. We also find novel evidence that excitation of either the l-TPJ or l-dlPFC increases valence effects on outcome binding, potentially reflecting a strengthening of self-favouring predictions of action-dependent outcomes. The results improve our understanding of the causal neural processes associated with a sense of agency and provide targets for future clinical interventions across a range of brain-based conditions.

CRedit authorship contribution statement

Amber Pryke: Writing – review & editing, Writing – original draft, Project administration, Investigation, Formal analysis, Data curation. **Krishnapriya Jayachandran:** Writing – review & editing, Data curation. **Andrew K. Martin:** Writing – review & editing, Writing – original draft, Supervision, Project administration, Methodology, Formal analysis, Conceptualization.

Funding

The project was partially funded by an EU COFUND Junior International Fellowship awarded to A.K.M. A.P conducted this project under funding from the South east Network for Social Sciences (SeNSS) Doctoral Training Partnership accredited by the ESRC.

Declaration of competing interest

The authors report no conflict of interests.

Acknowledgements

The authors would like to thank all participants for their participation. We thank Frank Gasking for technical support.

Scientific transparency statement

DATA: All raw and processed data supporting this research are publicly available: <https://osf.io/3jsfu/files/osfstorage>.

CODE: This research did not make use of any analysis code.

MATERIALS: All study materials supporting this research are publicly available: <https://osf.io/3jsfu/files/osfstorage>.

DESIGN: This article reports, for all studies, how the author(s) determined all sample sizes, all data exclusions, all data inclusion and exclusion criteria, and whether inclusion and exclusion criteria were established prior to data analysis.

PRE-REGISTRATION: At least part of the study procedures was pre-registered in a time-stamped, institutional registry prior to the research being conducted: <https://osf.io/uk9sf>. At least part of the analysis plans was pre-registered in a time-stamped, institutional registry prior to the research being conducted: <https://osf.io/uk9sf>.

For full details, see the Scientific Transparency Report in the supplementary data to the online version of this article.

Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.cortex.2025.04.014>.

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