

An amygdala-mediated simulation-to-decision cascade aligns interbrain dynamics to solve social dilemmas

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Mutual cooperation (MC) requires independent decision-makers to coordinate under uncertainty about each other's intentions. Using simultaneous intracranial electroencephalography from epilepsy–patient dyads playing a real-time iterated prisoner's dilemma, we identified neurocognitive mechanisms that enable interacting brains to align during mutual cooperation. Amygdala interbrain representational alignment (IBRA), similarity in fine-grained neural spectral patterns across partners, emerged selectively during MC and predicted trial-by-trial outcomes. Early θ -suppression-characterized IBRA reflected rapid, history-sensitive alignment, whereas later IBRA, characterized by low-frequency suppression and γ increases, supported coordinated commitment and action. Furthermore, mutual cooperation relied on an amygdala-mediated, simulation-to-decision cascade, operationalized here as a model-based prospective estimate of partner cooperation that guided self-choice. Amygdala low-frequency suppression tracked this prospective estimate of partner cooperation and θ – γ coupling linked this partner estimate to self-decision processes. Conditional cooperators strategically relied on partner tracking, whereas unconditional cooperators leveraged dispositional tolerance of uncertainty to transition into anticipatory neural alignment. These findings reveal the neural interdependency underpinning MC, redefining cooperation not as isolated decision-making but as a dynamic, interbrain, cognitive alignment.

Mutual cooperation represents one of humanity's most remarkable strengths, yet also one of its most fragile achievements. From interpersonal alliances to large-scale endeavors, cooperation enables collective achievements that far exceed individual capabilities^{1,2}. Defined as joint, interdependent commitments to align actions toward shared benefits^{3,4}, mutual cooperation (MC) emerges when individual decisions dynamically converge into collective action. However, despite its fundamental importance, MC often fails to emerge even among well-intentioned

individuals^{5,6}. This paradox manifests prominently in social dilemmas such as the prisoner's dilemma (PD), wherein mutual cooperation yields the optimal collective outcomes, yet rational agents typically default to defection—the individually dominant strategy⁷. A central question therefore remains unresolved: how do two autonomous minds, each with private intentions and incentives to act selfishly, align their decisions and take a joint leap toward cooperation? Addressing this question is central across disciplines, including economics, psychology and neuroscience^{8,9}.

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Classic game-theory models identify two fundamental barriers to MC: the defection equilibrium^{5,6} and the commitment problem¹⁰. The defection equilibrium arises from independent, self-interested reasoning: individuals conclude that defection maximizes personal payoff regardless of the partner's choice^{5,6}. Mutual defection thus becomes the stable Nash equilibrium, despite being collectively suboptimal. This reflects decisional independence, wherein individuals treat their partner's intentions as irrelevant or opaque^{11,12}. Second, even when both parties prefer cooperation and consider each other's intentions, they still face the commitment problem: each fears unilateral exploitation without guarantees that the partner will also cooperate^{6,13}. This barrier stems not from selfishness as such, but from informational uncertainty in decentralized decision-making: the fundamental opacity of the partner's mind. Together, these challenges highlight that MC is not merely an aggregate of individual cooperative acts, but a relational achievement requiring intentional alignment and mutual commitment under uncertainty^{10,14}.

Previous neuroscience studies have implicated the temporoparietal junction (TPJ), prefrontal cortex and amygdala in cooperative decision-making, highlighting mechanisms of social valuation that integrate external incentives with intrinsic motivations for cooperation^{9,15–17}. However, existing studies have predominantly treated cooperation as discrete, isolated choices made by individuals in parallel, necessitating a conceptual shift: from viewing cooperation as independent choice events to understanding it as a dynamically constructed, interbrain process^{18,19}. This perspective relates to, but is distinct from, classic theory-of-mind accounts of social cognition^{20,21}. Theory of mind primarily addresses an epistemic challenge: how individuals infer another agent's beliefs, intentions or mental states²¹. Although such inference is fundamental for social interaction, accurate mental-state inference alone does not guarantee coordinated outcomes in strategic environments characterized by interdependence and incentive conflict. For example, in the PD, individuals may correctly infer a partner's cooperative intention yet still defect to maximize personal payoff²². Thus, beyond understanding another mind, successful cooperation requires mechanisms that transform partner-related information into coordinated action.

To address this coordination-level challenge, we proposed that MC becomes feasible through what we term 'interdependent cognition': a process in which each individual's decision-making process is shaped by internal representations and predictions of the partner's likely intentions and actions^{23–25}. Within this framework, cooperation does not arise from isolated decisions, but from dynamically interdependent cognitive processes across interacting individuals. We formalized this idea as a neural interdependency framework, positing that cooperative convergence emerges when individuals, despite making private decisions, become functionally coupled through aligned neural representations and prospective partner-estimation processes.

This framework specifies two neural mechanisms that operationalize such interdependency: interbrain representational alignment (IBRA) and social prospective simulation. IBRA addresses decisional independence by aligning task-relevant neural representations across interacting partners, whereas social prospective simulation may address informational uncertainty by enabling individuals to generate a prospective estimate of their partner's forthcoming decisions and integrate that estimate into their own decision process.

Specifically, IBRA enables interacting individuals to develop similar internal representations of ongoing interactions, such as prior cooperative exchanges or partner-related expectations, by converging on similar, task-relevant neural patterns. This mechanism builds on foundational work on neural reinstatement, in which neural patterns from encoding episodes are reactivated during retrieval^{26,27}, and on the representational similarity framework²⁸, which quantifies neural pattern similarity across cognitive states, attitudes and abstract mental constructs^{29,30}. Recent intracranial electroencephalography

(iEEG) studies have shown that spectral power patterns carry distinct cognitive content at millisecond resolution^{27,31}, tracking episodic reinstatement, shared abstract structures and internal task models^{32–34}. Extending these insights, we operationalize IBRA as within-dyad similarity in time-resolved spectral patterns between interacting brains. Unlike traditional metrics of interbrain synchrony, which quantify co-fluctuations in amplitude or phase without reference to represented content^{3,35,36}, IBRA captures crossbrain similarity in representational structure, allowing us to test whether convergent neural representations specifically emerge during mutual cooperation.

Second, social prospective simulation addresses the challenge of informational uncertainty by enabling individuals to form a prospective estimate of their partner's upcoming decisions and integrate that estimate into their own decision-making processes^{13,23–25,37,38}. This mechanism is motivated by three lines of evidence: (1) human neuroimaging studies showing prospective inference about others' mental states^{8,39}; (2) primate electrophysiological research demonstrating that amygdala neurons encode predictions about partner choices³⁷; and (3) behavioral findings indicating shifts in cooperative strategies when partners' decision-making becomes transparent^{13,38}. Translating these insights into real-time human interactions, we hypothesized that, during MC, the amygdala supports a model-based prospective estimation of partner intent, with this prospective estimate linked to self-decisions, possibly via cross-frequency neural coupling. This mechanism enables individuals to align not only based on history, but also through forward simulation that mentally bridges informational gaps.

Here we leverage simultaneous intracranial recordings (Fig. 1a) from interacting patients with epilepsy to enable millisecond-resolution tracking of neural representational alignment and prospective simulation across brains. We focused on the amygdala (Fig. 1b), crucial for social valuation and partner inference^{16,17,37}, and the TPJ (Fig. 1b), which is central to mentalizing and social prediction^{8,39}. We distinguished two cooperation phenotypes: pure (unconditional) cooperators, who consistently achieve MC from the outset, and conditional cooperators, whose cooperation varies depending on historical interactions and contextual factors. This distinction enabled us to probe whether the proposed mechanisms are essential prerequisites for MC or compensatory processes that emerge under uncertainty. We found that MC was selectively accompanied by amygdala IBRA, with early and late alignment reflecting distinct history-sensitive and decision-related neural dynamics. Computational modeling further revealed an amygdala-mediated prospective partner estimation-to-decision process, in which partner-related estimates are linked to self-decisions through low-frequency and cross-frequency neural dynamics. Finally, pure mutual cooperators exhibited anticipatory interbrain alignment, suggesting that MC can arise through distinct neural routes depending on how individuals resolve social uncertainty.

Results

Behavioral signatures of MC in the iterated PD

To investigate the neurocognitive dynamics underlying MC, defined here as both individuals choosing to cooperate within the same trial, we simultaneously recorded iEEG signals from pairs of patients with epilepsy ($n = 45$ dyads; Supplementary Table 1 and Supplementary Fig. 1) engaged in a real-time, feedback-based, iterated PD game (Fig. 1c), a classic paradigm for studying cooperation decision-making. In this task, each individual faced a binary choice to cooperate (C) or defect (D), with the payoff matrix designed to create a tension between individual self-interest and collective benefit. This matrix ensures that MC yields optimal collective gains, whereas unilateral defection maximizes the defector's payoff at the expense of the cooperator (Fig. 1d and Methods). Our primary analyses focused on 26 conditional-cooperator (CC) dyads (Supplementary Table 1a) who exhibited variability across trials, achieving both MC and non-MC outcomes (unilateral cooperation or mutual defection), enabling within-dyad comparisons. The remaining

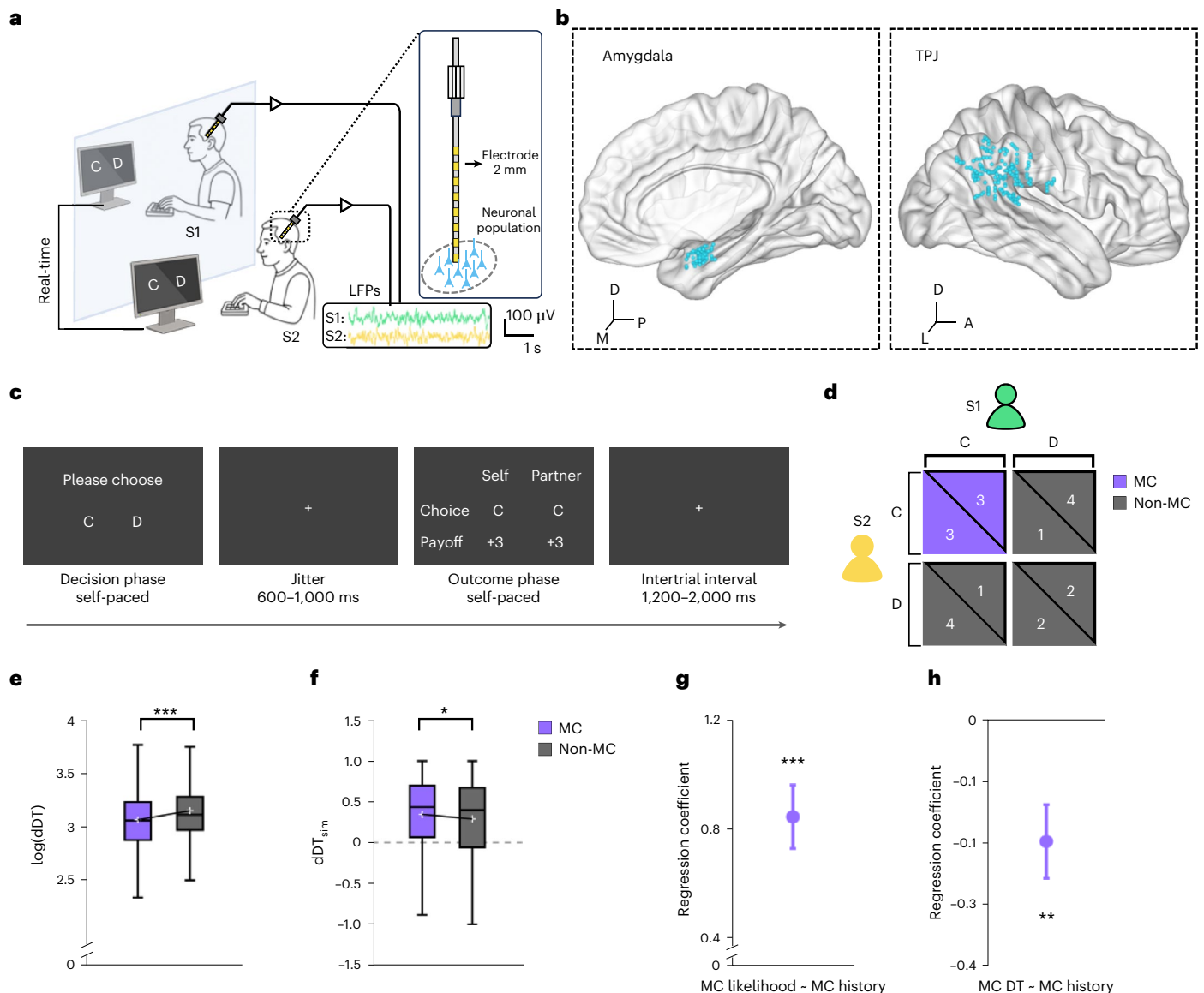


Fig. 1 | Experimental setup and behavioral results. a, Schematic of simultaneous intracranial recordings from dyads of patients with epilepsy engaged in a real-time PD game. Implanted electrodes recorded local field potentials (LFPs) from neuronal populations. **b**, Locations of 208 amygdala (left) and 410 TPJ (right) channels. The anatomical localization of each channel was determined and visually inspected within the native space of each patient. For visualization, electrode locations were registered to standard Montreal Neurological Institute (MNI) space and projected onto the surface of the right hemisphere of the ICBM152 template (channels located in the left hemisphere are mirrored at their symmetrical locations in the right hemisphere). Each colored sphere represents a single channel. D, dorsal; P, posterior; M, medial; A, anterior; L, lateral. **c**, Trial timeline: example of an MC trial. Each trial included a decision phase (cooperate versus defect), followed by the outcome phase (both decisions and the resulting payoffs) and an optional punishment phase when defection occurred. Phases were separated by a 600-ms to 1,000-ms jitter and trials were separated by an intertrial interval of 1,200–2,000 ms. **d**, Payoff matrix specifying monetary

rewards for all choice combinations. **e,f**, MC trials showing faster decision times (**e**) and greater dyadic decision-time similarity (**f**) than non-MC trials. The box plots show the medians (center line), interquartile ranges (IQRs, box edges), whiskers (1.5 $\times$ IQR) and mean values (crosses). Black lines connect mean values across all trials in each condition. Significance was assessed through two-sided linear mixed-effects models (see Supplementary Tables 2 for full statistics). **g,h**, Regression coefficients for MC rate in the previous three trials predicting MC likelihood (**g**) and MC decision time (**h**), derived from (generalized) linear mixed-effects models, two sided. MC history rate positively predicted MC likelihood (**g**) and negatively predicted MC decision time (**h**). Model formulas are specified below the x axes, with tildes linking dependent (left) and independent variables (right). Center dots and corresponding error bars represent estimated coefficients and s.e. values, respectively. See Supplementary Tables 3 for full statistics: *** $P < 0.001$, ** $P < 0.01$, * $P < 0.05$. C, cooperate; D, defect. Illustration in **a** created in BioRender; Ni, J. <https://www.biorender.com/adev72z> (2026).

19 dyads (Supplementary Table 1b), who consistently cooperated throughout, were analyzed separately.

Participants achieved MC in 53% of trials, a rate consistent with prior reports in healthy individuals¹⁵. Compared to non-MC trials, mutually cooperative decisions occurred significantly faster ($P = 3.33 \times 10^{-4}$; Fig. 1e and see full statistical reports in Supplementary Table 2) and exhibited greater temporal alignment between partners ($P = 0.021$;

Fig. 1f and Supplementary Table 2) even after controlling for individual decision speed or choice similarity. These findings suggest that MC involves not only joint choices but also temporal alignment within the dyad.

We next examined how interaction history influenced current cooperation decisions. Both the likelihood ($P = 4.27 \times 10^{-13}$; Fig. 1g and Supplementary Table 2) and speed ($P = 0.001$; Fig. 1h and

see Supplementary Table 2 for full statistics) of achieving MC were predicted by recent MC history defined as the MC rate over the preceding three trials (see history-length analyses in Supplementary Table 3a). We next performed forward stepwise model selection to assess the relative predictive contributions of recent MC rate, individual-level cooperation rate and punishment rates. The MC rate entered the models first and was the only predictor retained for both MC likelihood ($\chi^2 = 253.16$, $P = 5.33 \times 10^{-57}$) and MC speed ($F_{395} = 16.37$, $P = 6.28 \times 10^{-5}$). After the MC rate had entered the models, neither individual cooperation rate nor punishment rate met the criterion for further inclusion ($P > 0.05$; Supplementary Table 3b). In addition, this interaction-history effect cannot be reduced to simple reciprocal strategies (such as Tit-for-Tat or Win-Stay-Lose-Shift strategies; Methods and Supplementary Table 4). These findings suggest that MC in this task is driven not solely by momentary individual cooperative preferences or fixed rule-based reciprocity, but also by history-dependent interaction dynamics. This raises the possibility that MC may rely on shared interaction-dependent processes between partners, motivating our investigation of IBRA in the following neural analyses.

Amygdala-mediated IBRA supports mutual cooperation

We tested whether MC involves IBRA. Leveraging the broadband spectral coverage and high spatiotemporal resolution of iEEG, we extracted trial-wise spectral power from each intracranial electrode (4–200 Hz, 200-ms sliding windows, 10-ms steps; analyses with a shorter 100-ms sliding window replicated the main findings; Supplementary Fig. 2). IBRA was quantified by calculating Fisher's z -transformed Spearman's correlations between time-resolved spectral power vectors within the decision-making epoch (0–1,500 ms) for each pair of same-region channels between interacting brains. This yielded a time-resolved IBRA profile per dyad and trial, enabling us to track representational alignment over time (Fig. 2a).

For each region (amygdala: 141 channel-pairs; TPJ: 332 channel-pairs; Supplementary Table 1a), we compared IBRA time courses between MC and non-MC conditions across all channel-pairs using two-sided, paired Student's t -tests ($P < 0.05$, cluster corrected via permutation tests). In the amygdala, IBRA was significantly higher during MC than non-MC trials in two time windows (early: 70–380 ms, $P_{\text{corr}} = 0.002$; later: 480–1,160 ms, $P_{\text{corr}} = 1.00 \times 10^{-4}$, 10,000 permutations; Fig. 2b). The increased IBRA within each time window was specific to MC (versus 0: early: $P_{\text{corr}} = 0.001$, late: $P_{\text{corr}} = 1.00 \times 10^{-4}$, one-sided, one-sample Student's t -test), with no significant IBRA increases observed in non-MC trials (early: $P_{\text{corr}} = 0.227$; late: $P_{\text{corr}} = 1.000$; full statistical reports in Supplementary Table 5a). In contrast, TPJ showed early IBRA increases (90–410 ms) in both MC ($P_{\text{corr}} = 0.003$; Fig. 2c and Extended Data Fig. 1a–c) and non-MC ($P_{\text{corr}} = 0.003$; Fig. 2c and Extended Data Fig. 1d–f) trials, without condition differences, suggesting that TPJ IBRA may reflect a more general process engaged during real-time interaction, rather than being specific to MC. These results highlighted the role of amygdala IBRA in MC, leading us to

focus further analyses on the amygdala. To ensure that the regional dissociation between amygdala and TPJ was not driven by differences in the number of channel-pairs, we performed a subsampling control in which 141 TPJ channel-pairs were randomly selected to match the amygdala sample size. Repeating the MC versus non-MC comparison across 5,000 subsamples yielded no significant MC-specific IBRA in TPJ (significant clusters observed in <5% of subsamples), confirming that the absence of MC modulation in TPJ cannot be attributed to unequal statistical power.

As a confirmatory analysis accounting for the hierarchical structure of the iEEG data, we further applied time-resolved, linear mixed-effects models (LMEs) with dyad and channel-pair as random effects (Methods). These analyses replicated the two temporally distinct windows with MC > non-MC effects in the amygdala IBRA and the absence of condition differences in TPJ IBRA (Extended Data Fig. 2). Furthermore, the MC-increased amygdala IBRA was consistently observed in 75% of dyads and remained at the dyad level (Supplementary Table 5a).

Specificity of amygdala IBRA for MC

To rule out alternative explanations for the observed increases in amygdala IBRA, we performed permutation-based control analyses targeting shared task settings, concurrent decision-making behaviors and same choices or outcomes (Fig. 2d and Supplementary Note 1). First, we performed a full-trial permutation by randomly pairing choices across trials, thereby disrupting trial-level simultaneity while maintaining the overall task settings. The observed amygdala IBRA during MC exceeded the 95% confidence interval (CI) of the null distribution in both early ($P_{\text{corr}} = 1.00 \times 10^{-4}$; Fig. 2e) and late ($P_{\text{corr}} = 1.00 \times 10^{-4}$; Fig. 2f) windows, ruling out shared task settings as a sufficient explanation. Second, we randomly paired cooperation choices while ensuring that both participants chose to cooperate, but disrupting whether these choices occurred on the same trial. Again, amygdala IBRA during real MC exceeded this control (early: $P_{\text{corr}} = 2.00 \times 10^{-4}$; late: $P_{\text{corr}} = 4.00 \times 10^{-4}$; Fig. 2g,h), ruling out simple co-cooperation as a source. Third, we restricted the random pairing to trials in which dyads achieved MC, preserving the MC outcome but disrupting real-time dyadic simultaneity and shared trial-specific interaction history. Here a temporal dissociation emerged: early IBRA remained significantly increased ($P_{\text{corr}} = 1.00 \times 10^{-4}$; Fig. 2i), whereas late IBRA did not ($P_{\text{corr}} = 0.684$; Fig. 2j). To assess whether this dissociation could be attributed to the different lengths of the early and late windows, we randomly extracted shorter segments of the same duration as the early window from the late IBRA window in each permutation. The observed late IBRA still did not exceed the outcome-paired null distribution ($P_{\text{corr}} = 0.805$), indicating that the early–late dissociation was not attributable to window length alone. These results suggest that early amygdala IBRA requires real-time cooperative alignment, whereas late IBRA may occur whenever MC is achieved, even across different trials.

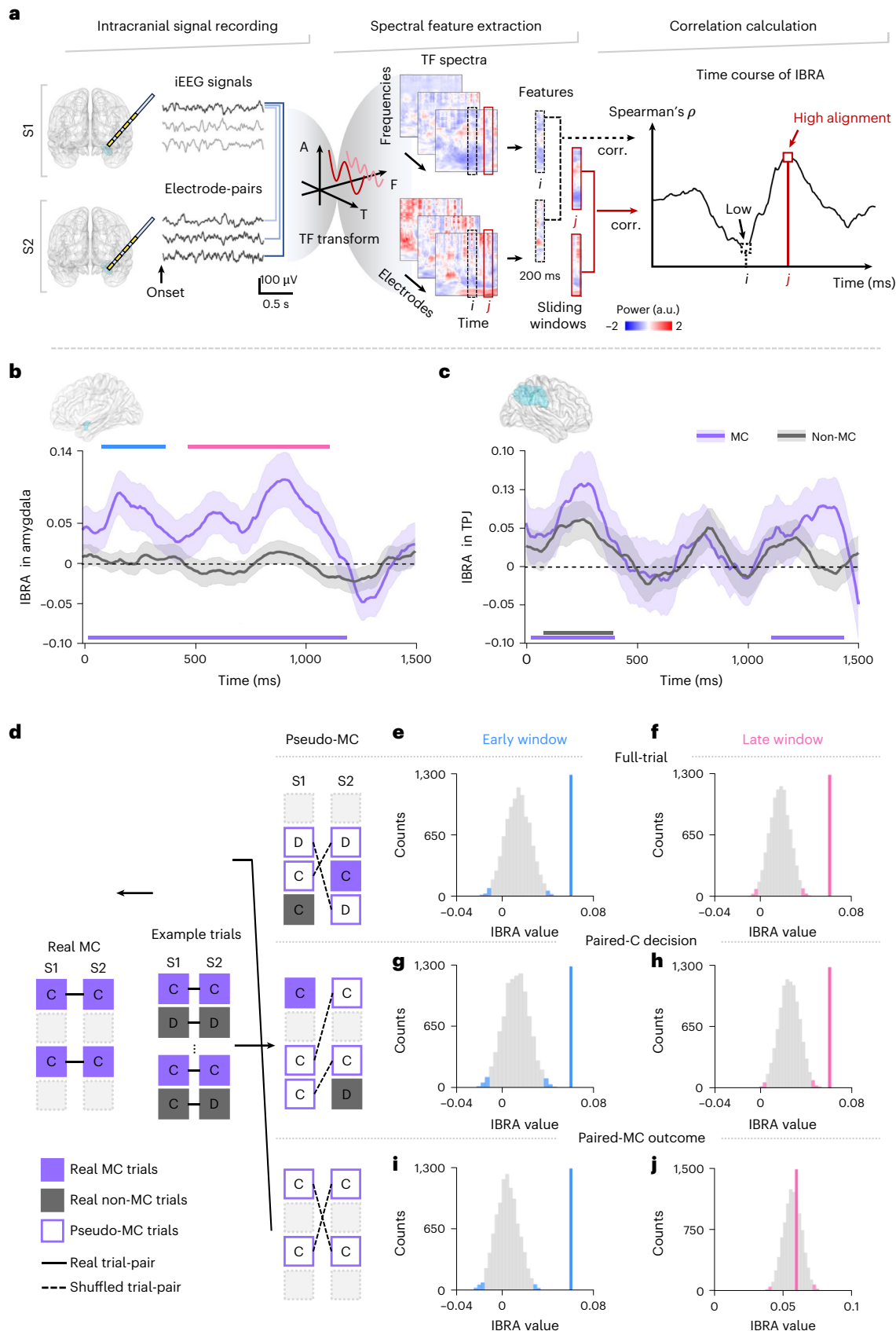
These control analyses confirmed that MC-increased amygdala IBRA cannot be explained by task structure, same choices or the mere

Fig. 2 | Amygdala-mediated IBRA underlies mutual cooperation. **a**, Schematic of IBRA calculation. For each dyad, simultaneous iEEG signals from same-region electrodes were transformed into time–frequency spectra (4–200 Hz). Spectral power across all frequencies was extracted in sliding windows (200 ms, 10-ms steps) and represented as feature vectors. Trial-wise Fisher's z -transformed Spearman's correlations between feature vectors from paired electrodes yielded a time-resolved IBRA profile. Example windows with high (red) and low (black) alignment are illustrated. **b,c**, Time courses of IBRA in amygdala (**b**) and TPJ (**c**) during MC and non-MC (colored curves representing mean values across channel-pairs with shaded areas indicating the 95% CI). Horizontal lines indicate significant time windows (cluster-based permutation corrected, $n = 10,000$); blue or pink: MC versus non-MC (two-sided, paired Student's t -tests); purple or gray: MC or non-MC > 0 (one-sided, one-sample Student's t -tests). Amygdala exhibited two distinct MC-specific IBRA increases (70–380 ms and

480–1,160 ms), whereas TPJ showed early IBRA increases (90–410 ms) in both MC and non-MC trials. **d–j**, Validation of MC-specific amygdala IBRA. **d**, Illustration of three trial-shuffling schemes used to generate pseudo-MC-trials: full-trial permutation (top), paired-cooperation-choice permutation (middle) and paired-MC-outcome permutation (bottom). **e,f**, In full-trial permutations, real MC IBRA exceeded the 95% CI of the null distribution in both early (**e**) and late (**f**) windows. **g,h**, In paired-cooperation-choice permutations, real MC IBRA again exceeded the 95% CI in both windows (early (**g**) and late (**h**)). **i,j**, In paired-MC-outcome permutations, real MC IBRA exceeded the 95% CI in the early (**i**) but not the late (**j**) windows, indicating a temporal dissociation. Null distributions are plotted with values exceeding 95% CI colored, whereas vertical lines indicate the observed IBRA values. See Supplementary Table 5 for full statistics. TF, time frequency; corr., correlation.

co-occurrence of MC outcomes. Furthermore, MC recruits two temporally dissociable phases of amygdala IBRA, with early IBRA sensitive to real-time cooperative alignment, whereas late IBRA reflects a more generalized alignment of the cooperation state, independent of real-time simultaneity. To further rule out timing-related confounds,

we conducted additional analyses including trial-wise decision speed and dyadic decision–time similarity as covariates in LMEs, and the MC-specific IBRA effects in both early and late windows remained significant (Extended Data Fig. 3a and Supplementary Table 6). Similar results were obtained when recomputing IBRA on behavior-matched subsets



of MC and non-MC trials (Extended Data Fig. 3b and Supplementary Table 6). Moreover, aligning neural activity to each participant's response (that is, response-locked IBRA analysis) did not reveal significant MC-specific IBRA effects (Extended Data Fig. 3c), indicating that the observed stimulus-locked alignment cannot be explained by shared motor preparation or synchronized response timing.

Finally, to determine whether the two-stage structure of IBRA was tied to absolute physical timing or instead tracked the cognitive stages of the decision-making process, we performed a decision-time (DT)-normalization (time-warping) analysis. By rescaling each trial's neural time series into a fixed number of bins from stimulus onset to response execution, we aligned neural dynamics by relative decision progress. This analysis replicated the two-phase temporal structure, revealing robust MC-specific alignment in both the early and the late stages of the normalized decision interval (Extended Data Fig. 3d and Supplementary Table 6). Thus, early and late IBRA may reflect distinct stages of the cooperative decision process rather than fixed absolute time intervals.

Interaction-history similarity organized amygdala IBRA

The permutation-based controls above constrained several nonrepresentational explanations for amygdala IBRA. However, these analyses did not directly test whether IBRA was organized by task-relevant content. To address this, we performed a crossbrain, crosstrial representational similarity analysis (RSA). For each dyad, we constructed a 30×30 crossbrain neural similarity matrix, in which each cell (i, j) represented the similarity between the spectral pattern of trial i from one participant and trial j from the partner (Extended Data Fig. 4a). We then tested whether interaction-history similarity organized this crossbrain neural similarity. Interaction-history similarity significantly accounted for crossbrain neural similarity specifically within MC–MC trial pairs, even after controlling for the same-trial diagonal component as well as the overall history-similarity and condition-similarity structure (early: $t = 2.391$, $P = 0.009$; late: $t = 2.626$, $P = 0.005$; Extended Data Fig. 4b,c). Time-resolved analyses yielded similar effects (Extended Data Fig. 4d), whereas no comparable positive history-structured effect was observed for pairs of non-MC trials (Extended Data Fig. 4e,f). Thus, although similar interaction histories can occur in both cooperative and noncooperative contexts, only MC was associated with reliable history-structured alignment across brains.

These results support the interpretation of amygdala IBRA as content-structured representational alignment. Specifically, they show

that crossbrain neural similarity was organized by behaviorally meaningful interaction-history across trials, rather than merely by same-trial timing, shared sensory events or generic dyadic co-fluctuation. Importantly, these findings complement rather than contradict the earlier observation of the real-time specificity of early IBRA: real-time interaction appears to amplify an underlying history-structured alignment across brains during MC.

Trial-level prediction of MC by amygdala IBRA

Although we observed stronger amygdala IBRA during MC at the condition level, this does not necessarily imply that it can distinguish MC from non-MC on individual trials. We therefore tested whether amygdala IBRA dynamics during decision-making carried information about upcoming cooperative outcomes. We performed time-resolved decoding in which amygdala IBRA strength was used to classify each trial as MC or non-MC. To ensure robust out-of-sample generalization and minimize overfitting, we implemented leave-one-dyad-out cross-validation using a linear discriminant classifier (Methods). Cooperation outcomes could be decoded significantly above chance within two sustained temporal windows (90–350 ms, $P_{\text{corr}} = 2.00 \times 10^{-4}$; 490–1,130 ms, $P_{\text{corr}} = 2.00 \times 10^{-4}$; Extended Data Fig. 5), closely overlapping with the early and late IBRA periods (Fig. 2b). These findings indicate that amygdala IBRA dynamics contain trial-level predictive information about forthcoming MC outcomes.

Dissociation of early and late amygdala IBRA

Having identified two temporally distinct amygdala IBRA phases during MC, we next sought to determine whether they reflected distinct functional and neural processes. We addressed this question at three levels: behavioral relevance, spatiotemporal organization and representational content.

Functional dissociation. We began by examining the relationship between IBRA and past interaction history, which was identified as a behavioral predictor of MC. Past cooperation history influenced both early and late IBRA, but through different response features. Specifically, a higher proportion of recent MC history predicted both an earlier onset (but not intensity) of early IBRA ($\beta = -0.109$, $P = 0.009$; Fig. 3a and Supplementary Table 5b) and a reduced intensity of late IBRA ($\beta = -0.110$, $P = 0.004$; Fig. 3a and Supplementary Table 5b). Further mediation analysis revealed that the effect of history on late IBRA intensity was partially mediated by early IBRA onset (indirect

Fig. 3 | Dissociable dynamics and representational content of early and late amygdala IBRA. a–d, Functional dissociation. Early and late amygdala IBRA showed different relationships with past and current cooperation. **a**, Recent cooperation history advancing the onset of early IBRA but reducing the intensity of late IBRA. **b, c**, Both IBRA predicting dyadic decision–time similarity (**b**), yet their relationship to decision speed diverging: stronger early IBRA predicting faster MC, but stronger late IBRA predicting slower cooperation (**c**). In **a–c**, regression coefficients were derived from LMEs, two sided. Center dots and corresponding error bars indicate the estimated coefficients and s.e. values, respectively. Multiple comparisons were all corrected by FDR. **d**, Mediation analyses revealed that early IBRA timing directly linked past history to faster current MC (pathway colored in blue) and also acted serially with late IBRA to shape decision speed (pathway colored in pink). See Supplementary Table 5 for the full statistics. **e–h, Spectral dissociation.** Time–frequency spectrograms (t -maps, **e** and **g**) and power amplitude averaged within each frequency band (**f** and **h**) for MC. Early IBRA was characterized by θ suppression (**e** and **f**), whereas late IBRA involved θ and α suppression together with γ increases (**g** and **h**). Significance was assessed through two-sided, one-sample Student's t -tests. Multiple comparisons were corrected by cluster-based permutation tests (**e** and **g**, $n = 10,000$; black contours delimit significant clusters) and FDR (**f** and **h**). Black contours delimit significant clusters after two-sided, cluster-based permutation tests. These frequency-specific signatures were unique to mutual cooperation. **i–l, Spatial dissociation.** **i**, IBRA strength across timepoints for

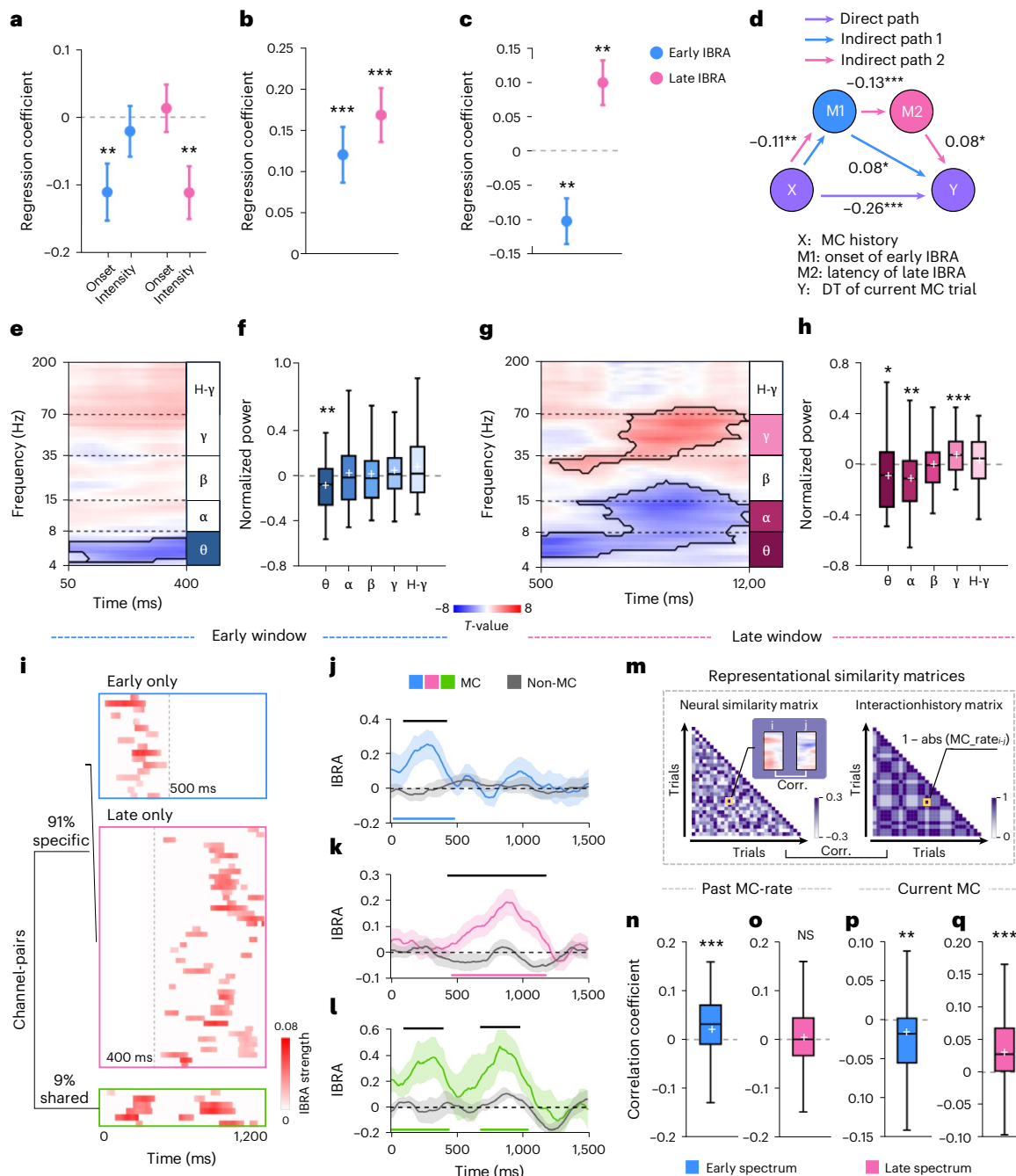
amygdala-responsive channel-pairs, sorted by channel-pair types. Most channel-pairs exhibited either early-specific or late-specific IBRA increases, with only 9% showing overlap. Each row represents an amygdala channel-pair responsive to MC, with the color indicating the IBRA strength differences between MC and non-MC conditions. The white areas indicate timepoints without significant MC-increased IBRA relative to 0 or non-MC. **j–l**, Average IBRA time courses for early-only (**j**), late-only (**k**) and both-responsive (**l**) channel-pairs (colored curves representing mean values across channel-pairs with shaded areas indicating the 95% CI). The horizontal lines indicate time windows with significant effects (cluster-based permutation corrected, $n = 10,000$): black: MC versus non-MC (two-sided, paired Student's t -tests); blue, pink or green: MC > 0 (one-sided, one-sample Student's t -tests). **m–q, Representational dissociation.** **m**, Illustration of RSA. Trial-wise representational similarity matrices were constructed for neural spectrograms, illustrated with the early IBRA time windows, where similarity was defined as the correlation coefficient of trial-pair spectral power, and for past MC rate (MC_rate _{t_i}), where similarity was indexed by $1 - \text{the absolute difference in prior MC rate between two trials}$. Neural spectral (left) and history-based (right) similarity matrices were then correlated. Spectral pattern in the early (**n**), but not late (**o**), windows represented past MC history. The spectral patterns in the early (**p**) and late (**q**) windows encoded discrimination and similarity across MC trials. In **f, h** and **n–q**, box plots show medians (center line), IQR (box edges), whiskers ($1.5 \times \text{IQR}$) and mean values (crosses). See Supplementary Table 7 for the full statistics: *** $P < 0.001$, ** $P < 0.01$, * $P < 0.05$, NS, not significant. abs, absolute.

effect = 0.014, 95% CI: 0.003, 0.029, $P = 0.012$ (two-sided equivalent), bootstrapping, $n = 5,000$; Supplementary Table 5b), indicating that early IBRA partially offsets the direct influence of past interactions on reducing late IBRA strength.

We then examined how IBRA related to behaviors in current MC trials. With regard to behavioral coordination, both early and late IBRA intensity predicted greater dyadic decision–time similarity (early: $\beta = 0.121$, $P = 3.71 \times 10^{-4}$; late: $\beta = 0.169$, $P = 3.01 \times 10^{-7}$; Fig. 3b and Supplementary Table 5b), suggesting a role for both early and late IBRA in behavioral coordination between partners. However, their relationships with decision-making speed differed: stronger early IBRA predicted faster cooperation decisions ($\beta = -0.102$, $P = 0.002$; Fig. 3c and Supplementary Table 5b), whereas stronger late IBRA was linked to slower decisions ($\beta = 0.099$, $P = 0.002$; Fig. 3c and Supplementary Table 5b). These behavioral relationships were not restricted to cluster-averaged IBRA measures. Time-resolved analyses confirmed similar associations across sustained time

windows (Extended Data Fig. 6). These findings suggested that early neural alignment was associated with rapid, coordinated cooperation, whereas late IBRA may be engaged during more deliberate decision-making processes.

Last, we tested whether IBRA mediated the influence of past cooperation history on current decision speed. Two significant mediation pathways emerged: recent MC history influenced cooperation decision–time: (1) through earlier onsets of early IBRA (pathway colored in blue: indirect effect = -0.009 , 95% CI: -0.018 , -0.001 , $P = 0.020$, two-sided, bootstrapping, $n = 5,000$; Fig. 3d and Supplementary Table 5b); and (2) via a serial path involving both (earlier) early and (weaker) late IBRA (pathway colored in pink: indirect effect = 0.001 , 95% CI: 1.11×10^{-4} , 0.003 , $P = 0.014$, two-sided, bootstrapping, $n = 5,000$; Fig. 3d and Supplementary Table 5b). No significant mediation was observed through late IBRA alone ($P > 0.05$; Supplementary Table 5b). Together, these results highlight the importance of early interbrain alignment



in linking history to faster cooperation decisions, while suggesting that late IBRA may be recruited more strongly during slower or more deliberative coordination.

Spectrospatial dissociation. We next examined whether the early and late IBRA differed in their spectral and spatial profiles. We performed time–frequency decomposition for early and late time windows, respectively, revealing distinct frequency-band-specific responses. Early IBRA during MC was characterized by significant θ -band suppression in the amygdala ($P = 0.005$; Fig. 3e,f; survived false discovery rate (FDR) correction across frequency bands; Supplementary Table 7a). Late IBRA was marked by θ and α suppression and γ -band increases (θ : $P = 0.011$; α : $P = 0.002$; γ : $P = 1.47 \times 10^{-4}$; Fig. 3g,h; survived FDR correction; Supplementary Table 7a). Importantly, suppressed low-frequency power was specific to MC and not observed during unilateral cooperation (Supplementary Fig. 3). These findings indicate distinct spectral profiles for early and late amygdala IBRA, supporting the hypothesis that different neural dynamics underlie the two phases of amygdala-mediated interbrain alignment.

We then examined whether the two IBRA were also spatially dissociable within the amygdala. We identified amygdala interbrain channel-pairs showing significant MC-related IBRA increases in the early window, late window or both. Among channel-pairs showing significant IBRA increase during MC relative to both 0 and non-MC ($n = 69$), only 9% exhibited IBRA increases in both windows. The majority (91%) showed temporal selectivity (early only: 25%; late only: 66%; Fig. 3i–l), a significant imbalance relative to a shared response (9% shared; $\chi^2 = 28.386$, $P = 9.94 \times 10^{-8}$). This pattern suggested that early and late IBRA emerged from partially distinct neural processes, rather than simply reflecting a single temporally extended effect. Although late-selective channel-pairs appeared more common than early-only ones, this difference may partly reflect the longer duration of the late window. Spatial clustering analyses showed no anatomically distinct amygdala subregions exclusively supporting early or late IBRA, suggesting distributed rather than topographically segregated representations (Supplementary Table 7b).

Representational dissociation. Having established that early and late IBRA differ in behavioral correlates and neural signatures, we next asked what task-relevant information was reflected in their associated neural spectrograms. To address this, we constructed trial-by-trial pairwise similarity matrices for neural spectrograms (of early and late IBRA), past MC rates (Fig. 3m) and current outcomes. First, correlating the neural spectral and history-based matrices revealed that the similarity in past MC rates was positively associated with similarity in neural spectral patterns of the early IBRA ($t = 5.421$, $P = 1.28 \times 10^{-7}$; Fig. 3n) but not late IBRA ($t = 1.434$, $P = 0.153$; two-sided, one-sample Student's t -tests, Fig. 3o and Supplementary Table 7c). Thus, early, but not late, spectral patterns carried information about interaction history.

We then tested whether neural spectrograms of early and late IBRA represented current MC outcomes by quantifying neural similarity among MC–MC trial pairs. The neural spectra in the early time window showed a significant negative correlation between MC–MC trials ($t = -2.804$, $P = 0.005$; Fig. 3p and Supplementary Table 7c), suggesting that different MC trials were discriminated by early spectral patterns. In contrast, neural spectra in the late time window showed robust positive similarity across MC–MC trials ($t = 6.421$, $P = 5.76 \times 10^{-10}$; Fig. 3q and Supplementary Table 7c), reflecting shared neural representations of MC across trials. Trial-pair shuffle analyses further confirmed that the observed MC–MC discrimination (early) and similarity (late) were specific to MC (early: $P_{\text{corr}} = 0.009$; late: $P_{\text{corr}} = 0.014$, $n = 10,000$ permutations). Together, these analyses specified the task-relevant information carried by the spectral patterns contributing to IBRA: early IBRA-related spectrograms encoded shared interaction history and discriminated different cooperative events, whereas late IBRA-related

spectrograms reflected a shared anticipation of forthcoming decisions and generalized across cooperative trials.

From shared representations to individual decisions: a prospective partner-estimation process

Although IBRA captures dyadic-level interbrain alignment, MC ultimately depends on two autonomous individuals each making private decisions to cooperate. This raises the classic commitment problem: how do individuals commit to MC without knowing their partner's upcoming decision? We therefore shifted from interbrain alignment to the individual-level neurocognitive processes that could support such commitment, asking whether and how participants formed a prospective estimate of their partner's forthcoming choice and used that estimate to guide their own behavior.

To formalize this process, we constructed a set of models that differed in the information used to update expected partner cooperation. Bayesian model selection identified the self-and-other-history model as the winning model (protected exceedance probability > 0.999 ; Methods), indicating that participants' trial-wise estimate of partner cooperation was best captured when both their own and their partner's past choices were taken into account. From this model, we derived three trial-wise variables: (1) a model-derived prospective estimate of partner cooperation, indexing the estimated probability that the partner would cooperate on the current trial; (2) estimate uncertainty, indexing uncertainty in that estimate; and (3) retrospective cooperative prediction error (PE), indexing whether the partner had been more cooperative than estimated on the previous trial.

We first asked whether these model-derived variables related to actual behavior. The model-derived prospective estimate of partner cooperation positively predicted the partner's actual cooperation ($\beta = 0.651$, $P = 1.26 \times 10^{-9}$, survived FDR correction for multiple comparisons), whereas estimate uncertainty negatively predicted it ($\beta = -0.413$, $P = 3.45 \times 10^{-4}$); retrospective cooperative PE was unrelated to the partner's current decision ($P = 0.989$; Fig. 4a and Supplementary Table 8a). Thus, partner cooperation was most likely when the partner was estimated to cooperate and when that estimate was relatively certain. These effects remained after controlling for the participant's own current choice (Supplementary Table 8a), indicating that the model captured a meaningful prospective estimate of partner behavior rather than merely reflecting concurrent choice coupling.

We then examined whether participants used this prospective estimate to promote MC or instead to exploit expected partner cooperation. The model-derived prospective estimate of partner cooperation positively predicted the participant's own cooperation ($\beta = 0.656$, $P = 2.18 \times 10^{-9}$), whereas estimate uncertainty was unrelated to self-decision ($P = 0.323$; Fig. 4b and Supplementary Table 8a). Thus, when participants estimated that their partner was likely to cooperate, they were themselves more likely to cooperate, suggesting that prospective partner estimation supported MC rather than opportunistic defection. In addition, when the partner had been more cooperative than estimated on the preceding trial, participants became more likely to cooperate on the current trial ($\beta = 0.371$, $P = 6.99 \times 10^{-8}$; Fig. 4b and Supplementary Table 8a), indicating that unexpectedly cooperative feedback further reinforced commitment to MC.

We next asked how the amygdala implemented these computations. We focused on spectrotemporal features linked to MC (that is, early θ suppression, late low-frequency suppression and late γ increase in the amygdala; Fig. 3e–h) and asked how these signals related both to observable choices and to the model-derived latent variables. A double dissociation emerged at the level of observable choices: low-frequency suppression predicted whether the partner would cooperate (early θ : $\beta = -0.131$, $P = 0.022$; late low-frequency: $\beta = -0.173$, $P = 0.003$; Fig. 4c; survived FDR correction for multiple comparisons; Supplementary Table 8b), but was unrelated to the participant's own choice (all $P > 0.05$; Fig. 4c and Supplementary Table 8b). In contrast,

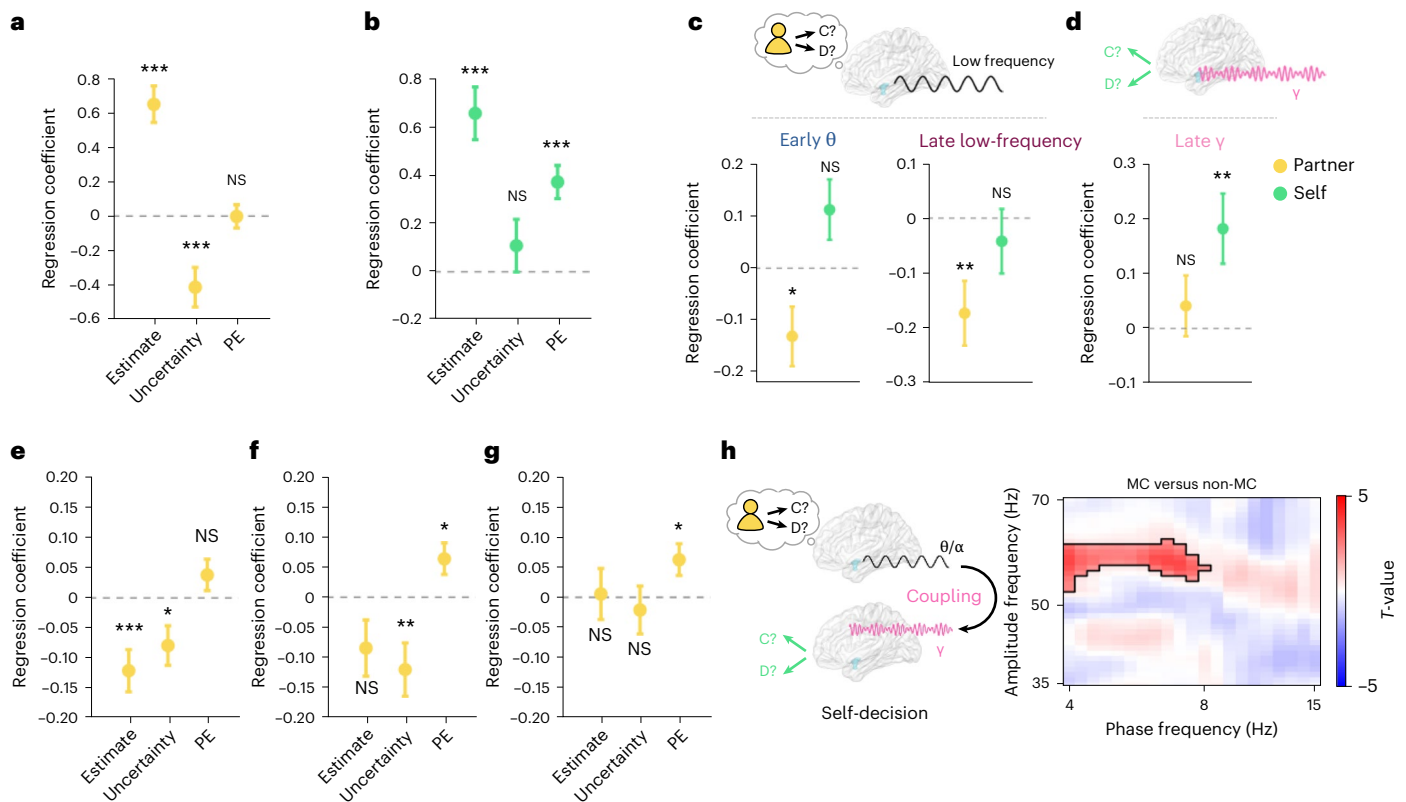


Fig. 4 | An amygdala-mediated simulation-to-decision cascade.

a,b, Model-derived variables showing significant relations to actual behavior. **a**, The partner's actual cooperation associating positively with the prospective estimate of partner cooperation and negatively with estimate uncertainty. **b**, The prospective estimate of partner cooperation and retrospective cooperative PE positively predicting upcoming self-cooperation. **c,d**, Amygdala spectrotemporal features significantly predicting observable partner and self-decisions. **c**, Early θ (left) and late low-frequency (right) power in the amygdala selectively predicting the partner's upcoming cooperation, but not the participant's own choice. **d**, In contrast, late γ activity signaling the participant's own cooperation decision, but bearing no relation to the partner's choice. **e–g**, Amygdala spectrotemporal features significantly encoding model-derived variables. **e**, Early θ power suppression associating with the prospective estimate of partner cooperation

and the uncertainty of that estimate. **f**, Late low-frequency power negatively associating with estimate uncertainty and positively associating with retrospective cooperative PE. **g**, Late γ power correlated with only retrospective cooperative PE. **h**, Partner-prediction-related and self-decision-related signals interacting through PAC. The center dots and corresponding error bars indicate estimated coefficients and s.e. values, respectively. All reported effects survived FDR correction for multiple comparisons; see Supplementary Table 8 for full statistical reports. **h**, Partner-prediction-related and self-decision-related signals interacting through PAC. Amygdala PAC co-modulogram (*t*-value map) for MC versus non-MC, revealing θ phase (4–8 Hz) entraining γ amplitude (53–62 Hz) selectively during MC. The black contour delimits the overlapping significant cluster for both MC versus non-MC (two-sided, cluster-based permutation tests) and MC (non-MC) versus null distribution (one-sided). *** $P < 0.001$, ** $P < 0.01$, * $P < 0.05$.

late γ activity selectively signaled the participant's own decision to cooperate ($\beta = 0.183$, $P = 0.004$; Fig. 4d and Supplementary Table 8b), but bore no relationship to the partner's choice ($P = 0.452$; Fig. 4d and Supplementary Table 8b). This dissociation suggested that amygdala low-frequency power was preferentially linked to partner-oriented prospective processing, whereas γ activity was preferentially linked to self-decision.

The model-based analyses further clarified the computations carried by these signals. Early θ power selectively tracked the model-derived prospective estimate of partner cooperation, such that stronger early θ suppression was associated with higher estimated probability of partner cooperation ($\beta = -0.121$, $P = 6.05 \times 10^{-4}$; Fig. 4e; survived FDR correction for multiple comparisons; Supplementary Table 8c). Estimate uncertainty was encoded more broadly by low-frequency power, including both early θ ($\beta = -0.079$, $P = 0.016$; Fig. 4e) and later low-frequency responses ($\beta = -0.120$, $P = 0.007$; Fig. 4f and Supplementary Table 8c), indicating that low-frequency dynamics also carried an uncertainty-related component of the prospective partner-estimation state. In contrast, retrospective cooperative PE, defined relative to the model-derived prospective estimate, was encoded only at a later stage, with significant effects in late low-frequency ($\beta = 0.064$, $P = 0.015$, Fig. 4f) and γ

power ($\beta = 0.062$, $P = 0.018$; Fig. 4g), but not in early θ ($P = 0.141$; Fig. 4e and Supplementary Table 8c). This temporal and spectral dissociation indicates that early low-frequency suppression was most closely linked to the prospective estimate of partner cooperation, whereas later low-frequency and γ power reflected retrospective evaluation of whether the partner had been more cooperative than estimated.

Finally, we asked whether partner estimation-related and self-decision-related signals interacted during MC. Building on previous work suggesting that low-frequency phase can modulate high-frequency amplitude to facilitate cross-timescale coordination^{40,41}, we performed phase-amplitude coupling (PAC) analyses within the amygdala. The phase of low-frequency oscillations entrained γ -band amplitude selectively during MC (MC > non-MC; θ phase (4–8 Hz) to γ amplitude (53–62 Hz); $P_{\text{corr}} = 0.027$, paired Student's *t*-tests with cluster-based permutation correction; Fig. 4h). A paired-choice permutation test ($n = 10,000$) showed that PAC values in real MC trials exceeded the 95% CI of the null distribution ($P_{\text{corr}} = 0.001$), ruling out the possibility that stronger PAC during MC was merely driven by the same choice and confirming its specificity to MC. Moreover, significant MC-related PAC was observed only in the late window, but not in the early phase ($P = 1$, one-sided, paired Student's *t*-test), suggesting temporal specificity. This phase-mediated modulation

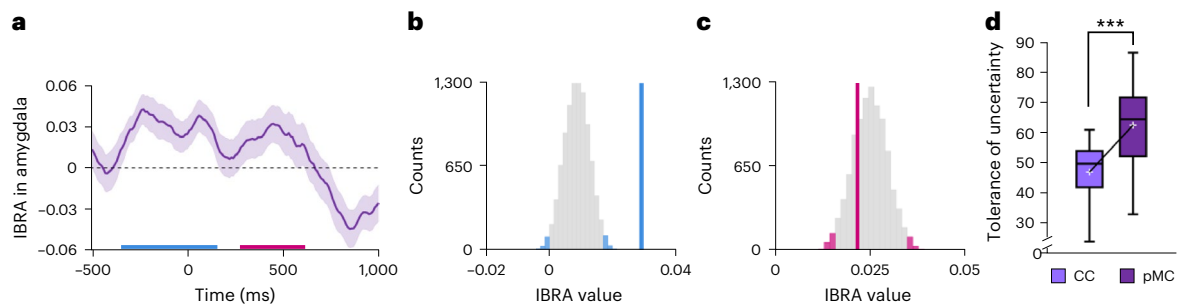


Fig. 5 | Neural mechanisms of unconditional cooperation. **a**, Amygdala IBRA time course (colored curves representing mean values across channel-pairs with shaded areas indicating 95% CI) in pMC dyads revealing an ultra-early alignment window (−340 ms to 170 ms) and a later window (290–630 ms). The horizontal lines indicate significant timepoints against 0 (one-sided, one-sample Student's *t*-test; multiple-comparison corrected by cluster-based permutation test, $n = 10,000$). **b, c**, Null distributions from pseudo-MC IBRA ($n = 10,000$) confirming that the real value of early IBRA exceeded the 95% CI of

the null distribution (**b**), whereas the late IBRA did not (**c**). Null distributions are plotted with values exceeding 95% CI colored, whereas vertical lines indicate the observed IBRA values. **d**, The pMC dyads reporting higher uncertainty tolerance than conditional-cooperation dyads (two-sided, independent-samples Student's *t*-test). Box plots show medians (center line), IQR (box edges), whiskers (1.5× IQR) and mean values (crosses). See Supplementary Table 9 for full statistics: *** $P < 0.001$.

of γ power in the amygdala suggests a temporally specific interaction through which partner-estimation-related, low-frequency suppression could become linked to γ -band-associated, self-decision processes during MC.

Together, these findings suggest a neurocognitive architecture for resolving the commitment problem through prospective inference. By estimating the partner's intentions through amygdala low-frequency suppression and engaging in temporally specific cross-frequency coupling during self-decision, individuals may achieve mutual commitment.

Neural mechanisms in unconditional cooperation

Finally, we examined a distinct subset: pure MC dyads (pMC dyads, $n = 19$), who achieved MC from the outset and maintained it throughout. As this subgroup lacks non-MC trials, it does not allow the same within-dyad condition contrast used in conditional cooperation (CC) dyads. Nevertheless, it provides an exploratory opportunity to ask whether neural mechanisms identified in CC dyads are universally applicable regardless of how cooperation is achieved or whether they are specific to situations requiring transformations from non-MC to MC. If the neural processes observed in CC dyads reflect a general feature of MC, then they should also be observed in pMC dyads.

We observed significant amygdala ($n = 137$ channel-pairs) IBRA increases in pMC dyads relative to 0, in two distinct temporal windows. An ultra-early alignment window emerged before trial onset (−340 ms to 170 ms, $P_{\text{corr}} = 1.00 \times 10^{-4}$, 10,000 permutations, one-sided, one-sample Student's *t*-test; Fig. 5a), a pattern unique to real-time alignment and stronger than temporally shuffled controls ($P_{\text{corr}} = 1.00 \times 10^{-4}$, 10,000 permutations; Fig. 5b). A later IBRA window (290–630 ms, $P_{\text{corr}} = 0.003$; Fig. 5a) was also present in both real-time and shuffled-control conditions, suggesting a more MC-outcome-related alignment pattern (real versus shuffled, $P_{\text{corr}} = 0.477$; Fig. 5c). The earlier IBRA onset in pMC (pMC: −340 ms; CC: 70 ms, pre-onset IBRA of CC dyad was insignificant; Supplementary Fig. 4) suggested a potential for pre-emptive neural alignment when freed from commitment struggles or trust stabilized.

Time–frequency decomposition revealed distinct spectral patterns of early and late amygdala IBRA in pMC dyads. We observed significant increases in amygdala low-frequency power in pMC dyads (θ in the earlier window: $P = 0.007$; low-frequency in the later window: $P = 9.26 \times 10^{-4}$; survived FDR correction across frequency bands; Supplementary Fig. 5a,b), different from the low-frequency suppression observed in CC dyads (Fig. 3e–h). In contrast, late IBRA showed γ -band increases in pMC dyads ($P = 6.33 \times 10^{-5}$; Supplementary Fig. 5c), resembling that observed in CC dyads. This spectral similarity suggested that

high-frequency activity may represent a shared neural substrate for cooperation-related convergence, irrespective of cooperative stability.

To explore why some dyads naturally fall into the pMC category, we conducted trait-level assessments. Although pMC and CC dyads showed comparable levels of empathy and prosocial preference ($P > 0.05$; Supplementary Table 9), pMC dyads scored significantly higher on uncertainty tolerance than CC dyads ($P = 6.75 \times 10^{-4}$; Fig. 5d and Supplementary Table 9). This suggests that dispositional comfort with unpredictability may scaffold early cooperative commitment, enabling MC to emerge more reflexively, without requiring deliberate strategic simulation.

In summary, these results indicate that the core mechanism of interbrain alignment, indexed by amygdala IBRA, is preserved across both CC and pMC dyads, suggesting real-time neural convergence as a general feature of MC. However, differences in their temporal onset and low-frequency spectral profiles of amygdala alignment suggest that MC may be supported by partially overlapping, but not identical, neural processes across dyads. Given the absence of within-dyad condition contrasts in pMC dyads and the state-dependent spectral differences between CC and pMC dyads (Supplementary Fig. 6), these differences should be interpreted with caution.

Discussion

How do two autonomous minds, each harboring private intentions and uncertainty about the other, converge on MC in real time? Our findings suggest two amygdala-mediated mechanisms that scaffold this convergence: (1) IBRA, which aligns task-relevant representations across interacting brains to overcome decisional independence; and (2) social prospective simulation, which we operationalize here as a model-based prospective estimate of the partner's likely cooperation that can be linked to one's own decision process under uncertainty. These mechanisms suggest a neurocognitive route through which interacting individuals may overcome the two barriers emphasized by classic game theory—the defection equilibrium and the commitment problem. MC, we argue, emerges not merely from thinking about the other, but from dynamically embedding a prospective estimate of the partner's likely cooperation into one's own decision process.

By tracking fine-grained spectral patterns using hyper-iEEG, we show that MC is scaffolded by amygdala-mediated, temporally structured representational alignment. Notably, our core measure (IBRA) was initially quantified as time-resolved similarity in spectral patterns between partners. Given the inherent variability of human cognitive processing, such temporally precise alignment may seem counterintuitive. Follow-up time-lagged analyses (Supplementary Note 2) revealed

that IBRA was better understood as temporally diffuse alignment, in which one's neural patterns at a given moment resembled an extended temporal window in their partner's brain (Extended Data Fig. 7). This temporal diffuseness suggests that IBRA reflects alignment at the level of cognitive processes, rather than simple neural entrainment. Importantly, this process-level alignment also carried behaviorally meaningful directional structure. By anchoring time-lagged IBRA to behaviorally meaningful asymmetries, we found that temporal precedence was related to differences in partner-predictive signal strength (which measured how strongly the participant's low-frequency power predicted the partner's upcoming choice; Extended Data Fig. 8a–c) and trial-wise decision speed (Extended Data Fig. 8d–f). During MC, the participant with stronger partner-predictive signals and the faster decision-maker tended to lead the lagged alignment structure, whereas non-MC trials showed different or reversed lag profiles (Extended Data Fig. 8b,c,e,f). Thus, IBRA was not merely symmetrical co-fluctuation, but also organized by asymmetries in ongoing decision processes. Time-resolved decoding and behavioral association analyses further revealed sustained relationships between IBRA strength and cooperation-related behavioral metrics, indicating that the functional relevance of neural alignment itself unfolds across extended temporal windows. This process-level interpretation is further reinforced by decision–time-normalized analyses, which demonstrated that the early–late structured, sustained IBRA remained robust when trials were aligned by relative decision progress, irrespective of absolute physical duration. Together, the observation that both IBRA and its behavioral relevance are temporally diffuse, directional and bound to decision stages suggests that amygdala-mediated alignment reflects a process-level mechanism, possibly reflecting the sustained, predictive coordination of internal decision processes during MC.

In addition to the amygdala, IBRA was also observed in the TPJ. However, unlike the amygdala, TPJ IBRA did not exhibit condition specificity, emerging across both cooperative and noncooperative interactions. Rather than suggesting that TPJ IBRA is unnecessary for cooperation, this pattern indicates that TPJ IBRA may reflect a more general process engaged during real-time interaction rather than a mechanism specific to cooperative convergence. One plausible interpretation is that TPJ-mediated alignment reflects ongoing mental-state inference^{8,39} through which interacting individuals attempt to interpret their partner's intentions during strategic exchange. By contrast, the selective emergence of amygdala IBRA during MC suggests that successful cooperative convergence requires an additional layer of coordination beyond mentalizing: the alignment and integration of partner-related representations into one's own decision dynamics.

What, then, is the nature of this aligned process? Our data suggest that an amygdala-mediated 'simulation-to-decision' cascade links prospective partner-estimation and self-decision processes and orchestrates MC. In the present task, this cascade is likely expressed as a prospective partner-estimation process. Early amygdala θ suppression was shaped by prior interactions and prospectively tracked the partner's intention and likely cooperative behavior. Given the role of amygdala θ oscillations in fear detection and social vigilance^{42,43}, this θ suppression may reflect reduced social vigilance or uncertainty-related processing, thereby enabling forward modeling of the partner's intent. Early within-dyad aligned θ suppression suggests simultaneous engagement in social forecasting, creating the cognitive conditions necessary for trust. Importantly, the model-based analyses refine this interpretation by showing that early low-frequency dynamics carried a prospective partner-estimation signal, whereas broader low-frequency power carried an uncertainty-related component of that prospective estimate. This partner-estimation process may lay the foundation for one's own decision to cooperate, encoded by amygdala γ activity. Notably, this γ activity did not appear as an isolated execution-related signal, but rather had its amplitude modulated by the phase of θ oscillations. Amygdala θ – γ coupling therefore provides a plausible link between

prospective partner-estimation and cooperative action. This interpretation is also broadly consistent with recent computational work suggesting that cross-frequency interactions can support coordinated neural dynamics in social interaction and interbrain synchronization⁴⁴. Although these findings were consistent with the idea that aligned amygdala dynamics may help reduce uncertainty during interaction, the present study did not directly manipulate or quantitatively measure uncertainty. Thus, the proposed role of uncertainty processing should be interpreted as a theoretical inference grounded in prior literature^{45,46} and trait-level behavioral patterns, rather than as direct evidence of uncertainty modulation in the amygdala. Likewise, although the temporal profiles of amygdala IBRA and time-resolved decoding indicate temporal precedence of IBRA relative to cooperative outcomes, they do not establish that the amygdala causally drives MC. Future work that experimentally manipulates informational uncertainty or partner predictability or uses causal perturbation approaches (for example, electrical stimulation or lesion studies) will be necessary to test this mechanism directly.

However, the extent to which this simulation-to-decision cascade generalizes across different cooperative modes remains an open question. Pure cooperators, who consistently choose cooperation, showed a distinct neural profile: early amygdala θ increases and anticipatory IBRA before trial onset. Although these findings are suggestive rather than decisive due to the absence of within-dyad condition contrasts, they point toward a possible functional shift in amygdala θ 's role under stable cooperation conditions. θ increases in pure cooperators may tentatively reflect proactive coordination, affiliative expectation confirmation or dispositional trust engagement. Supporting this interpretation, prior work has linked amygdala θ to context-specific salience, safety signaling in predictable environments and motivational internal-state tracking^{47–49}. Notably, pure cooperators lacked the characteristic low-frequency power increase during their very early interactions (Supplementary Fig. 6), yet their behavior remained consistently cooperative. This initial behavioral resilience may be gated by individual traits. Our trait-level analyses indicated that pMC dyads, relative to CC dyads, were not uniquely prosocial, but more tolerant of uncertainty—highlighting uncertainty tolerance, rather than conventional assumptions of altruistic dispositions, as the key differentiator facilitating stable MC. High uncertainty tolerance may enable unconditional cooperators to behaviorally overcome the initial interactive uncertainty that otherwise would require prospective partner estimation. Rather than a simple linear progression, these two modes may represent distinct 'stable states' in cooperative dynamics, gated by individual differences. This distinction aligns with dual-process models of cooperation⁵⁰, suggesting that both deliberative and intuitive pathways can support MC.

Yet despite these differences, both cooperative profiles shared a fundamental reliance on IBRA. Although their temporal dynamics and representational contents vary, the presence of IBRA underscores within-dyad neural convergence as a hallmark of MC. Moreover, both dyad types address uncertainty: CC through interaction-based social forecasting and pMC through trait-level tolerance accelerating the transition to stable coordination. Together, these findings identify two components necessary for MC: (1) shared representational modes enabling dyadic alignment and (2) uncertainty-resolution mechanisms. These components map directly onto the two classic dilemmas in game theory: decisional independence and informational uncertainty^{10,14}. Our findings thus offer an amygdala-mediated neural solution that dynamically integrates individual neural computations into collective, cooperative outcomes.

In conclusion, our study provides a mechanistic account of MC as a neurally interdependent phenomenon, instantiated by amygdala-mediated representational alignment and a prospective partner-estimation process. By elucidating distinct pathways to cooperative convergence, we concretize the theoretical construct of neural

interdependency, addressing MC's two Achilles' heels—interdependence and uncertainty—and binding separate cognitive systems into a unified cooperative unit. This interdependency offers a bridge between individual neural computations and shared social outcomes—transforming MC from a theoretical ideal into a biologically instantiated process. Conceptually, these findings encourage a transformative shift from viewing social cognition primarily as the individual representation of others, toward understanding social interdependence as the dynamic alignment of internal models across minds. Future research combining noninvasive neuroimaging with more diverse populations should extend this framework to examine the generalizability of interbrain dynamics and partner-estimation-to-decision cascades across broader domains, such as moral consensus building, collective problem-solving, negotiation, social bonding and other forms of shared cognition.

Online content

Any methods, additional references, Nature Portfolio reporting summaries, source data, extended data, supplementary information, acknowledgements, peer review information; details of author contributions and competing interests; and statements of data and code availability are available at <https://doi.org/10.1038/s41593-026-02473-z>.

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Methods

Participants and ethics

Fifty-eight patients with epilepsy participated while undergoing iEEG monitoring for clinical seizure localization. Electrode placement was determined exclusively by clinical needs. During each experimental session, two concurrently hospitalized patients were paired opportunistically to play an iterated PD game. Some patients participated in multiple sessions with different partners because recruitment was constrained by overlapping clinical availability. Each pairing constituted a distinct dyad ($n = 47$) and each patient in the dyad was treated as a separate session-participant observation for individual-level analyses. Patients were seated separately and unable to view each other's screens.

Eligibility required at least one electrode in the amygdala or TPJ, seizure free for ≥ 3 h before testing, no major psychiatric disorder, head trauma or encephalitis and the capacity to complete computerized decision-making tasks. Two dyads were excluded because they failed to complete the task and never achieved MC, leaving a final behavioral dataset of 45 dyads (corresponding to 90 session-participant observations; Supplementary Table 1 and Supplementary Fig. 1). Among these 45 dyads, 15 had amygdala channel-pairs (that is, at least one clean amygdala electrode in each patient, $n_{\text{channel-pair}} = 278$) and 15 had TPJ channel-pairs ($n_{\text{channel-pair}} = 812$); some dyads contributed to both regions (Supplementary Table 1).

Behaviorally, 45 dyads fell into two patterns: 19 cooperated on all 30 trials and were classified as pure mutual cooperators (pMC dyads; Supplementary Table 1b); the remaining 26 dyads (Supplementary Table 1a) exhibited both MC and non-MC and were classified as conditional cooperators (CC dyads). The MC rate in CC dyads was stable across trials (no significant change with trial number, $P = 0.107$).

All procedures conformed to the Declaration of Helsinki and were approved by the Institutional Review Boards of Chinese PLA General Hospital (protocol no. S202139402) and Beijing Tiantan Hospital (protocol nos. KY 202008002 and KY 2023-034-02). All participants provided informed consent and could withdraw without affecting clinical care. The study involved no deception and participants were compensated.

Sample-size estimation

Similar to prior iEEG studies^{33,51–54}, no formal a priori sample-size estimation was conducted. However, the numbers of dyads, patients, clean channels and channel-pairs (Supplementary Table 1) were comparable to or larger than those in recent iEEG studies^{55,56} and multi-brain recording studies^{3,18,19}. Our main findings were statistically robust and survived multiple-comparison corrections.

Real-time iterated PD

We employed a two-player, real-time iterated PD to elicit cooperation and defection in a controlled yet ecologically valid social setting. On each trial, each participant chose to cooperate (C) or defect (D). Payoffs (in monetary units (MU)) followed a standard PD ordering: T (temptation payoff to unilateral defector, 4 MU) $> R$ (reward for mutual cooperation, 3 MU) $> P$ (punishment for mutual defection, 2 MU) $> S$ (sucker payoff to unilateral cooperator, 1 MU), with $2R > T + S$ satisfied. Participants knew that both choices jointly determined their earnings. An optional costly punishment stage was included to maintain reputational stakes and discourage an always-defect strategy. Neutral instructions were used to minimize demand characteristics.

Each experiment session consisted of 30 trials (Fig. 1c). The trial events were as follows: (1) decision phase (self-paced, +fixation jitter 600–1,000 ms): both players simultaneously viewed the choice screen and chose between cooperate and defect. (2) Dyadic reveal and outcome screen (self-paced, +fixation jitter 600–1,000 ms): both players' choices and resulting payoffs were displayed simultaneously

on each player's screen. (3) Optional punishment phase (self-paced): the player (defected upon) could choose to punish the partner or not (cost-to-penalty ratio, 1:2), +2,000-ms feedback. (4) Intertrial interval: 1,200–2,000 ms, jittered. Participants were not told the total number of trials. Testing computers were synchronized over a local network.

Behavioral analysis

Trials were categorized as MC when both players chose C and as non-MC otherwise. For participant p , decision time (DT_p) was measured from decision-screen onset to response. Dyadic mean DT was $dDT = (DT_1 + DT_2)/2$. Dyadic DT similarity was $dDT_{\text{sim}} = 1 - \text{abs}(DT_1 - DT_2)/(DT_1 + DT_2)$, with larger values indicating greater temporal similarity. DT and dDT were log-transformed and z-scored across trials.

Behavioral differences between MC and non-MC trials were tested with LMEs (fitlme, MATLAB; restricted maximum likelihood), which accommodate unequal trial counts. Condition (MC versus non-MC) was entered as a fixed effect, with dyads and its interaction with trial number as two random-effect variables. Control analyses (1) added individual DTs as covariates (confirming faster MC effect; $P = 0.004$) and (2) used within-dyad trial-shuffling (randomly paired cooperation choices across trials) and confirmed that observed MC-related dDT_{sim} exceeded that expected from shared cooperation choices alone ($P = 1.00 \times 10^{-4}$, one sided). It should be noted that overall behavioral trajectories remained largely distinct between interacting partners (choices: $r = 0.058$, $P = 0.330$; decision times: a significant but small-effect correlation, $r = 0.134$, $P = 0.007$).

To characterize how interaction history influenced current MC in CC dyads, we first fitted generalized LMEs (GLMEs; logit link) predicting MC on trial t ($t > 1$) from three history measures computed over the preceding $k = 1$ –5 trials: (1) MC rate; (2) the dyad's overall cooperation-choice rate (C_{rate}); and (3) the punishment rate (Pun_{rate}). We capped k at 5 to remain within a plausible working-memory capacity⁵⁷ and to limit model complexity given the 30-trial task. Each z-scored regressor and history-length k was tested separately, with FDR correction. The three-trial MC rate produced the largest standardized β and was therefore used in subsequent analyses (Supplementary Table 3a). Furthermore, we conducted forward stepwise model selection (stepwiseglm function in MATLAB) beginning with an intercept-only model, with three-trial MC_{rate} , C_{rate} and Pun_{rate} specified as candidate predictors. At each step, the remaining candidate predictors were evaluated individually and entered only if their inclusion significantly improved model fit at the threshold of $P < 0.05$. For MC likelihood, changes in deviance were evaluated using χ^2 tests; for MC speed, model improvement was evaluated using F tests (Supplementary Table 3b).

We also compared the observed history dependence of MC with two classic reciprocal strategies³⁸ (that is, Tit-for-Tat and Win-Stay-Lose-Shift). Tit-for-Tat was defined as copying the partner's preceding action. Win-Stay-Lose-Shift was defined as repeating one's action after higher-payoff outcomes (MC or unilateral defection) and switching after lower-payoff outcomes (mutual defection or unilateral cooperation). GLMEs were then fitted to predict trial-wise MC from each strategy.

Computational modeling of prospective partner estimation

We constructed four Bayesian models^{58,59} in which participants estimated the partner's probability of cooperation from different sources of information: (1) the partner's own choice history (consistency-assumption model); (2) the participant's own choice history as a proxy for expected reciprocity (reciprocity-assumption model); (3) dyadic MC versus non-MC history (dyadic-outcome model); or (4) the combination of both self-choice and partner-choice histories (self-and-other-history model).

For each information source, accumulated evidence for cooperation-like and defection-like outcomes was represented by a conjugate

β distribution: $\beta(\alpha_t, \beta_t)$. Following the trial ($t-1$), the evidence counts were updated as:

$$\alpha_t = \alpha_{t-1} + C_{t-1}$$

$$\beta_t = \beta_{t-1} + D_{t-1}$$

where $C_{t-1} = 1$ and $D_{t-1} = 0$ for cooperation-like outcomes (cooperation choice or MC outcome), and $C_{t-1} = 0$ and $D_{t-1} = 1$ for defection-like outcomes (defection choice or non-MC outcome), depending on the information source used in a given model.

For each source-specific β distribution, the prospective estimate of partner cooperation and uncertainty of that estimate were the posterior mean and variance:

$$\mu_t = \alpha_t / (\alpha_t + \beta_t)$$

$$\sigma_t^2 = (\alpha_t \times \beta_t) / ((\alpha_t + \beta_t)^2 \times (\alpha_t + \beta_t + 1))$$

The probability of partner cooperation on trial t was obtained using a logistic decision rule:

$$P_t = \frac{1}{1 + e^{-\zeta \times (\mu_t - \psi)}}$$

where ζ is an inverse-temperature parameter and ψ is a cooperation-threshold parameter.

Models were fitted separately for each participant using maximum a posteriori estimation⁶⁰. Group-level Bayesian model selection⁶¹ identified the self-and-other-history model as the winning model (expected posterior model frequency: 0.911; protected exceedance probability: >0.999). This winning model was then used to generate trial-wise computational variables for subsequent behavioral and neural analyses.

In the winning model, posterior estimates based on partner-choice and self-choice histories were updated in parallel and integrated using a participant-specific weighting parameter (w): (1) the final model-derived prospective estimate of partner cooperation was calculated as the weighted mean of the two source-specific posterior means; (2) estimate uncertainty was calculated as the correspondingly weighted mean of their posterior variances; and (3) retrospective cooperative PE was defined at the estimate level, before the logistic response transformation, as the discrepancy between the partner's actual choice on the previous trial (partner _{$t-1$} , cooperate = 1, defect = 0) and the model-derived prospective estimate for that trial (μ_{t-1}). Positive values indicated that the partner had been more cooperative than estimated.

To examine how these model-derived variables related to observable behavior, we fitted GLME models predicting either the partner's current choice or the participant's own current choice. Fixed effects included estimate mean, estimate uncertainty and retrospective cooperative PE. Random intercepts were included for participant and P values were adjusted using FDR correction. In additional control analyses, the participant's own current choice was included as a covariate when predicting the partner's current choice, to ensure that the effects of the model-derived variables were not reducible to concurrent choice coupling.

Acquisition of iEEG data, electrode localization and preprocessing

The iEEG data were acquired with Nicolet amplifiers at 4,096 Hz (Chinese PLA General Hospital) or Nihon-Kohden amplifiers at 2,000 Hz (Beijing Tiantan Hospital). During recording, raw iEEG signals were referenced to an electrographically silent channel outside the seizure

foci. Depth electrodes were 0.8 mm in diameter and contained 8–16 contacts (2-mm contact length, 1.5-mm spacing).

Electrode locations were determined in each patient's native space by co-registering postoperative head computed tomography and pre-operative structural magnetic resonance imaging using established methods⁶². These locations were then projected onto a normalized cortical surface in the MNI space and visualized on the 'ICBM152' template⁶³. These steps were performed using the FieldTrip toolbox⁶⁴, Freesurfer software⁶⁵ and Statistical Parametric Mapping (SPM12)⁶⁶ implemented in MATLAB.

An established iEEG data-preprocessing pipeline^{32,53} was implemented as follows: raw iEEG signals were downsampled to 1,000 Hz, bandpass filtered at 1–250 Hz and notch filtered at 50 ± 3 Hz and harmonics. All channels underwent a quality check. Channels in the epileptic zone or showing substantial epileptic activity, abnormal power spectra, extreme variance ($>5\times$ or $<5\times$ the median variance across all channels) or excessive ($\geq 3\times$ the median number) spikes (defined as 100- μ V changes between consecutive samples) were excluded. All remaining channels were further visually inspected. Clean channels were re-referenced to the average activity across all clean channels (that is, common average reference)^{34,51,53}. We opted for common average reference because it effectively reduces global noise while preserving the broad, distributed spectral signals across electrodes. This is critical for our core metric (that is, IBRA) calculated from the high-dimensional pattern of spectral power across a broad frequency range⁶⁷. Our main condition-specific IBRA effects were replicated (Extended Data Fig. 9a) using white-matter referencing^{41,67}.

Interictal epileptiform discharges (IEDs) were detected using a double-thresholding algorithm⁵²: (1) the envelope of the unfiltered signal exceeded 5 s.d. values above baseline or (2) the envelope of the filtered signal (15-Hz to 80-Hz bandpass-filter) exceeded 6 s.d. values above baseline. IEDs were further processed by smoothing the vector of identified IED samples using a one-dimensional, Gaussian-weighted, moving average filter (window length = 0.4 s). Any samples with values >0.01 after smoothing were also marked as IED samples. Our neural analysis focused on the decision-phase epochs (from -200 ms to 1,500 ms relative to decision-screen onset). Epochs containing IEDs were excluded and channels with $>30\%$ rejected epochs were removed. These procedures resulted in the exclusion of 7.89% epochs and 3.5% channels. All neural analyses were conducted on the clean trials and clean channels.

Spectral decomposition

Spectral power was estimated using a multitaper-based method^{54,68} (4–32 Hz: 6 cycles and 3 Slepian tapers per window; 32–200 Hz: a fixed 240-ms window, with 4–31 tapers increasing with frequency per window). Spectral power was calculated at 60 logarithmically spaced frequencies from 4 Hz to 200 Hz and downsampled to 100 Hz. For each frequency and channel, we pooled all fixations (200–500 ms) as baselines and normalized the power by subtracting the baseline mean and dividing by its s.d.⁶⁹.

IBRA

IBRA quantified similarity between the spectral patterns of interacting participants. Specifically, normalized power from 4 Hz to 200 Hz (60 logarithmically spaced frequencies) was averaged in 200-ms windows (10-ms increments) from decision-screen onset to 1,500 ms. For each time window and trial, spectral power across frequencies formed a feature vector for each channel. Spearman's correlations were then computed between feature vectors from paired same-region channels in the two interacting brains and Fisher's z -transformed, yielding a time-resolved IBRA profile for each channel-pair and trial. Analyses using shorter 100-ms windows replicated the MC-specific IBRA effects (Supplementary Fig. 2).

To identify MC-specific IBRA, MC and non-MC IBRA time courses were compared at each timepoint using two-sided, paired-sample

Student's *t*-tests across channel-pairs, with cluster-based permutation tests ($n = 10,000$; Supplementary Note 3). Significant condition effects were then further tested against 0 using one-sided, one-sample Student's *t*-tests (Supplementary Table 5). Amygdala analyses identified early (70–380 ms) and late (480–1,160 ms) MC-specific clusters. For subsequent window-averaged analyses, these clusters were standardized to adjacent 50-ms-aligned windows of 50–400 ms and 500–1,200 ms. This standardization did not affect their original time-resolved identification.

As channel-pairs were nested within dyads and some patients participated in multiple dyads, we performed confirmatory analyses to verify that the primary condition effects remained after accounting for the hierarchical structure of the data. Time-resolved LMEs included condition (MC versus non-MC) as a fixed effect and random intercepts for dyad and channel-pair (Extended Data Fig. 2). Additional analyses used a reduced dataset retaining only one channel-pair for any physical contact repeated across dyads (Extended Data Fig. 9b), included patient identity as a random effect (Extended Data Fig. 9c) and aggregated IBRA at the dyad level. These supplementary analyses replicated the primary results (Supplementary Table 5).

IBRA controls and content validation

We assessed possible baseline and timing confounds using complementary controls. Pre-stimulus fixation IBRA did not differ between MC and non-MC (Extended Data Fig. 9d), confirming comparable baseline representational alignment. With regard to timing-related confounds, we performed the following: (1) covariate control: trial-wise dDT and dDT_{sim} were included as fixed-effect covariates in the LMEs (Extended Data Fig. 3a). (2) Propensity-score matching analysis: MC and non-MC trials were propensity-score matched on dDT and dDT_{sim} (Extended Data Fig. 3b). (3) Response-locked analysis: trial-wise neural time series were realigned to each participant's response (−1,500 ms to +500 ms; Extended Data Fig. 3c). (4) DT normalization: for each trial, stimulus-to-response neural time series was rescaled to a standardized length of 150 time-bins (cubic interpolation; Extended Data Fig. 3d). IBRA was then recomputed at each bin using the same method as described above. We further compared observed IBRA with three permutation distributions that disrupted trial correspondence, paired-cooperation choices or paired dyadic outcomes while preserving trial counts. Full procedures are described in Supplementary Note 1.

To test whether amygdala IBRA reflected behaviorally meaningful alignment across brains beyond same-trial simultaneity, we performed a crossbrain, crosstrial RSA. For each dyad, we constructed a 30×30 crossbrain neural similarity matrix, in which each element (i, j) was defined as the Spearman's correlation between the spectral vector (4–200 Hz) from trial i in one participant and trial j in the partner. The diagonal of this matrix (that is, $i = j$) corresponds to the original same-trial IBRA, whereas off-diagonal elements capture across-trial, across-brain similarity. A corresponding interaction-history similarity matrix (30×30) indexed 1 – the absolute difference in three-trial MC history. History similarity was masked separately by MC–MC and non-MC–non-MC trial-pair matrices to generate condition-specific, history-similarity matrices. Partial correlations tested whether condition-specific history similarity explained neural similarity after controlling for the full history-similarity matrix, the corresponding condition-pair matrix (MC–MC or non-MC–non-MC) and a diagonal matrix indexing same-trial comparisons (Extended Data Fig. 4).

We also tested whether amygdala IBRA carried information predictive of cooperative outcomes by performing time-resolved decoding analyses. IBRA was averaged across all amygdala channel-pairs within each dyad. At each timepoint, these values were used as features to classify trial outcomes using a linear discriminant classifier. Leave-one-dyad-out cross-validation^{3,70} assessed generalization and

statistical significance was assessed using a cluster-based permutation test (500 within-dyad label permutations; Extended Data Fig. 5).

Dissociation of early and late amygdala IBRA

Relationship between IBRA and behavior. For each MC trial, we extracted early IBRA onset and mean early and late IBRA values for each channel-pair. Onset was the first significant timepoint within the early window, based on one-sided tests against 0 (100-ms windows, 10-ms steps; FDR corrected across all timepoints). LMEs that included dyad and channel-pair identity as random effects tested relationships of recent three-trial MC history with IBRA onset or intensity, and relationships of IBRA with dDT and dDT_{sim}. Time-resolved LMEs provided a window-independent confirmation. All continuous variables were standardized and two-sided *P* values were adjusted via FDR correction. LME-based mediation with 5,000 bootstrap samples tested whether early IBRA onset and/or late IBRA intensity mediated the association between MC history and current MC decision speed.

Spectral profile of IBRA. To characterize the spectral profiles of MC-specific early and late IBRA, baseline-normalized power within MC trials was tested against 0 across channels using cluster-based permutation tests ($n = 10,000$). Power was additionally averaged in the θ (4–8 Hz), α (8–15 Hz), β (15–35 Hz), γ (35–70 Hz) and high-frequency broadband (70–200 Hz), for both early and late time windows, with FDR corrected for multiple frequency bands. For visualization, power time series at each frequency were smoothed using a one-dimensional, Gaussian-weighted, moving average filter (window length = 200 ms).

Responsive channel-pair identification. We identified interbrain channel-pairs as responsive if they showed both IBRA > 0 during MC and MC > non-MC for at least 50 ms consecutively. We resampled the data with replacement over 1,000 iterations for each condition (MC versus non-MC), matching the trial numbers of the condition with fewer trials. Mean IBRA across these 1,000 iterations was taken as the representative IBRA value for each condition⁵¹. Responsive channel-pairs were further classified as early selective, late selective or shared. Their frequencies and spatial distributions were compared using the χ^2 test and MNI-coordinate tests, respectively (detailed in Supplementary Table 7b).

Neural representation of IBRA-related spectral patterns. We investigated the representational contents of the spectral patterns of the early and late IBRA by examining how their spectral patterns encoded interaction history and current MC outcomes. We constructed 30×30 similarity matrices for: (1) neural spectrograms (spectral power from 4 Hz to 200 Hz of early and late IBRA window), (2) three-trial MC rates and (3) current outcomes. The neural similarity matrix was indexed by the Spearman's correlation between neural spectral patterns of each trial-pair, separately for early and late time windows. The interaction-history matrix was indexed by 1 – the absolute difference in the three-trial MC rate between each pair of trials. To test whether neural spectral patterns reflected past interaction history, we correlated the lower off-diagonal elements of the neural and history-based behavioral matrices. Resulting correlation coefficients were Fisher's *z*-transformed and tested to 0 across all channels using two-sided, one-sample Student's *t*-tests.

Next, we examined whether these IBRA-related spectral patterns were sensitive to current MC outcomes. From the neural similarity matrix, we extracted the similarity values for MC–MC trial pairs, separately for early and late time windows and compared them against 0 across all channels using two-sided, one-sample Student's *t*-tests. Positive values indicated shared representations across MC trials, whereas negative values indicated discriminative patterns. To establish specificity for mutual cooperation, we performed trial-pair shuffle analyses: for each channel, trial labels were randomly shuffled to

generate pseudo-MC–MC pairs, trial-wise neural spectral similarity was recalculated and this process was repeated 10,000 times to yield permutation distributions. The observed MC–MC neural dissimilarity (similarity) was then compared against the 95% CI of the null distribution for each time window.

Neural analysis of prospective partner estimation

We first tested whether amygdala spectrotemporal features associated with IBRA (early θ suppression, later θ and α suppression and late γ increases) predicted overt choices. GLMEs were fitted across trials and channels for self-choice and partner choices, with cooperation coded as 1. The target choice was predicted from neural power while controlling for the other player's concurrent choice (Supplementary Table 8b). The regression coefficient (β) was extracted for each regressor and statistical significance was assessed using FDR correction. We utilized random-intercept-only models to ensure model stability and prevent overfitting, while acknowledging the assumption that the predictive effect of neural power is constant across the participants.

Next, to determine which computational components of the partner-estimation process were encoded by IBRA-related amygdala spectrotemporal features, we related trial-wise neural responses to latent variables derived from the winning model (including model-derived prospective estimate of partner cooperation, estimate uncertainty and retrospective cooperative PE; Supplementary Table 8c). Random intercepts were included for participant and channel nested within participant. An expanded intrabrain dataset, including 11 additional dyads with amygdala coverage in only one participant, replicated the findings (Extended Data Fig. 10).

PAC analysis

We investigated cross-frequency communication within the amygdala, by calculating the PAC values^{40,41}. Specifically, iEEG data were 0-padded and bandpass-filtered to extract instantaneous phase and amplitude. θ and α phase (4–15 Hz, 21 logarithmically spaced frequencies, 2-Hz bandwidth) and γ amplitude (35–75 Hz, step: 2 Hz, adaptive filter bandwidth: 2 $\times$ corresponding low frequency) were obtained using Hilbert's transform. PAC values were calculated as the circular–linear correlation coefficients between the phase and amplitude time series within the late-IBRA time window (corresponding to the time window with γ increase). These values were Fisher's z -transformed and averaged within each condition.

To identify significant PAC clusters for each condition, we performed permutation tests. The amplitude time series were randomly segmented at a given timepoint and the order of the two segments was reversed; afterward, PAC values were recalculated⁷¹. This process was repeated 1,000 times to generate a null distribution. Significant frequency clusters were required both to exceed the within-condition surrogate threshold (95% CI) and to show an MC versus non-MC difference (Fig. 4h). Moreover, mixed-effects models controlling θ and γ power confirmed the MC-related PAC effect.

Analyses in pMC dyads

IBRA in pMC dyads. The amygdala IBRA during the decision phase was examined in pMC dyads ($n_{\text{dyads}} = 19$, $n_{\text{channel-pairs}} = 137$; Supplementary Table 1b). Preprocessing (7.75% bad epochs and 0.8% bad channels were excluded) and IBRA calculation followed the CC-dyad procedure, but the analysis epoch was –500 ms to 1,000 ms relative to stimulus onset because pMC decisions were faster (median, 730 ms versus 1,200 ms in CC). IBRA was averaged across all 30 MC trials and tested against 0. Time-resolved analysis identified two temporal clusters of increased amygdala IBRA in pMC dyads (–340 ms to 170 ms and 290 ms to 630 ms). Subsequent window-averaged analyses used adjacent 50-ms-aligned windows of –350 ms to 200 ms and 300 ms to 650 ms. Full-trial permutation tested whether observed IBRA exceeded randomly paired trials (Supplementary Note 1).

Spectral profile in pMC dyads. The spectral patterns underlying early and late IBRA in pMC dyads were analyzed using the same procedures as for CC dyads.

Trait-level individual differences between pMC and CC dyads.

To explore potential individual differences between pMC and CC dyads, we assessed three key traits: (1) empathic concern (interpersonal reactivity index)⁷²; (2) social preference, measured by social value orientation⁷³; and (3) uncertainty tolerance (intolerance of uncertainty scale)⁷⁴. For each trait, we averaged the scores of both participants within each dyad and compared the dyadic trait scores between pMC and CC dyads using two-sided, independent sample Student's t -tests.

Reporting summary

Further information on research design is available in the Nature Portfolio Reporting Summary linked to this article.

Data availability

The raw magnetic resonance imaging and iEEG data that contain patients' personal information are protected and have restricted access for ethical and privacy reasons. Access can be obtained from the corresponding author upon reasonable request. Preprocessed data used to generate the figures and perform statistical tests are available in the following repository: <https://osf.io/gy829>.

Code availability

Analysis codes are available in the following repository: <https://osf.io/gy829>.

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Author contributions

Y.M. conceived the project, designed the experiments, interpreted the results and wrote the original and final versions of the paper. J.N. analyzed the data under the supervision of Y.M. J.N., F.M., C.Z., H.T., X.Z., J.Y., H.L., Y.Q., Y.Y., Y.Z. and K.Z. performed the experiments and collected data. All the authors approved the final version of the paper for submission.

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Competing interests

The authors declare no competing interests.

Additional information

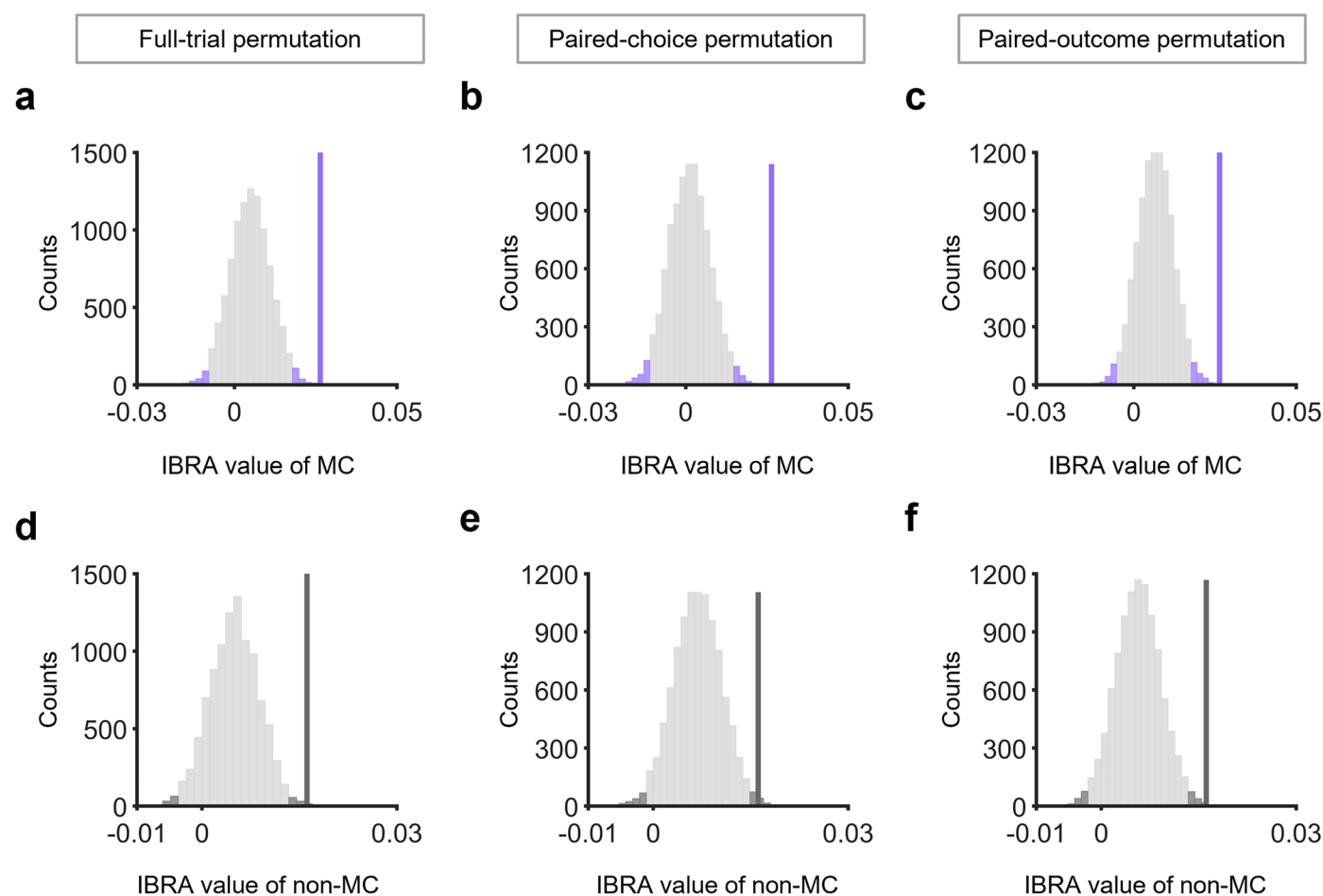
Extended data is available for this paper at <https://doi.org/10.1038/s41593-026-02473-z>.

Supplementary information The online version contains supplementary material available at <https://doi.org/10.1038/s41593-026-02473-z>.

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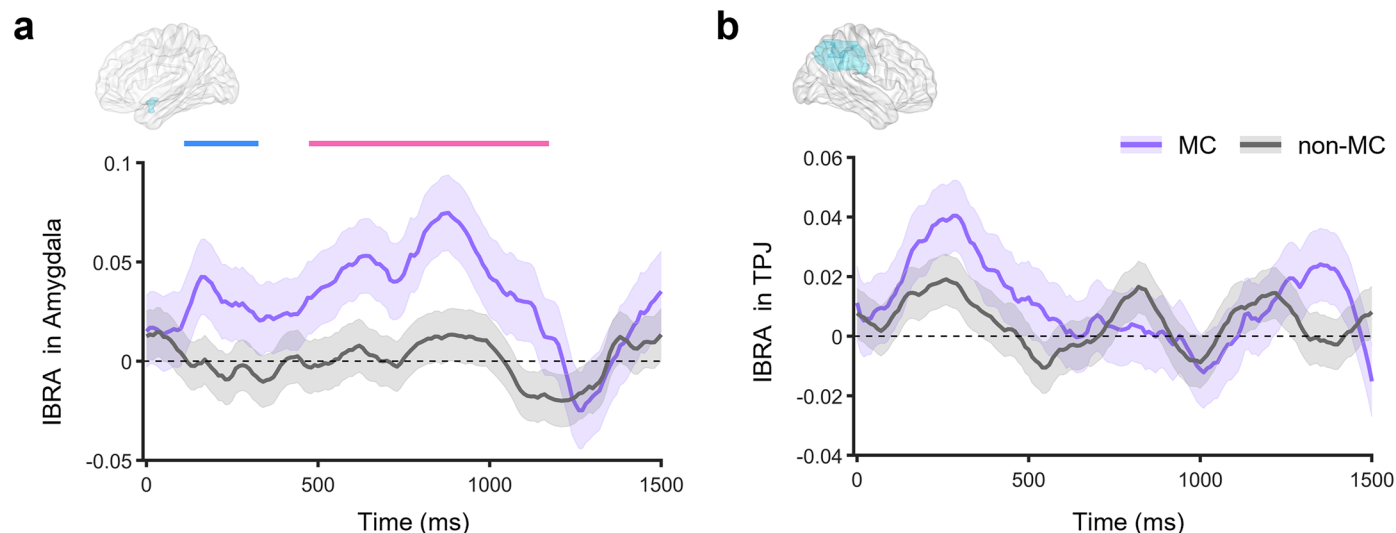
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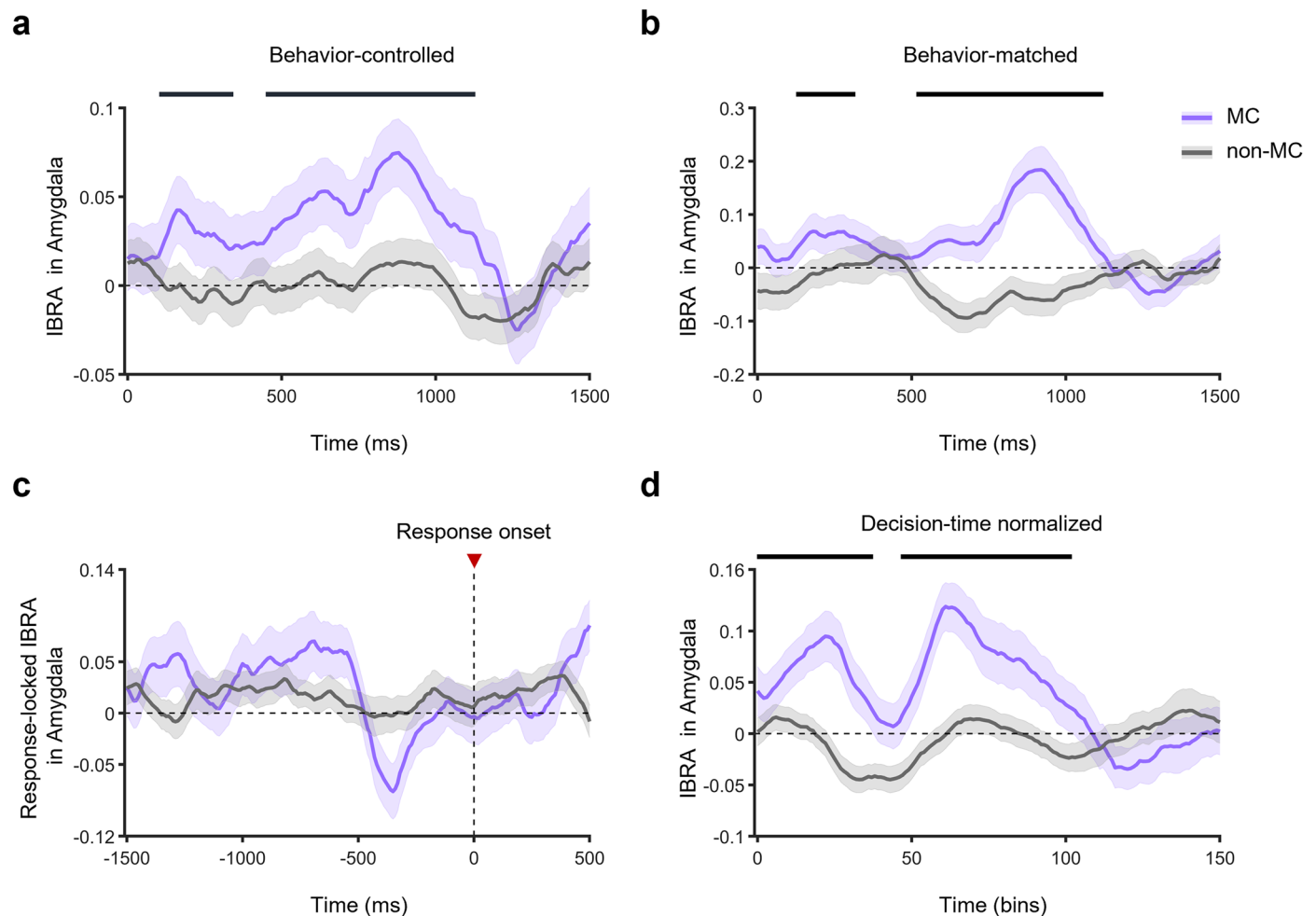
Extended Data Fig. 1 | Validation of real-time-specific TPJ IBRA in both MC and non-MC conditions. Real TPJ IBRA exceeded the 95% CI of the null distribution in both MC (a-c) and non-MC (d-f) conditions in the full-trial permutations (a, MC: $p_{corr} = 8.00 \times 10^{-4}$; d, non-MC: $p_{corr} = 0.003$, $n = 10,000$ permutations, two-sided), the paired-choice permutations (b, MC: $p_{corr} = 1.00 \times 10^{-4}$; e, non-MC: $p_{corr} = 0.012$),

and the paired-outcome permutations (c, MC: $p_{corr} = 7.00 \times 10^{-4}$; f, non-MC: $p_{corr} = 0.004$). Null distributions are plotted with values exceeding 95% CI colored, and purple/grey vertical lines indicate the observed IBRA values of the real MC/non-MC condition.



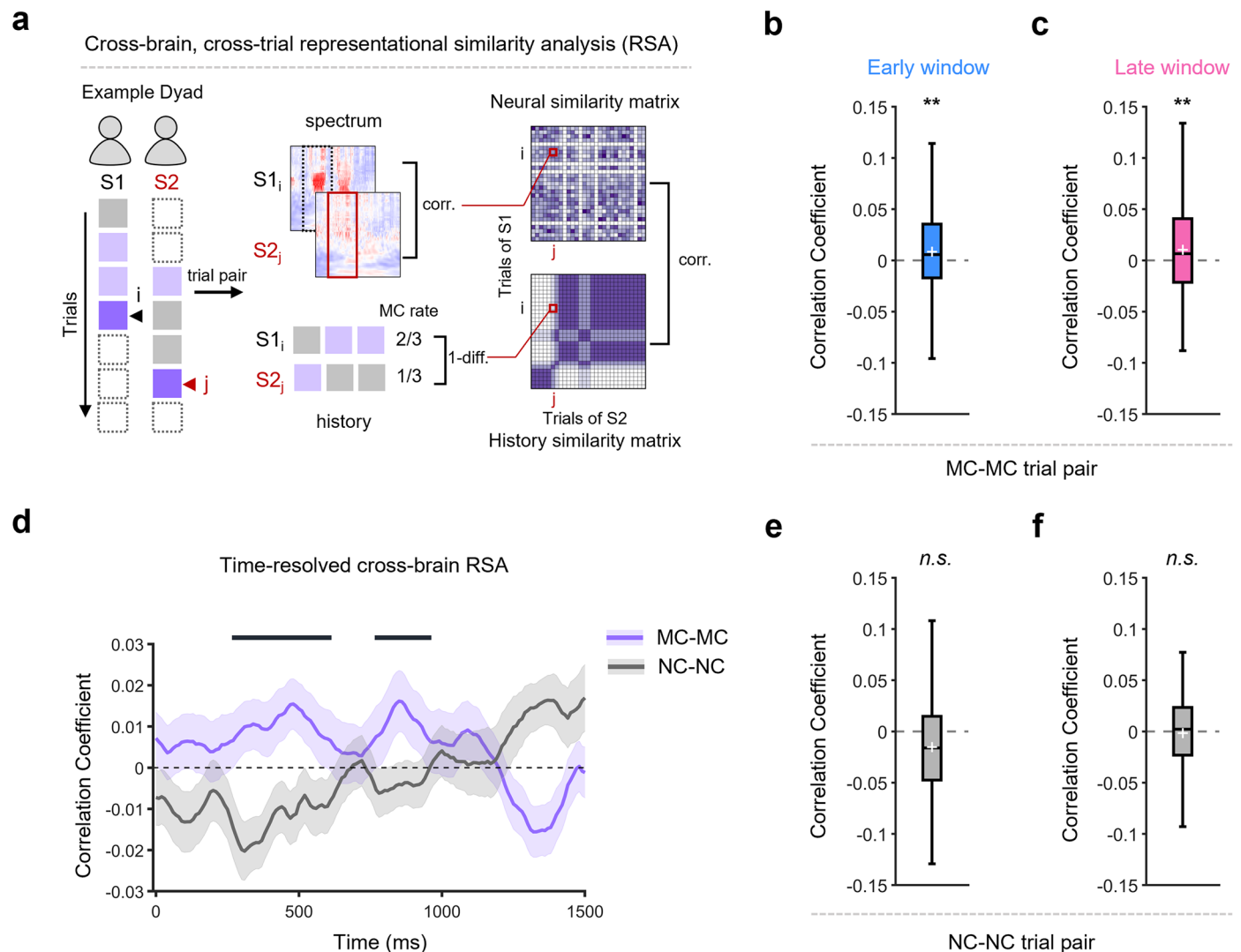
Extended Data Fig. 2 | IBRA results in the amygdala and TPJ were replicated with time-resolved LME analysis. Time courses of IBRA in the amygdala (**a**) and TPJ (**b**) during MC and non-MC conditions across all trials, assessed using time-resolved linear mix-effects models (LME). MC-specific IBRA increases were observed in two time windows in the amygdala (**a**): 120 ms to 330 ms ($t_{\text{sum}} = 64.050$, $p_{\text{corr}} = 0.021$) and 480 ms to 1190 ms ($t_{\text{sum}} = 290.523$, $p_{\text{corr}} = 2.00 \times 10^{-4}$), whereas no significant condition difference was found in TPJ (**b**).

LMEs were conducted at each time point with condition (MC/non-MC) as a fixed effect dyad and channel-pair as random effects. Multiple comparisons were corrected through cluster-based permutation testing ($n = 5,000$, two-sided). Colored curves represent mean values across trials with shaded areas indicating 95% CIs. Horizontal lines indicate time windows with significant conditional differences.



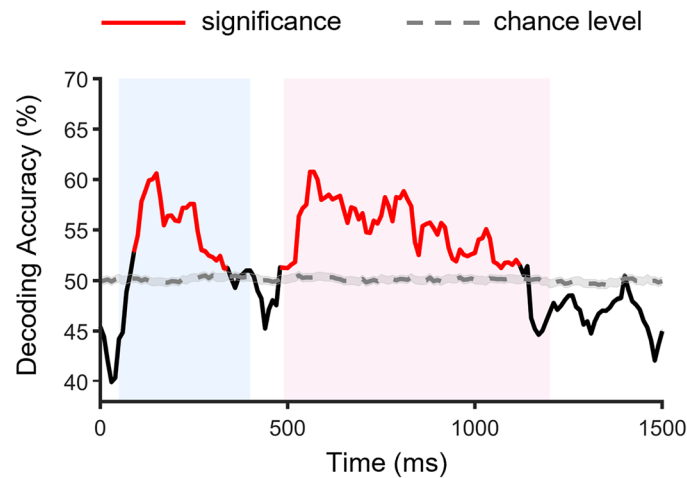
Extended Data Fig. 3 | Validation of MC-increased amygdala IBRA after controlling for timing-related confounds. **a**, Time courses of amygdala IBRA during MC (purple) and non-MC (grey) across all trials. MC-specific IBRA increases were observed in two time windows (130–370 ms and 480–1190 ms) after controlling for dDT and dDT_{sim}. Linear mix-effects models were conducted at each time point with condition (MC/non-MC) as a fixed effect and dDT and dDT_{sim} as covariates. Multiple comparisons were corrected through cluster-based permutation testing ($n = 5,000$, two-sided). Colored curves represent mean values across trials with shaded areas indicating 95% CIs. **b**, Time courses of amygdala IBRA during dDT- and dDT_{sim}-matched MC and non-MC conditions. Propensity-score matching was used to create subsets of MC and non-MC trials matched on dDT and dDT_{sim}. MC-specific IBRA increases were observed in two time windows (130–320 ms and 540–1130 ms). **c**, Time courses of response-

locked IBRA of MC and non-MC, from -1500 ms to 500 ms relative to individual response onset. No significant MC-specific IBRA was found. **d**, Time courses of DT-normalized amygdala IBRA during MC and non-MC conditions. Each participant's trial-wise neural time series were temporally normalized according to their decision time. The neural time series between stimulus onset and the participant's response were interpolated into a fixed 150 time-bins (shape-preserving piecewise cubic interpolation) and IBRA was recalculated for each dyad. MC-specific IBRA increases were observed in two time windows (bins 1–39 and 49–104). In **b–d**, colored curves represent mean values across channel-pairs with shaded areas indicating 95% CIs. Multiple comparisons were corrected through cluster-based permutation testing ($n = 10,000$, two-sided). Horizontal solid lines denote significant timepoints. Supplementary Tables 6 for full statistics. dDT, dyadic decision time, dDT_{sim}, dyadic decision-time similarity.



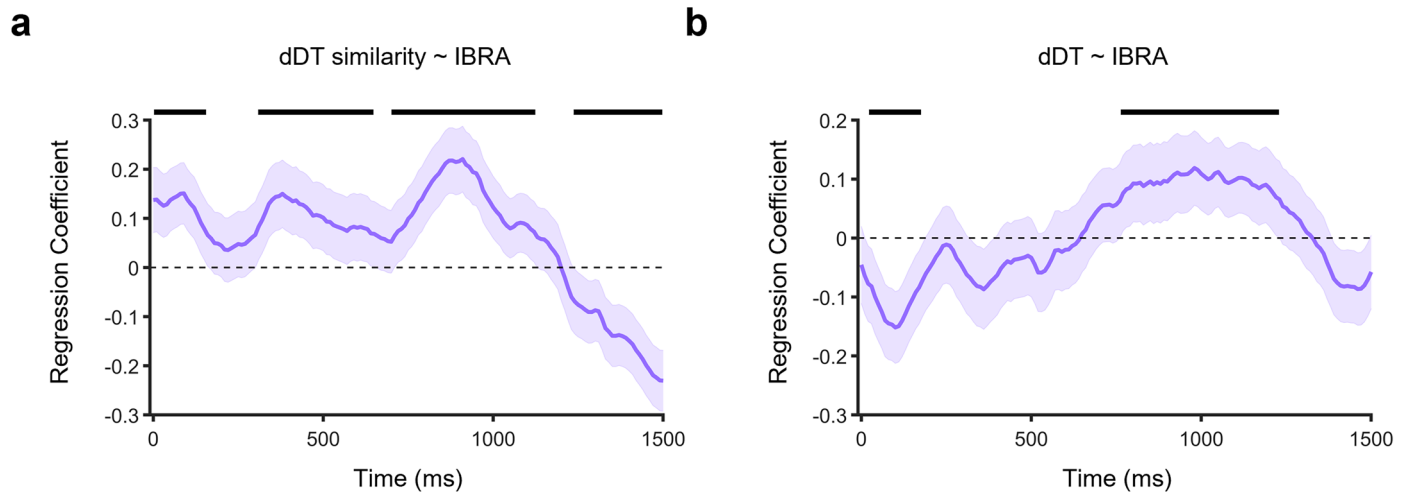
Extended Data Fig. 4 | Interaction-history similarity organized cross-brain neural similarity specifically for MC-MC trial pairs. **a**, Schematic of the cross-brain, cross-trial representational similarity analysis (RSA). For each dyad, a 30×30 cross-brain neural similarity matrix was constructed, in which each cell (i, j) was defined as the Spearman correlation between the spectral pattern of trial i from one participant (S1) and trial j from the partner (S2). The interaction-history similarity matrix was constructed as 1 minus the absolute difference in MC rate between trial pairs. It was then multiplied with a binary MC-MC or non-MC-non-MC pair matrix to generate condition-specific history-similarity matrix, isolating the component of history similarity expressed specifically within trial pairs of MC or non-MC, respectively. Partial correlations were conducted between the cross-brain neural similarity matrix and each condition-specific history-similarity matrix while controlling for the same-trial diagonal component and the overall history- and condition-similarity structure. **b, c, e, f**, Time-window-averaged cross-brain, cross-trial RSA. Interaction-history

similarity was significantly associated with cross-brain neural similarity within MC-MC trial pairs for both early (**b**) and late (**c**) spectra. While, interaction-history similarity was not associated with cross-brain neural similarity within trial pairs of non-MC for either early (**e**) or late (**f**) spectra. **d**, Time-resolved cross-brain, cross-trial RSA. Significant associations between cross-brain neural spectral similarity and interaction-history similarity were observed specifically for MC-MC trial pairs in two time-windows: 270–620 ms ($t_{sum} = 115.298$, $p_{corr} = 0.003$), 770–970 ms ($t_{sum} = 62.579$, $p_{corr} = 0.035$). Multiple comparisons were corrected through cluster-based permutation testing ($n = 10,000$, one-sided). Colored curves represent mean values across channel-pairs and shaded areas indicating 95% CIs. Horizontal solid lines denote significant timepoints. In **b, c, e, f**, statistical significance was assessed using one-sided one sample t -tests. Box plots show medians (center lines), interquartile ranges (IQR, box edges), whiskers ($1.5 \times IQR$), and mean values (crosses). Supplementary Table 5 for full statistics. CI: confident interval. $**p < 0.01$, n.s., not significant.



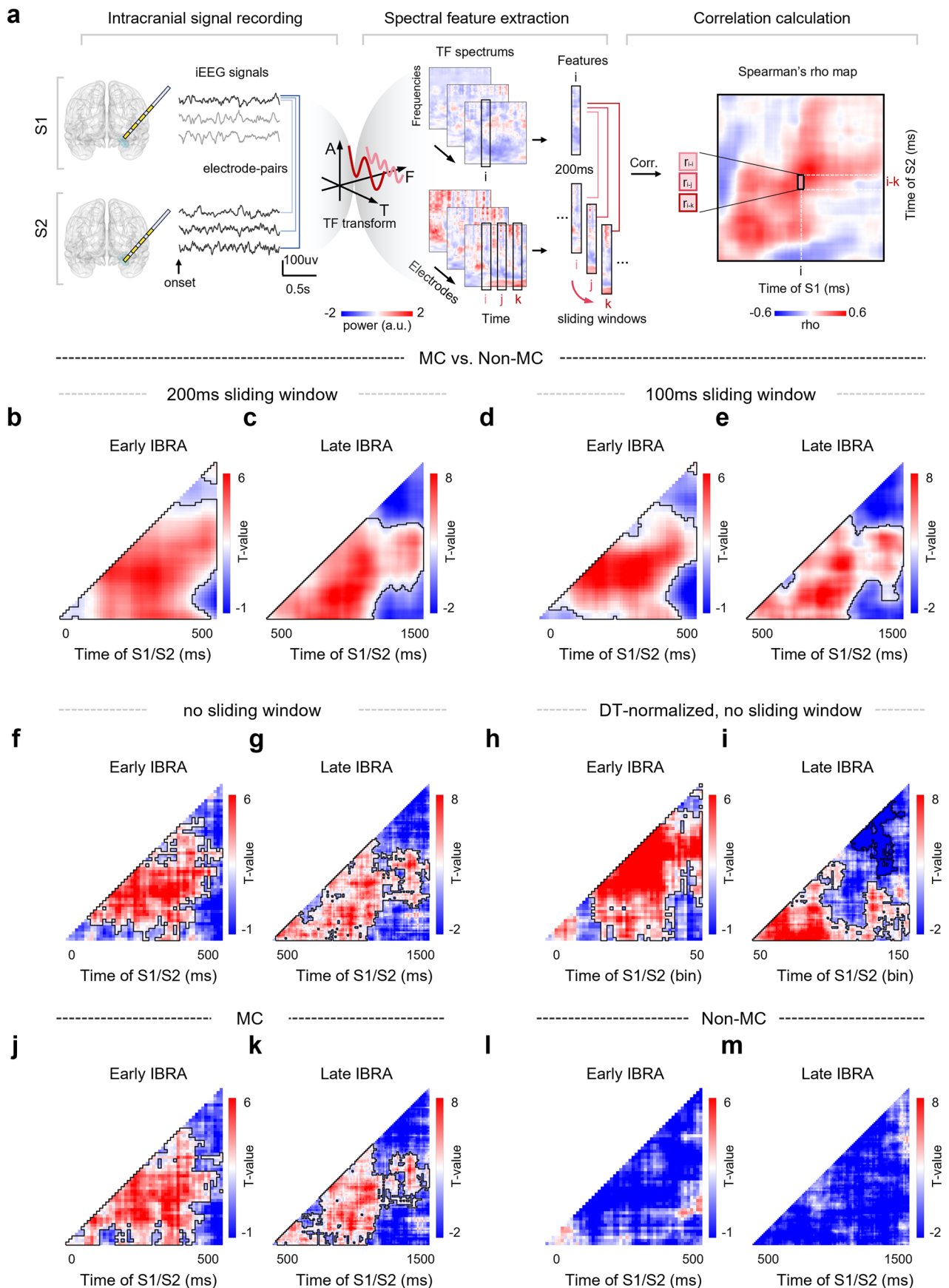
Extended Data Fig. 5 | Trial-level prediction of mutual cooperation by amygdala IBRA. Time-resolved decoding accuracy of trial-wise IBRA values predicting upcoming mutual cooperation outcome (MC/non-MC). For each dyad, IBRA strength was first averaged across all available amygdala channel pairs, yielding a single IBRA time series per trial. Decoding was then performed using a linear discriminant analysis (LDA) classifier and a leave-one-dyad-out cross-validation procedure. In each fold, data from one dyad was used as the testing set and the remaining dyads were used as the training set. The training set was balanced by randomly selecting a number-matched subset of MC and non-MC trials. This procedure was repeated until each dyad served once as the test set. Classification accuracy was computed at each time point and averaged across folds. Decoding was repeated for 500 times while considering

randomization during dataset construction and cross-validation. Statistical significance was assessed using a cluster-based permutation test to compare with the chance level, in which trial labels were randomly shuffled within each dyad (two-sided). Colored curves represent mean values across repetitions with shaded areas indicating 95% CIs. Red and black solid curves indicate time points with significant and non-significant decoding accuracy on test trials, respectively, relative to chance-level performance (gray dotted line). Light blue and red shaded area indicate the early and late MC-specific IBRA windows (Fig. 2b). IBRA in both early (90–350 ms, $t_{\text{sum}} = 695.210$, $p_{\text{corr}} = 2.00 \times 10^{-4}$) and late (490–1130 ms, $t_{\text{sum}} = 1562.742$, $p_{\text{corr}} = 2.00 \times 10^{-4}$) windows predicted upcoming mutual cooperation.



Extended Data Fig. 6 | Behavioral metrics relate to sustained IBRA. a,b, Time-resolved regression coefficients of IBRA in MC trials predicting dyadic decision-time similarity (**a**) and dyadic decision time (**b**). Regression coefficients were derived from linear mixed-effects models. **a**, IBRA in both early and late windows exhibited positive correlations with decision-time similarity between partners (10-170 ms, $t_{sum} = 65.470$, $p_{corr} = 0.023$; 320-660 ms, $t_{sum} = 106.217$, $p_{corr} = 0.003$; 720-1130 ms, $t_{sum} = 177.304$, $p_{corr} = 2.00 \times 10^{-4}$). **b**, Early IBRA showed sustained

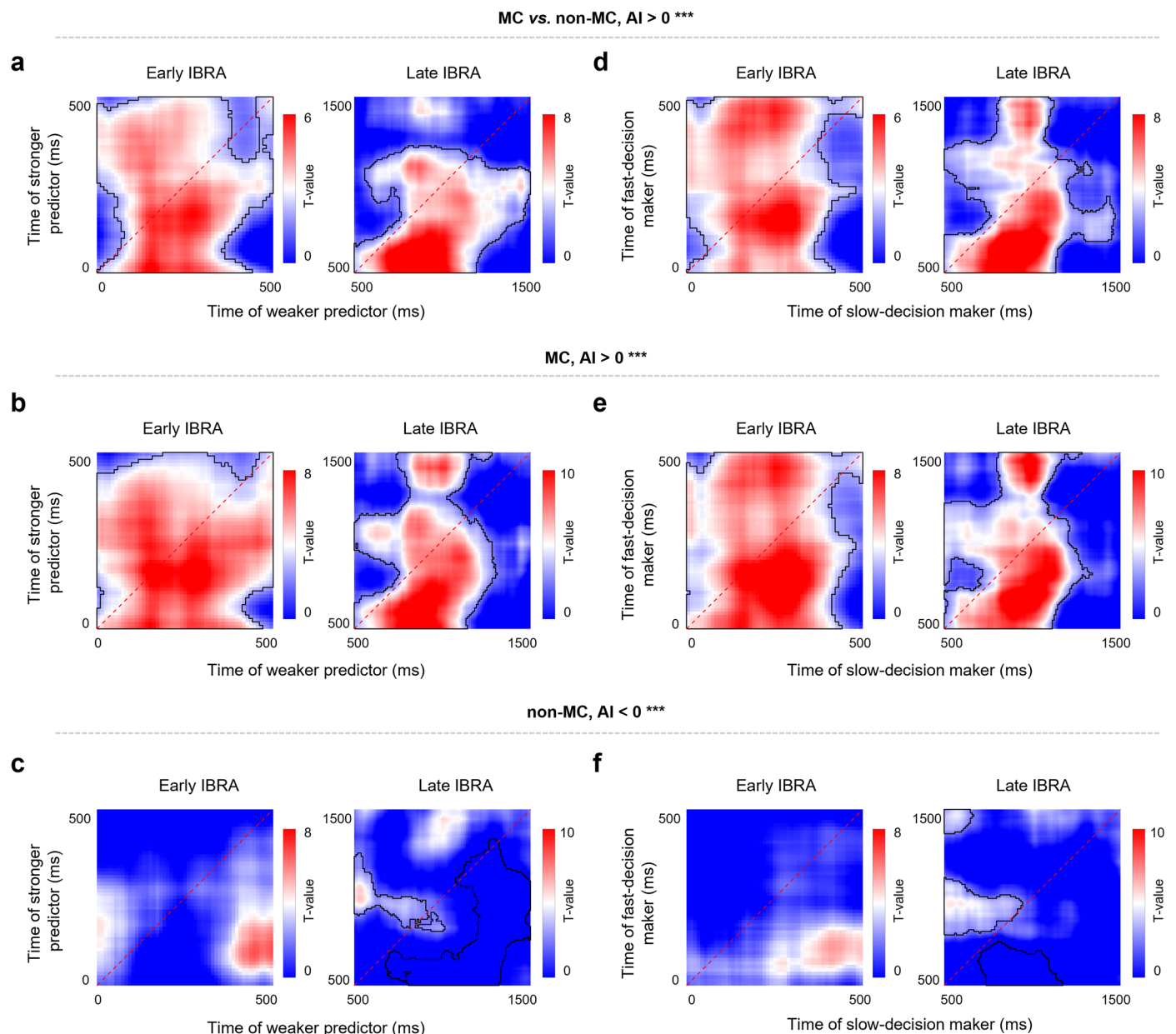
negative correlations with decision time (30-190 ms, $t_{sum} = -62.423$, $p_{corr} = 0.032$), whereas IBRA showed positive correlations with decision time across a later time window (780-1240 ms, $t_{sum} = 140.397$, $p_{corr} = 2.00 \times 10^{-4}$). Multiple comparisons were corrected through cluster-based permutation testing ($n = 5,000$, two-sided). Colored curves represent estimated coefficients with shaded areas indicating standard errors of each coefficient. Horizontal solid lines indicate significant time windows.



Extended Data Fig. 7 | See next page for caption.

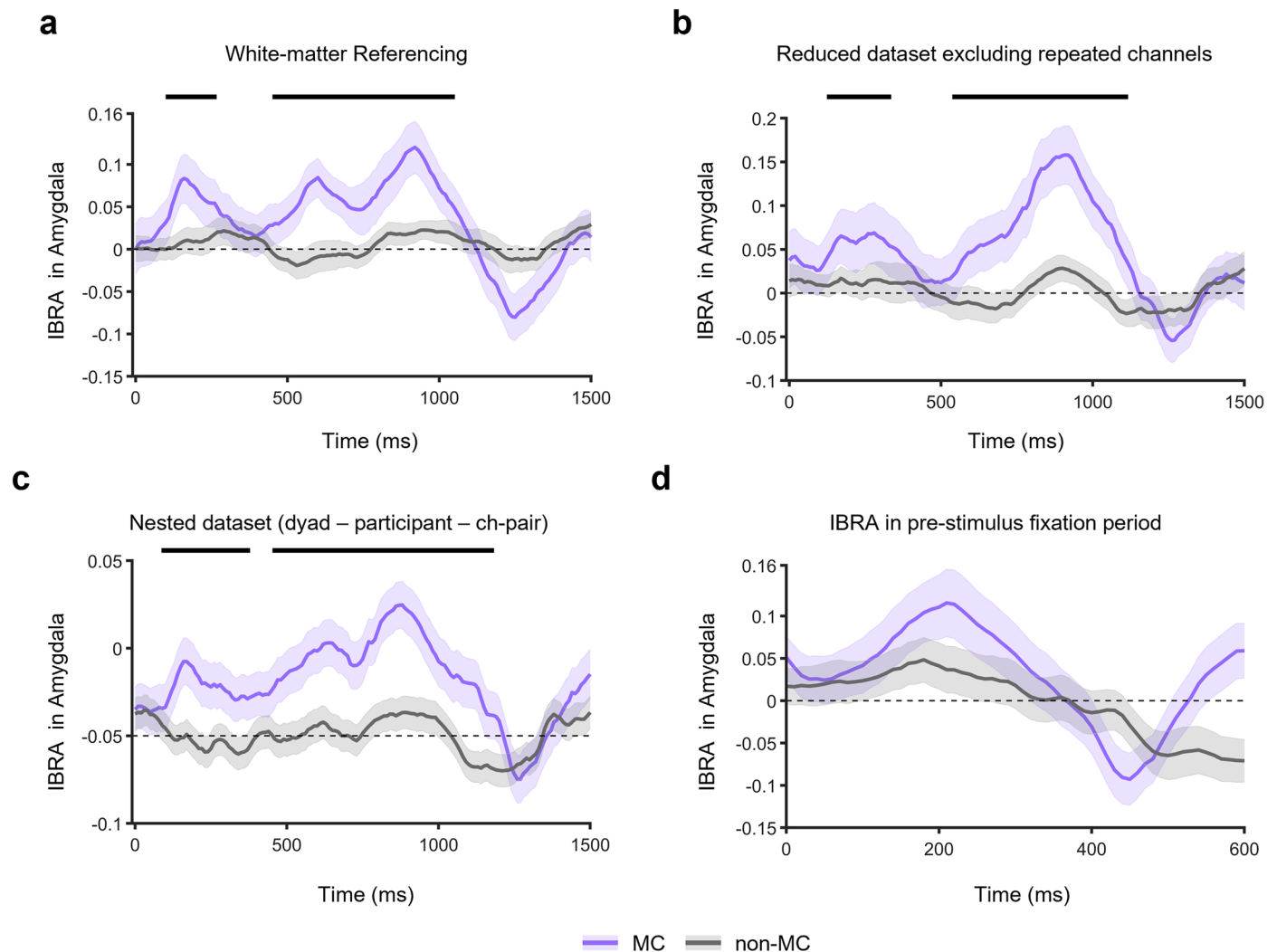
Extended Data Fig. 7 | Temporally diffuse alignment between inter-brain neural spectra. a, Schematic of time-lagged IBRA analysis. For each dyad, simultaneous iEEG signals from same-region electrodes were transformed into time-frequency spectra (4–200 Hz). Spectral power across all frequencies was extracted using sliding windows (200-ms/100-ms, 10-ms steps; or no smoothing) and represented as feature vectors. Trial-wise Fisher- z -transformed Spearman correlations between one channel's spectral patterns across all time-windows and those of another channel yielded a time-by-time IBRA map for each channel-pair. Example window i from one patient (S1) and temporally shifted windows $i-k$ from another patient (S2) are illustrated. **b–g**, Time-by-time t -maps (paired t -tests, two-sided) showing amygdala IBRA increases (MC *vs.* non-MC) for early (**b,d,f**) and late (**c,e,g**) windows using 200-ms (**b,c**) and 100-ms (**d,e**) sliding window and no smoothing (**f,g**) analyses. In both early and late windows, IBRA increases extended from the diagonal to off-diagonal regions, indicating

temporally diffuse alignment between inter-brain neural spectra. **h,i**, Time-by-time t -maps (paired t -tests, two-sided) showing amygdala IBRA increases for early (**h**) and late (**i**) windows with decision-time normalization. For DT-normalized IBRA, each participant's trial-wise neural time series was temporally normalized according to decision time (details in Extended Data Fig. 3), and IBRA was recalculated for each dyad. In both early and late windows, IBRA increases extended from the diagonal to off-diagonal regions, indicating robust temporally diffuse alignment between inter-brain neural spectra, even without temporal smoothing or after controlling for individual DT differences. **j–m**, Time-by-time t -maps (one-sample t -tests, one-sided) showing amygdala IBRA in MC (early (**j**) and late (**k**)) and non-MC (early (**l**) and late (**m**)) condition without smoothing. Diffuse IBRA increases extending from the diagonal to off-diagonal regions were specific for MC rather than non-MC condition. Black contours delimit significant clusters after cluster-based permutation tests ($n = 5,000$).



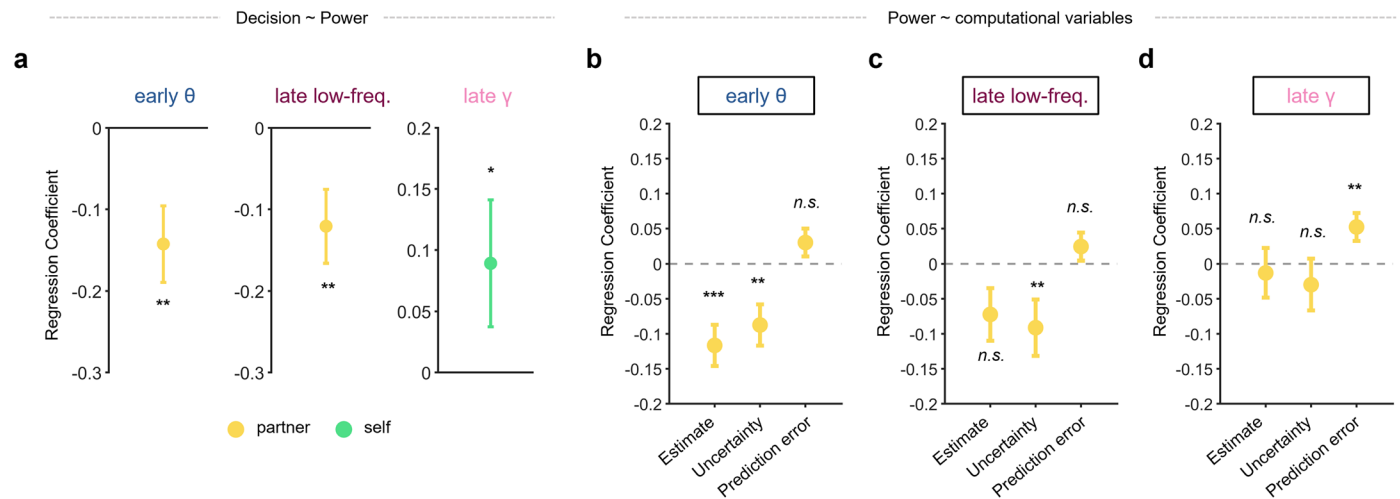
Extended Data Fig. 8 | Directionality in IBRA was linked to partner-predictive signal strength and decision speed. a–c, Time-by-time IBRA *t*-maps (left: early-window, 0–500 ms, right: late-window, 500–1500 ms) showing that amygdala neural spectra from participants with stronger partner-predictive signals (y axis) preceded those of their relatively weaker predictor partner (x axis). **a,** MC vs. non-MC, $AI = 0.307, p = 1.07 \times 10^{-8}$; **b,** MC, $AI = 0.181, p = 3.02 \times 10^{-4}$; **c,** non-MC, $AI = -0.291, p = 3.36 \times 10^{-6}$. **d–f,** Time-by-time IBRA *t*-maps showing that amygdala neural spectra from trial-wise faster decision-maker (y axis) preceded those from the relatively slower decision-maker (x axis). **d,** MC vs. non-MC, $AI = 0.254,$

$p = 8.96 \times 10^{-7}$; **e,** MC, $AI = 0.220, p = 1.99 \times 10^{-5}$; **f,** non-MC, $AI = -0.384, p = 3.04 \times 10^{-8}$. AI: asymmetry index, defined as $AI = (a - b) / (a + b)$, where *a* and *b* are the average value of significant IBRA in regions below and above the diagonal, respectively. A positive AI indicates neural spectra from the y-axis participant preceded those from the x-axis participant, whereas a negative AI indicates the opposite. Statistical significance was examined using Wilcoxon signed-rank tests. Black contours delimit significant clusters after cluster-based permutation tests ($n = 5,000$, two-sided). Red dashed lines indicate the diagonals. *** $p < 0.001$.



Extended Data Fig. 9 | MC-specific amygdala IBRA was validated through alternative referencing, control for repeated channels, linear mixed-effects modeling, and baseline checks. a, Time courses of amygdala IBRA during MC and non-MC condition using white-matter re-referencing. For each recording channel, neural signals were re-referenced to the average activity of all clean white-matter channels. IBRA was re-computed and compared between conditions. MC-specific IBRA increases were observed in two time windows: 110–270 ms ($t_{\text{sum}} = 60.580$, $p_{\text{corr}} = 0.037$) and 460–1060 ms ($t_{\text{sum}} = 274.751$, $p_{\text{corr}} = 1.00 \times 10^{-4}$). **b,** Time courses of amygdala IBRA during MC and non-MC conditions in the reduced dataset excluding repeated channels. For channels that formed multiple inter-dyad channel-pairs, only one pair was randomly retained and the remaining pairs were discarded. The condition difference of IBRA was re-examined in this reduced dataset. MC-specific IBRA increases were observed in two time windows: 140–350 ms ($t_{\text{sum}} = 56.665$, $p_{\text{corr}} = 0.047$) and 560–1140 ms ($t_{\text{sum}} = 308.922$, $p_{\text{corr}} = 1.00 \times 10^{-4}$). **c,** Time courses of amygdala IBRA

during MC and non-MC conditions across all participants, channel-pairs, and trials. Condition differences were examined using linear mix-effects models with dyad, participant, and channel-pair as nested random effects. MC-specific IBRA increases were also observed in two time windows: 100–390 ms ($t_{\text{sum}} = 113.050$, $p_{\text{corr}} = 0.004$) and 470–1200 ms ($t_{\text{sum}} = 425.827$, $p_{\text{corr}} = 2.00 \times 10^{-4}$). **d,** Time courses of amygdala IBRA during the pre-stimulus fixation period (minimal length: 600 ms) for MC and non-MC conditions. No significant MC-specific IBRA was found during fixation. In **a, b, d**, colored curves represent mean values across channel-pairs with shaded areas indicating 95% CIs. Multiple comparisons were corrected through cluster-based permutation testing ($n = 10,000$, two-sided). In **c**, colored curves represent mean values across trials with shaded areas indicating 95% CIs. Multiple comparisons were corrected through cluster-based permutation testing ($n = 5,000$, two-sided). Horizontal lines indicate significant time windows.



Extended Data Table 10 | Validation of prospective partner-estimation analyses in a larger sample ($n_{\text{sub}} = 27$). **a**, Amygdala low-frequency power consistently predicted the partner's upcoming cooperation, and late gamma power predicted the participant's own decision (early theta: $\beta = -0.143$, $t_{2658} = -3.049$, $p = 0.001$, 95% CI: -Inf, -0.066; late low-frequency: $\beta = -0.121$, $t_{2658} = -2.667$, $p = 0.004$, 95% CI: -Inf, -0.046; late gamma: $\beta = 0.089$, $t_{2658} = 1.722$, $p = 0.043$, 95% CI: 0.004, Inf). Regression coefficients were derived from generalized linear mixed-effects models (one-sided). **b-d**, Amygdala spectrotemporal features consistently encoded model-derived variables. **b**, Early theta suppression was associated the prospective partner-cooperation estimate

and its uncertainty (estimate: $\beta = -0.117$, $t_{2657} = -3.960$, $p = 7.69 \times 10^{-5}$, 95% CI: -0.174, -0.059; uncertainty: $\beta = -0.087$, $t_{2657} = -2.963$, $p = 0.003$, 95% CI: -0.145, -0.030). **c**, Late low-frequency power was negatively associated with estimate uncertainty ($\beta = -0.092$, $t_{2657} = -2.271$, $p = 0.023$, 95% CI: -0.170, -0.012). **d**, Late gamma power was positively correlated with retrospective cooperative prediction error ($\beta = 0.052$, $t_{2657} = 2.612$, $p = 0.009$, 95% CI: 0.013, 0.092). Regression coefficients were derived from linear mixed-effects models (two-sided). Central dots and corresponding error bars indicate estimated coefficients and standard errors, respectively. *** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$, *n.s.*, not significant.

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Software and code

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Data collection	Behavioral data were collected using Psychophysics Toolbox Version 3 (PTB-3) extension in MATLAB R2016a (MathWorks). iEEG data were collected using Nicolet EEG system (Alliance Biomedica Pvt. Ltd., India) and Nihon-Kohden system (Nihon Kohden Corporation Japan).
Data analysis	The data analysis of this manuscript was performed on the Matlab (R2020b, Mathworks). Analysis of (generalized) linear mixed-effect models were performed on Statistics and Machine Learning Toolbox, implemented in Matlab (R2020b, Mathworks). Imaging data were processed using FieldTrip (v20220310), Freesurfer (v5.3.0), Statistical Parametric Mapping (SPM12) and visualized using BrainNet Viewer (v1.7). Code for preprocessing is available at https://github.com/fieldtrip/fieldtrip .

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The raw MRI and iEEG data that contain personal information of the patients are protected and with restricted access for ethical and privacy reasons. Access can be obtained from the corresponding author upon request. Preprocessed data needed to generate the figures, and over which statistics were computed, are available in the following repository: <https://osf.io/gy829>.

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Reporting on sex and gender

Gender was not considered as a variable of interest in the experiment design due to limited sample size. Our dataset was not severely biased regarding patient gender (36 males out of 56 participants, 64%), and our results showed consistent patterns when controlling individual or dyad identities, indicating the findings are applicable to both genders.

Reporting on race, ethnicity, or other socially relevant groupings

No socially constructed or relevant grouping variable was used and reported in the manuscript.

Population characteristics

All participants (N=56, 36 males, age = 24.9±9.3 years old, demographic details in Supplementary Table 1) were implanted with depth electrodes for iEEG recordings aimed at identifying epileptic foci. The electrode placements were determined exclusively by clinical needs, with no electrodes implanted for research purposes. Dyads were formed opportunistically from inpatient patients who were overlapping in their hospital stay and had electrodes implanted in the amygdala or TPJ outside of the epileptogenic zone.

Recruitment

Dyads were recruited based on the following criteria: i) at least one electrode implanted in the amygdala or TPJ; ii) seizure-free for ≥3 hrs before experimental testing; iii) no history of major psychiatric disorders, head trauma, or encephalitis; and iv) ability to perform computerized decision-making tasks. Prior to their involvement, all participants provided informed consent after a full explanation of study procedures and were informed of their right to withdraw at any time without affecting their clinical care.

Ethics oversight

The experimental protocols were in accordance to the latest version of the Declaration of Helsinki and received approval from local Institutional Review Board at each hospital (i.e., Chinese PLA General Hospital, S202139402 and Beijing Tiantan Hospital, KY 202008002; KY 2023-034-02).

Note that full information on the approval of the study protocol must also be provided in the manuscript.

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Life sciences study design

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Sample size

Similar to the majority of iEEG studies, we did not conduct a prior sample size estimation. However, our sample sizes (the number of dyads, patients, clean channels, and channel-pairs) were consistent with or larger than those reported in previous iEEG studies. Specifically, for individual-level analyses, numbers of clean channels (CC dyads: 209 TPJ and 102 amygdala channels; pMC dyads: 106 amygdala channels; Supplementary Table 1) were comparable to or larger than those reported in other iEEG publications. The numbers of dyads and channel-pairs used in our analysis (Supplementary Table 1) also aligned with previous studies examining inter-brain correlations, including animal models and human hyper-iEEG studies. Our main findings were statistically robust and survived multiple comparison corrections.

Data exclusions

1. Participants: two dyads were excluded from analysis because they failed to complete the task and never achieved mutual cooperation.
2. Behavioral: for any analysis, which involves the interaction history (i.e., past mutual cooperation rate, past individual cooperation rate, past punishment rate), the first trial was excluded as it has no history.
2. Neural: first, the channels were identified as "bad" and excluded based on the following criteria: 1) located within the epileptic zone or severely contaminated by epileptic activity; 2) manually labeled as clear outliers on power spectra plots of all channels; 3) exhibiting a variance greater or less than five times the median variance across all channels; or 4) containing more than three times the median number of spikes (defined as 100 μ V changes between consecutive samples) across all channels. Second, the epoques containing IEDs were excluded

based on the following criteria: 1) the envelope of the unfiltered signal exceeded five standard deviations (s.d.) above baseline; or 2) the envelope of the filtered signal (15-80 Hz bandpass filter) exceeded six s.d. above baseline.

Replication

1. To verify the productivity of experimental findings in inter-brain representational alignment, we replicated our results after controlling for the variability across dyads and channel-pairs using linear mixed-effects models and also at the dyad level (Extended Data Fig. 2, Supplementary Table 5). We also replicated our results after controlling for timing-related confounds (Extended Data Fig. 3, Supplementary Table 6). 2. To verify the productivity of experimental findings in social prospective simulation, we replicated our results in a larger sample, including another 11 dyad in which one patient had Amygdala recordings. The experimental findings were fully replicated in this expanded sample (Extended Data Fig. 10).

Randomization

Experimental conditions and groups were based on participants' responses. Therefore, randomization of experimental conditions or stimulus presentation were not relevant in the current study.

Blinding

Experimental conditions and groups were based on participants' responses. Therefore, blinding of experimental conditions was not relevant during data collection.

Behavioural & social sciences study design

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Study description

Research sample

Sampling strategy

Data collection

Timing

Data exclusions

Non-participation

Randomization

Ecological, evolutionary & environmental sciences study design

All studies must disclose on these points even when the disclosure is negative.

Study description

Research sample

Sampling strategy

Data collection

Timing and spatial scale

Data exclusions

Reproducibility

Randomization

Blinding

Did the study involve field work? ☐ Yes ☐ No

Field work, collection and transport

Field conditions

Location	
Access & import/export	
Disturbance	

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Materials & experimental systems

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☒	☐ Eukaryotic cell lines
☒	☐ Palaeontology and archaeology
☒	☐ Animals and other organisms
☒	☐ Clinical data
☒	☐ Dual use research of concern
☒	☐ Plants

Methods

n/a	Involved in the study
☒	☐ ChIP-seq
☒	☐ Flow cytometry
☒	☐ MRI-based neuroimaging

Antibodies

Antibodies used	
Validation	

Eukaryotic cell lines

Policy information about [cell lines and Sex and Gender in Research](#)

Cell line source(s)	
Authentication	
Mycoplasma contamination	
Commonly misidentified lines (See ICLAC register)	

Palaeontology and Archaeology

Specimen provenance	
Specimen deposition	
Dating methods	
☐ Tick this box to confirm that the raw and calibrated dates are available in the paper or in Supplementary Information.	
Ethics oversight	

Note that full information on the approval of the study protocol must also be provided in the manuscript.

Animals and other research organisms

Policy information about [studies involving animals](#); [ARRIVE guidelines](#) recommended for reporting animal research, and [Sex and Gender in Research](#)

Laboratory animals	
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Wild animals

Reporting on sex

Field-collected samples

Ethics oversight

Note that full information on the approval of the study protocol must also be provided in the manuscript.

Clinical data

Policy information about [clinical studies](#)

All manuscripts should comply with the ICMJE [guidelines for publication of clinical research](#) and a completed [CONSORT checklist](#) must be included with all submissions.

Clinical trial registration

Study protocol

Data collection

Outcomes

Dual use research of concern

Policy information about [dual use research of concern](#)

Hazards

Could the accidental, deliberate or reckless misuse of agents or technologies generated in the work, or the application of information presented in the manuscript, pose a threat to:

No

Yes

☐☐

Public health

☐☐

National security

☐☐

Crops and/or livestock

☐☐

Ecosystems

☐☐

Any other significant area

Experiments of concern

Does the work involve any of these experiments of concern:

No

Yes

☐☐

Demonstrate how to render a vaccine ineffective

☐☐

Confer resistance to therapeutically useful antibiotics or antiviral agents

☐☐

Enhance the virulence of a pathogen or render a nonpathogen virulent

☐☐

Increase transmissibility of a pathogen

☐☐

Alter the host range of a pathogen

☐☐

Enable evasion of diagnostic/detection modalities

☐☐

Enable the weaponization of a biological agent or toxin

☐☐

Any other potentially harmful combination of experiments and agents

Plants

Seed stocks

Novel plant genotypes

Authentication

ChIP-seq

Data deposition

- ☐ Confirm that both raw and final processed data have been deposited in a public database such as [GEO](#).
- ☐ Confirm that you have deposited or provided access to graph files (e.g. BED files) for the called peaks.

Data access links

May remain private before publication.

Files in database submission

Genome browser session

(e.g. [UCSC](#))

Methodology

Replicates

Sequencing depth

Antibodies

Peak calling parameters

Data quality

Software

Flow Cytometry

Plots

Confirm that:

- ☐ The axis labels state the marker and fluorochrome used (e.g. CD4-FITC).
- ☐ The axis scales are clearly visible. Include numbers along axes only for bottom left plot of group (a 'group' is an analysis of identical markers).
- ☐ All plots are contour plots with outliers or pseudocolor plots.
- ☐ A numerical value for number of cells or percentage (with statistics) is provided.

Methodology

Sample preparation

Instrument

Software

Cell population abundance

Gating strategy

- ☐ Tick this box to confirm that a figure exemplifying the gating strategy is provided in the Supplementary Information.

Magnetic resonance imaging

Experimental design

Design type

Electrode localization without experiment design. Participants did not perform any experimental task during MRI scanning.

Design specifications

Not applicable

Behavioral performance measures

Not applicable

Acquisition

Imaging type(s)	structural T1-weighted
Field strength	3-T
Sequence & imaging parameters	Pre-implant structural T1-weighted scans were obtained using magnetization-prepared rapid gradient echo sequence with standard parameter settings.
Area of acquisition	whole brain scan
Diffusion MRI	☐ Used ☒ Not used

Preprocessing

Preprocessing software	Structural image were preprocessed using FieldTrip (v20220310) for alignment of ACPC (Anterior Commissure - Posterior Commissure) coordinate system and then reconstructed using the 'recon-all' function in Freesurfer (v5.3.0).
Normalization	Structural image was normalized to using SPM12 to obtain deformation parameters that transform electrode positions from patients' native space to standard MNI space
Normalization template	Default tissue probability map in SPM12.
Noise and artifact removal	Not applicable
Volume censoring	Not applicable

Statistical modeling & inference

Model type and settings	Not applicable
Effect(s) tested	Not applicable
Specify type of analysis:	☒ Whole brain ☐ ROI-based ☐ Both
Statistic type for inference	Not applicable
(See Eklund et al. 2016)	
Correction	Not applicable

Models & analysis

n/a	Involved in the study
☒	☐ Functional and/or effective connectivity
☒	☐ Graph analysis
☒	☐ Multivariate modeling or predictive analysis
Functional and/or effective connectivity	
Graph analysis	
Multivariate modeling and predictive analysis	