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Supplementary Information for

Increasing and decreasing inter-regional brain coupling increases and decreases oscillatory activity in the human brain

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Supplementary Information Text

Supplementary Experimental Procedures

A preliminary experiment was conducted with the aim of ensuring the effectiveness of the TMS protocol in the context of the current behavioural task. In this experiment we first checked that PMv TMS did indeed exert a causal influence over M1 by examining the impact of activity induced in PMv over activity induced in M1 on the Go trials of the Go/No-Go task. We did this by recording motor evoked potential (MEP) amplitudes in hand muscles when TMS pulses were applied to M1. We focus on two analyses. In both cases, the MEPs provided our dependent measure. This approach is similar to that used in previous studies of cortico-cortical paired associative stimulation (ccPAS) [1-3]

First, we compared MEPs when we applied either single-pulse TMS (spTMS) over right M1 [4, 5] or paired-pulse TMS (ppTMS) over right PMv (conditioning pulse) followed by right M1 [1, 2, 6, 7]. We recorded MEPs from the left first dorsal interosseus (FDI) muscle while participants performed Go trials in the Go/No-Go task. If the application of the conditioning pulse to PMv affects M1 then MEP amplitudes recorded from hand muscles after M1 TMS should be different when a prior pulse of TMS is applied to PMv. In total, we compared the sizes of MEPs on 36 spTMS Go trials and 36 ppTMS Go trials. The pulse applied to M1 always occurred 125ms after the Go cue stimulus onset. This stimulation time was chosen based on a pilot study (N=10) showing a greater the state-dependent (Go vs No-Go) influence of PMv on M1 in ppTMS trials at 125ms (in contrast to 100, 150, 175ms), and it accords with the early engagement of motor and pre-motor areas in action control shown in previous studies [4, 5]. Both spTMS and ppTMS trials were administered in alternation with no-TMS trials (324 trials per block) and in the context of No-Go trials. Trial presentation order was pseudorandomised within the same block of trials to ensure that there were no more than five consecutive TMS trials to avoid TMS after-effects.

Second, as in Experiments 1A and 1B, we examined the impact of repeatedly inducing PMv activity either just before or just after inducing M1 activity during ccPAS. Again, we did this by measuring MEPs recorded in response to single pulses of M1 TMS on Go trials, but we did so before and after a 15-minute period of ccPAS. In one session, we applied 15 minutes of ccPAS over PMv followed by M1 (PMv-M1-ccPAS) at 0.1 Hz (90 total stimulus pairings). In another session the order of the ccPAS stimulation was reversed i.e. applying the first paired pulse over M1 and the second pulse over PMv (Fig. S5). Exactly the same number of pulses were applied to PMv and M1 in both experimental sessions and the same combination of 6ms and 8ms IPIs were used.

Participants

12 healthy, right-handed adults participated across the two preliminary experiments (22.91 ± 3.11; 7; 0.88 ± 0.09) (where numbers correspond to mean age ± SD; number of female participants, handiness mean ± SD, as measured by the Edinburgh handedness inventory- adapted from [8]). All subjects participated in both experimental sessions and they were on average, 32.91 ± 22.58 days (mean ± SD) apart. All participants had no personal or familial history of neurological or psychiatric disease, were right handed, gave written informed consent (Medical Science Interdivisional Research Ethics Committee, Oxford RECC, No. R29477/RE004), were screened for adverse reactions to TMS and risk factors by means of a safety questionnaire, and received monetary compensation for their participation. Participants underwent high-resolution, T1-weighted structural MRI scans.

TMS and electromyography recordings

The ccPAS and TMS procedures used in the preliminary Experiments were identical to the procedures described in the main experiments - mean MNI coordinates of the right M1 “cortical hotspot” was at X = 41.37 ± 7.05, Y = -14.74 ± 9.01, Z = 64.71 ± 7.35; mean MNI coordinates of the cerebral location of the PMv stimulation was at X = 58.78 ± 3.13, Y = 18.03 ± 6.96, Z = 16.13 ± 10.77 (Fig 1).

Resting motor threshold (RMT) of the right M1 (mean ± SD, 41 ± 4.58% stimulator output) was determined as described previously [9]. Specifically, PMv TMS was proportional to RMT - 110% (45 ± 4.91); M1 stimulation intensity during experiments was set to elicit single-pulse MEPs of ±1 mV (45.08 ± 4.92% stimulator output). Left FDI electromyography (EMG) activity was recorded with bipolar surface Ag-AgCl electrode montages. Motor evoked potentials (MEPs) were computed as the peak-to-peak amplitude difference – see Materials and Methods in the main text for details.

As the distribution of MEP amplitudes was positively skewed, they were log-transformed for further statistical analyses. First, following previous observations showing greater MEP amplitudes when M1 TMS was preceded by a previous conditioning pulse over PMv in movement trials [1, 2, 5-7, 10], we contrasted MEP amplitudes in spTMS *vs* ppTMS in Go trials at baseline across both preliminary experiments using a t-test (Fig. S5b). Second, we compared MEPs on spTMS trials before and after the two forms of ccPAS (PMv-M1; M1-PMv; Fig. S5c) using a repeated-measures analysis of variance (ANOVA) with the factors of block (Baseline, Expression), trial type (Go, No-Go) and ccPAS order (PMv–M1-ccPAS, M1–PMv-ccPAS). As in previous studies we now focused on spTMS Go trials in order to measure neural excitability in M1 during action performance. This measure should, however, reflect the interactions of both PMv and M1 because these occur under normal circumstances during such movements. Thus, this analysis allowed us to look at an M1 read-out of the PMv-M1 interactions that occur during movement before and after the two types of ccPAS. To correct for non-sphericity, Greenhouse-Geisser corrected results are reported.

Behavioural analysis
Behavioural performance measures comprised median RTs (excluding trials with RT +/- 2SD from the mean) and accuracy (excluding omission errors in go trials and commission errors to No-Go trials). We tested the effect of the ccPAS protocol on RTs and accuracy measures. The analyses focused on data from no-TMS trials to avoid bias from the TMS impact on the hand muscles controlling the button press [11]. A repeated-measures analysis of variance (ANOVA) using the within-subject factors of block (Baseline, Expression), trial type (Go, No-Go) and ccPAS order (PMv–M1-ccPAS, M1–PMv-ccPAS) was used to analyse the behavioural data of the preliminary experiments.

Supplementary Results

MEP results
First, we compared MEPs when we applied either single-pulse TMS (spTMS) over right M1 [4, 5] or paired-pulse TMS (ppTMS) over right PMv (conditioning pulse) followed by right M1 [1, 2, 6, 7] recorded before ccPAS. Inducing activity in PMv just prior to inducing activity in M1 resulted in bigger MEPs. MEPs were larger when M1 TMS was preceded by a conditioning pulse over PMv on Go trials (Fig. S5.B, light *vs* dark blue bars) in the initial baseline testing periods prior to the two different forms ccPAS ($t(11)=-2.235$, $p=0.047$, $d=0.649$).

Second, to examine potentiation of physiological connectivity in the PMv-M1 corticocortical pathway [1, 2], we contrasted the neural excitability of M1 in the context of the PMv-M1 interactions that naturally occur during the course of movement initiation. We did this by focusing on MEPs recorded on Go spTMS trials during the Go/No-Go task before and after each type of ccPAS. The analysis of MEP amplitudes on spTMS trials revealed a significant interaction between block (Baseline, Expression) and ccPAS order (PMv–M1-ccPAS, M1–PMv -ccPAS) $F(11)=5.893$; $p=0.034$; $\eta p2 =.349$. Subsequent analysis demonstrated that the interaction was due to the impact that PMv–M1-ccPAS *vs* M1–PMv-ccPAS had on MEP amplitudes. When contrasting the MEPs recorded before and after PMv-M1 ccPAS, we found that the MEP sizes were significantly different, and that the MEP changes occurred on Go trials ($F(11)=6.489$; $p=0.027$; $\eta p2 =.371$). Specifically, when comparing the MEPs recorded for each trial type in Expression and Baseline blocks, there was a significant increase in MEP size only in Go trials ($t(11)=2.982$, $p=0.012$) after ccPAS (No-Go trials - $t(11)=1.159$,$p=0.271$). No changes were seen in the M1–PMv-ccPAS condition ($F(11)=0.378$; $p=0.551$; $\eta p2 =.033$).

In summary, these results demonstrate two types of influence exerted by PMv over M1 in the context of the Go/No-Go task. First, PMv exerted a transient influence on M1 each time a pulse was applied to PMv. At Baseline, conditioning pulses of PMv enhanced the MEPs induced by M1 TMS on Go trials when neurophysiological studies show that PMv neural activity is inducing changes in M1 activity [6, 7, 10, 12, 13] (Fig. S5.B). Second, PMv-M1 ccPAS in the PMv–M1-ccPAS condition caused sustained changes in activity in M1 whenever a movement was subsequently made (when PMv exerts an influence over M1 [6, 7, 10, 12, 13]) (Fig. S5.C). The sustained change was present on Go trials in contrast to No-Go trials. There was no evidence of a similar sustained change in M1 when an equal number of TMS pulses were applied to M1 and PMv but in the opposite order during M1-PMv ccPAS. Overall, these results show that augmenting cortical connectivity between PMv and M1, by evoking synchronous pre- and postsynaptic activity in the PMv-M1 pathway, leads to modulation of the causal influence of PMv over M1. These findings demonstrate that PMv-M1 ccPAS can be used to

manipulate interactions and coupling between PMv and M1 and therefore PMv-M1 ccPAS is an appropriate tool for examining whether perturbing brain connectivity between brain areas induces changes in oscillatory activity.

We note that in theory it might also have been possible to measure MEPs not while participants were moving but while they were at rest. When MEPs are measured after PMv-M1 ppTMS is applied at rest then the MEPs are smaller than after M1 spTMS. Moreover, this inhibitory effect is augmented after ccPAS; this was reported by Buch et al [2] in their figure 3 and by Chiappini et al. [14] in their figure 1b. Unfortunately, however, we did not collect MEPs under such rest conditions. We note, however, that this measure is a particularly attractive measure because it provides a very direct and transparent index that the change induced by the ccPAS is indeed in the PMv-M1 pathway. By contrast MEP changes measured in response to M1 spTMS could, in theory, reflect a more general change in cortical excitation as opposed to a more specific change in the PMv-M1 pathway (even though a number of other previous results suggest that indeed the change induced by ccPAS is specific to the PMv-M1 pathway that is stimulated).

We note that MEPs can be measured in response to PMv-M1 ppTMS during movement and that such measurements could be taken before and after the ccPAS. Our experience is that when this procedure is applied during movement, it produces a very consistent and clear result; the MEPs induced by PMv-M1 ppTMS increase in size after ccPAS, but the size of the MEPs induced by PMv-M1 ppTMS is not bigger than the size of the MEPs induced by M1 spTMS (see for example Buch et al. [2] figure 5a). We think that this happens during movement because the PMv-M1 pathway is already being activated *endogenously* because the brain is making a movement and so the PMv-M1 pathway cannot be excited any further by additional stimulation afforded by the PMv-M1 ppTMS. However, because, in our experience this pattern is not immediately intuitive, we have refrained from illustrating it here.

Finally, we note that an additional measure could be taken which is of the PMv-M1 ppTMS effect when a person is actively inhibiting the movement. However, identifying the precise point in time when a person inhibits a movement is difficult and requires examining a number of time points after the “stop” or “change” instruction [10, 12, 15] – i.e. the moment of inhibition is transient. Prior to this moment the PMv may actually be exciting M1. While catching this transient moment of inhibition has been possible, previous studies have not attempted to determine whether this transient moment of inhibition is augmented by ccPAS. We have therefore refrained from examining this issue here.

Behavioural results

The behavioural analysis did not show significant differences in median RT between Baseline and Expression blocks in the preliminary experiment when contrasting PMv-M1 ccPAS *vs* M1-PMv ccPAS (all ps > 0.05). Median RT was faster in the Expression *vs* the Baseline block, irrespectively of the direction of ccPAS ($F(1,11)=10.747$, $p= 0.007$, $\eta^2= .494$), most likely reflecting an effect of practice in the task. Median RTs in the PMv-M1-ccPAS condition: 387.75 ± 36.08, 378.02 ± 31.67; Median RTs in the M1-PMv-ccPAS condition: 389.87 ± 46.72, 375 ± 33.27 (where numbers correspond to mean RT ± SD for Go trial baseline, Go trial expression). Accuracy rates did not show any significant difference between blocks. Accuracy in the PMv-M1-ccPAS condition: 0.95 ± 0.02, 0.94 ± 0.02, 0.93±0.05, 0.91 ± 0.093; Accuracy in the M1-PMv-ccPAS condition: 0.93 ± 0.06, 0.94 ± 0.02, 0.93 ± 0.07, 0.89 ± 0.09 (where numbers correspond to mean accuracy rates ± SD for Go trial baseline, Go trial expression, No-Go trial baseline, No-Go trial expression).

In the main experiment, we found some behavioural evidence that Baseline-Expression effects might differ between the ccPAS protocols used in group A (PMv-M1 ccPAS) and group B (M1-PMv ccPAS) (Tables S1 and S2); there was an interaction between group and block ($F(1,31)=5.38$, $p=0.027$). Further analysis suggested that speeding of RTs was more prominent in group B (M1-PMv ccPAS; $t(16) =2.84$, $p=0.025$) whereas no differences in RT between baseline and expression were seen in the PMv-M1 ccPAS condition ($t(15) = - 1.28$,$p=0.219$). However, it was difficult to be confident in the effect; it disappeared when the data from three participants with outlying RTs were removed. This was also the case when the IPI was included as a covariate in the analysis. When the impact of ccPAS on accuracy was examined in the main experiment, we again found suggestions of a ccPAS effect but again the effect did not appear robust. When the accuracy index was based on both Go and No-Go trials the results indicated a lack of interaction in accuracy ($F(1,31)=2.70$, $p=0.110$) when contrasting groups A and B as a function of block (Baseline *vs* Expression). Further analysis of the performances

of each group separately revealed a trend for a difference between blocks (Baseline and Expression) in group A ($t(15)= 2.117$, $p= 0.051$) and a lack of difference between the Baseline and Expression periods in overall accuracy for group B ($t(15)= 0.227$, $p=0.824$). Moreover, further analysis did not reveal any clearer evidence for differences when Go and No-Go trials were analyzed separately.

EEG recordings and analysis

Visual observation of the Go trials in the PMv-M1 ccPAS group and of the No-Go trials in the group undergoing reverse order M1-PMv ccPAS suggested the possibility of baseline differences in beta and theta power. Specifically, in Go trials in the PMv-M1 ccPAS group (group A) these apparent differences were linked to high beta power in three participants, whereas in No-Go trials the visual differences were associated were associated with high theta power in a different three participants. To ensure that the effects observed in the theta range were not limited to participants with especially high or low theta power we left out the three participants with the lowest theta scores from the PMv-M1 ccPAS group and the three participants with highest theta scores from the M1-PMv ccPAS group in the window of interest (0.15 - 1.2s after stimuli onset). Following the same principle, in a separate analysis we left out the three participants with the highest beta scores from the PMv-M1 ccPAS group and the three participants with lowest beta scores from the M1-PMv ccPAS group in the window of interest (0.7 – 1.2s after stimuli onset). In total we left out six participants with the highest and lowest theta / beta power, for each analysis. We repeated the cluster-based permutation analysis to test differences in the cortical entrained effect in Go and No-Go trials in the beta and theta band, respectively. We observed that the significant differences in Go and No-Go trials between PMv-M1 ccPAS group *vs* M1-PMv ccPAS group remained after removing the participants with extreme values at baseline in both the beta range (Monte Carlo p value =0.001; electrode sites - C4, CZ, FC1, FC2, CP2, FCZ, C1, C2, FC3, FC4, CP4, CPZ; between 0.2 and 1.2s after stimulus onset) and the theta range (Monte Carlo p value =0.002; electrode sites - C4, CZ, FC1, FC2, FCZ, C1, C2, FC3, FC4, CP4, CPZ; between 0.15 and 0.85s after stimulus onset). These results indicate that this interaction was not driven by the group differences in theta or beta power at baseline.

Supplemental Figures

Figure S1

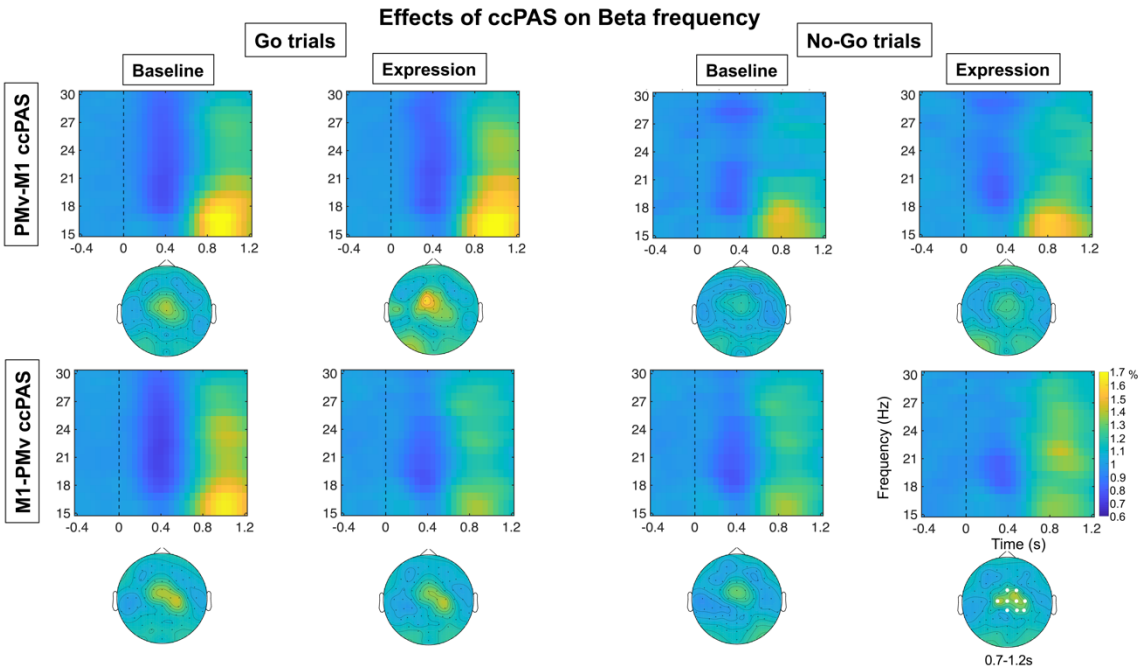

Figure S1: EEG time-frequency responses in the beta band (15-30Hz) in fronto-central and centro-parietal sites (C4, CZ, FC2, CP2, FCZ, C1, C2, FC4, CP4, CPz; electrodes highlighted in white in the bottom right topoplot) time-locked to the onset of the Go/No-Go stimuli, separated for Go and No-Go trials, and Baseline and Expression blocks, for group A (top row) and group B (bottom row) (N=33). All EEG time-frequency responses represent relative percentage change in power with respect to the pre-stimulus interval (1 = no percentage change). The electrodes represented here resulted from the *post-hoc* between-subject t-tests. Further within-subject tests yielded comparable results, including electrodes C4, CZ, FC2, CP2, FCZ, C1, FC4, CP4, CPZ.

Figure S2

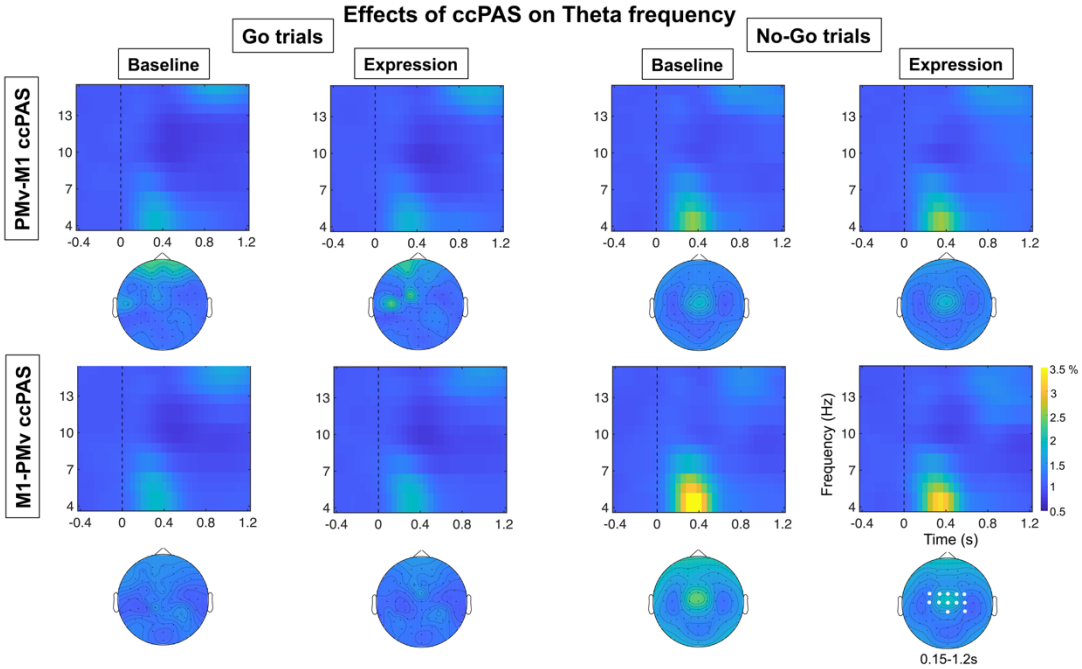

Figure S2: EEG time-frequency responses in the theta and low alpha band (4-15Hz) in fronto-central and centro-parietal sites (C3, C4, CZ, FC1, FC2, FCZ, C1, C2, FC3, FC4, CP4, CPZ electrodes highlighted in white in the bottom right topoplot) time-locked to the onset of the Go/No-Go stimuli, separated for Go and No-Go trials, and Baseline and Expression blocks, for group A (top row) and group B (bottom row) (N=33). All EEG time-frequency responses represent relative percentage change in power with respect to the pre-stimulus interval (1 = no percentage change). The electrodes represented here resulted from the *post-hoc* between-subject t-tests. Further within-subject tests yielded comparable results, including electrodes C4, CZ, FC1, FC2, FCZ, C1, C2, FC3, FC4, CPZ.

Figure S3

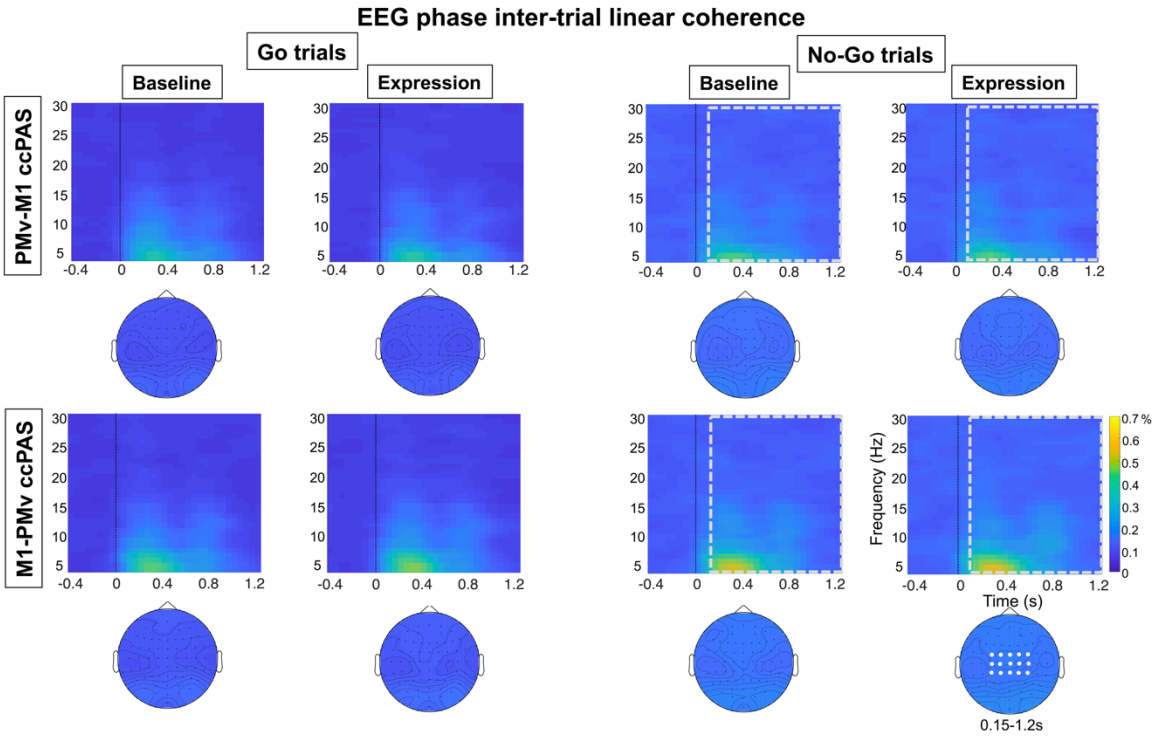

Figure S3: EEG phase inter-trial linear coherence in all bands tested (4-30Hz) in fronto-central and centro-parietal sites (C3, C4, CZ, FC1, FC2, CP1, CP2, FCZ, C1, C2, FC3, FC4, CP3, CP4, CPZ; electrodes highlighted in white in the bottom right topoplot) time-locked to the onset of the Go/No-Go stimuli, separated for Go and No-Go trials, and Baseline and Expression blocks, for group A (top row) and group B (bottom row) (N=33). This result indicates a greater coherence for No-Go trials *vs* Go trials in both Baseline and Expression blocks, irrespectively of ccPAS stimulation order. This main effect can be seen by comparing power in 3-40Hz frequency bands in No-Go (area circumscribed within the dashed grey line in the four right panels) *vs* Go trials. No other main effects or interaction were significant ($p>0.05$), indicating that the ccPAS protocol did not have an effect on phase coherence.

Figure S4

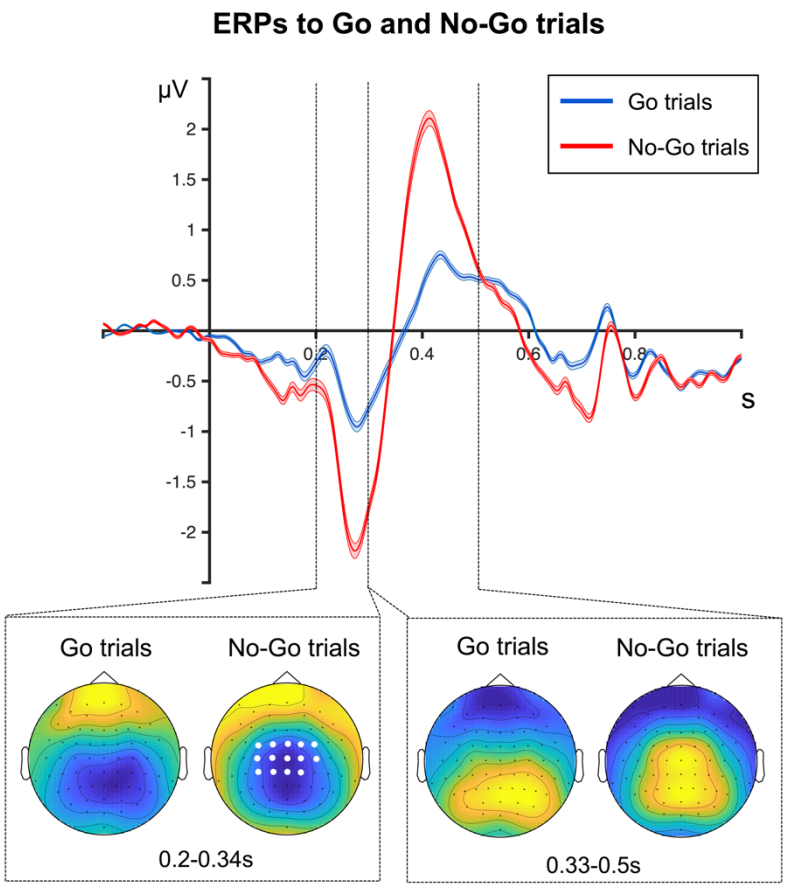

Figure S4: Visual ERPs time locked to the onset of the Go (blue line) and No-Go (red line) stimuli (averaged across electrodes - C3, C4, CZ, FC1, FC2, CP1, CP2, FCZ, C1, C2, FC3, FC4, CP3, CPZ; electrodes highlighted in white in the middle left topoplot), collapsed across the Baseline and the Expression block, and across groups A and B – shaded lines represent standard error for each condition (N=33). Topoplots illustrate ERP activity in the time windows where the main effect of trial type was found. The results of the cluster based premutation analysis on visual ERPs revealed a significant difference between Go *vs* No-Go trials at 0.20 - 0.34s (cluster; p = 0.001; electrode sites - C3, CZ, FC1, FC2, CP1, CP2, FCZ, C1, C2, FC3, CP3, CP4, CPZ), and 0.33 – 0.5s after stimulus onset (cluster; p= 0.001; electrode sites - C3, C4, CZ, FC1, FC2, CP1, CP2, FCZ, C1, C2, FC3, FC4, CP3, CPZ), coinciding with the latency and distribution of the N2 and P3 component. No other main effects or interaction were significant (Monte-Carlo *p* value >0.05), indicating that the ccPAS protocol did not have an effect on visual ERPs.

Figure S5

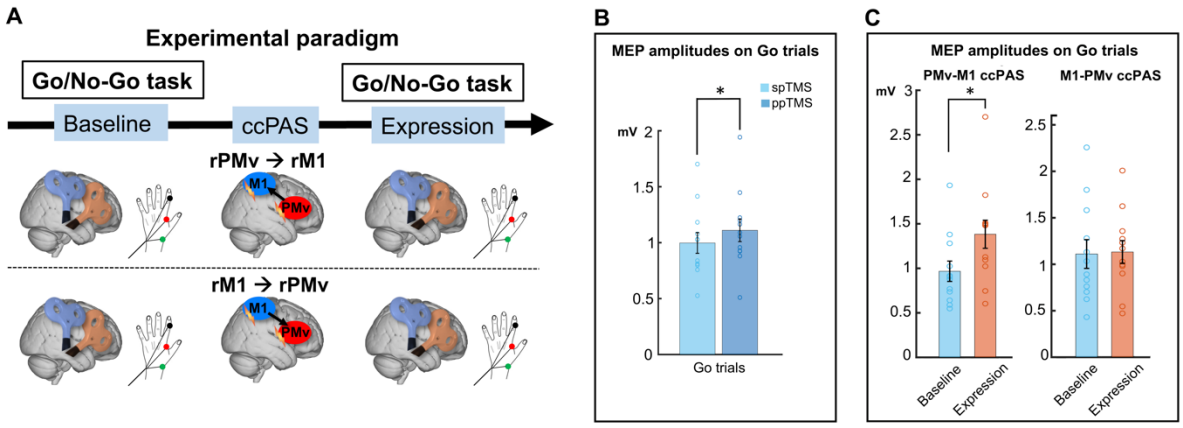

Figure S5: Experimental design and setup for the two preliminary experiments and group mean MEP amplitudes. **A:** Experimental design and setup for the two preliminary experiments (top: preliminary experiment 1; bottom: preliminary experiment 2). The corticocortical repetitive paired associative stimulation (ccPAS) period was preceded (Baseline) and followed (Expression) by Go/No-Go task blocks. TMS-induced motor-evoked potentials (MEP) were recorded from the left first dorsal interosseus (FDI) muscle during the task blocks. **B:** Group mean MEP amplitudes measured with either single pulse (sp)TMS over M1 (light blue) or paired pulse (pp) TMS over PMv/M1 (dark blue), in Go trials, recorded before ccPAS (Baseline) across both preliminary experiments. **C:** Group mean MEP amplitudes measured with spTMS over M1, in Go trials, recorded before (light blue - Baseline) and after (orange - Expression) ccPAS. Error bars represent SEM, single dots represent individual data points (N=12).

Supplemental tables

Table S1
Accuracy (accuracy rates) for Go/No-Go trials in Baseline & Expression blocks (mean ± SD)

		Go trials	No-Go trials
Group A	Baseline block	0.96 ± 0.02	0.89 ± 0.11
	Expression block	0.93 ± 0.02	0.87 ± 0.11
Group B	Baseline block	0.95 ± 0.01	0.90 ± 0.07
	Expression block	0.96 ± 0.01	0.89 ± 0.08

Table S2
Reaction time (ms) for Go trials in Baseline & Expression blocks (median ± SD)

		Go trials
Group A	Baseline block	360.38 ± 28.21
	Expression block	366.61 ± 38.06
Group B	Baseline block	345.02 ± 22.28
	Expression block	337.44 ± 18.64

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