

**RS 1: Experiment 1 (published in Chiappini S, Turrini S, Zanon M, Marangon M, Borgomaneri S, Avenanti A (2024). Driving Hebbian plasticity over ventral premotor-motor projections transiently enhances motor resonance. Brain Stimulation 17, 211-220).**

## Background

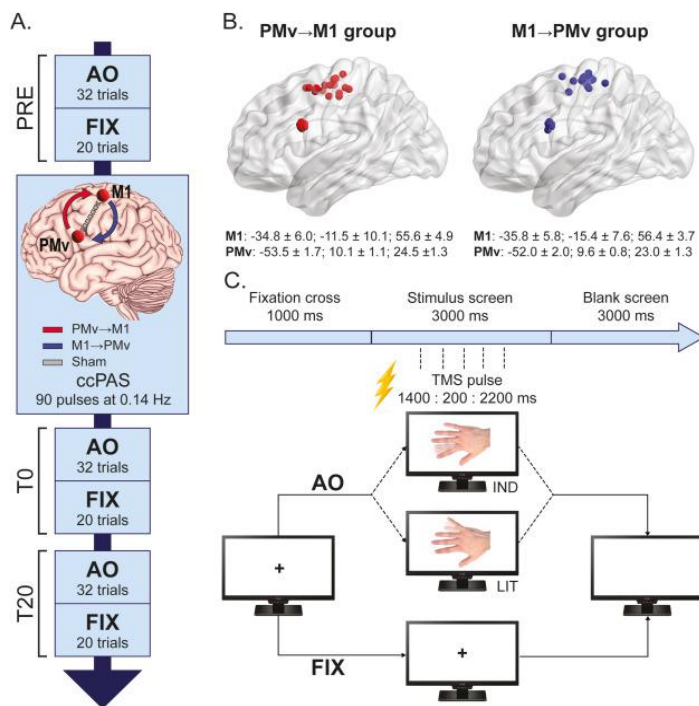
Making sense of others' actions relies on the activation of an action observation network (AON), which maps visual information about observed actions onto the observer's motor system. This motor resonance process manifests in the primary motor cortex (M1) as increased corticospinal excitability finely tuned to the muscles engaged in the observed action. Motor resonance in M1 is facilitated by projections from higher-order AON regions. However, whether manipulating the strength of AON-to-M1 connectivity affects motor resonance remains unclear.

## Methods

We used transcranial magnetic stimulation (TMS) in 48 healthy humans. Cortico-cortical paired associative stimulation (ccPAS) was administered over M1 and the inferior frontal cortex (IFC), specifically over the ventral premotor cortex (PMv), which constitutes a key AON node (Figure 1B). The protocol was used to induce spike-timing-dependent plasticity (STDP) in the pathway connecting IFC/PMv and M1.

We administered different ccPAS protocols over PMv and M1 in three different groups of participants (Figure 1A): the critical protocol aimed at strengthening projections from PMv to M1 (PMv→M1) by providing 90 pairs of pulses over PMv and M1, with PMv stimulation always preceding M1 stimulation (ccPAS<sub>PMv→M1</sub>). Two further protocols were used as a control. In one group, we actively stimulated the same areas but in reverse order (during ccPAS M1 stimulation preceded PMv stimulation) ccPAS<sub>M1→PMv</sub>, whereas in the other group we applied sham stimulation (ccPAS<sub>Sham</sub>).

Single-pulse TMS assessed motor resonance during action observation (Figure 1C): participants observed movies of index and little finger abductions while recording motor-evoked potentials (MEPs) to single pulse TMS over M1 from the two muscles involved in the observed movements, namely the first dorsal interosseous (FDI, controlling the index finger) and the abductor digiti minimi (ADM, controlling the little finger). Motor resonance was assessed before ccPAS (PRE), immediately after (T0) and 20 minutes after the end of the protocol (T20).

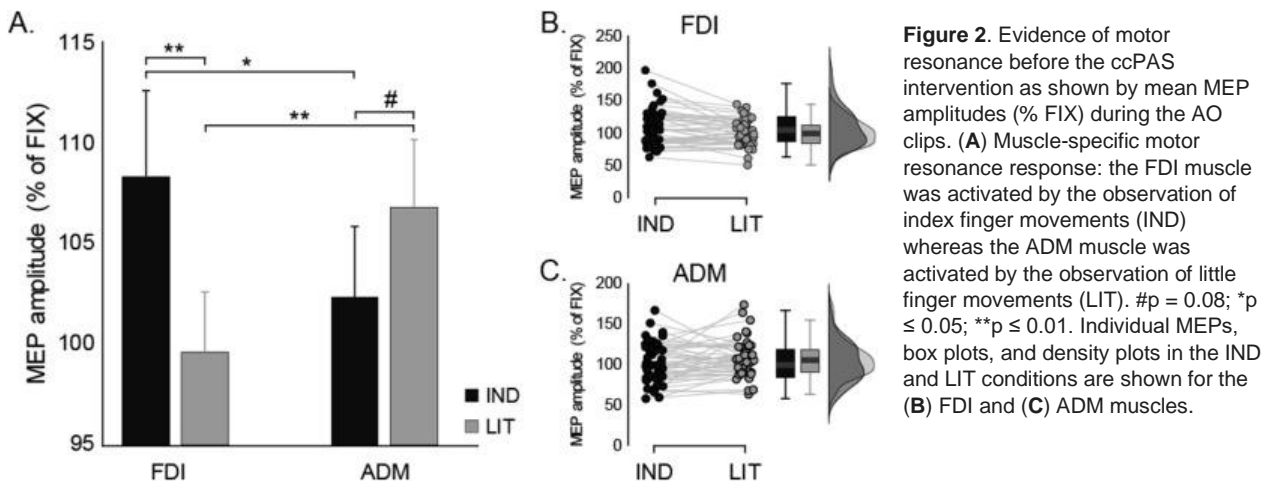


**Figure 1.** (A) Graphical representation of experimental design showing the three ccPAS groups (ccPAS<sub>PMv→M1</sub>, ccPAS<sub>M1→PMv</sub>, ccPAS<sub>Sham</sub>) and three Sessions (PRE, T0, T20) testing MEPs during action observation stimuli (AO) and a 'baseline' control condition showing a fixation cross (FIX). (B) Targeted cortical sites (C) Timeline of single-pulse TMS trials, showing an initial black fixation cross (duration: 1000 ms), followed by a stimulus screen (3000 ms) and a blank screen (3000 ms). The stimulus screen could display the same fixation cross (FIX; 20 trials), or a video-clip of a finger movement (AO; 32 trials) showing two cycles of an abduction/adduction movement of the index finger (IND) or the little finger (LIT). In every trial, a single TMS pulse was delivered to M1 at five randomized intervals ranging from 2400 to 3200 ms after the beginning of the trial.

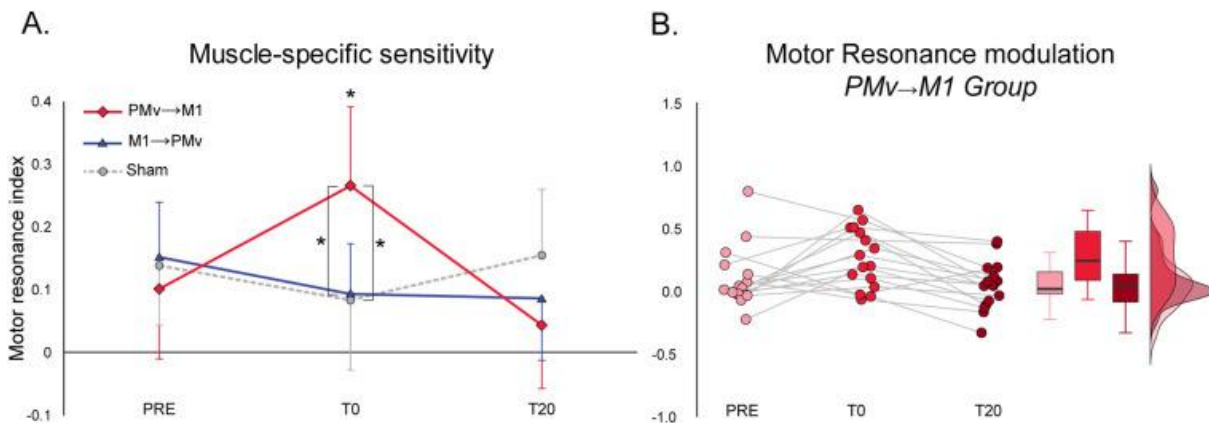
## Results

Before ccPAS, action observation increased corticospinal excitability in the muscles corresponding to the observed movements, reflecting motor resonance in M1. MEPs from the FDI were larger when observing the

movements of the index finger, whereas MEPs from the ADM were larger when observing little finger movements (Figure 2).



Notably, ccPAS aimed at strengthening projections from PMv to M1 (ccPAS<sub>PMv→M1</sub>) induced short-term enhancement of motor resonance, as shown by an index of muscle-specific motor resonance (red lines in Figure 3). The enhancement specifically occurred with the ccPAS configuration consistent with forward PMv→M1 projections and dissipated 20 min post-stimulation; ccPAS administered in the reverse order (ccPAS<sub>M1→PMv</sub>) and sham stimulation (ccPAS<sub>Sham</sub>) did not affect motor resonance.



## Conclusions

These findings provide the first evidence that inducing STDP to strengthen PMv input to M1 neurons causally enhances muscle-specific motor resonance in M1. This study sheds light on the plastic mechanisms that shape AON functionality and demonstrates that exogenous manipulation of AON connectivity can influence basic mirror mechanisms that underlie social perception.

## RS 1: Experiment 2 (Turrini et al. in preparation)

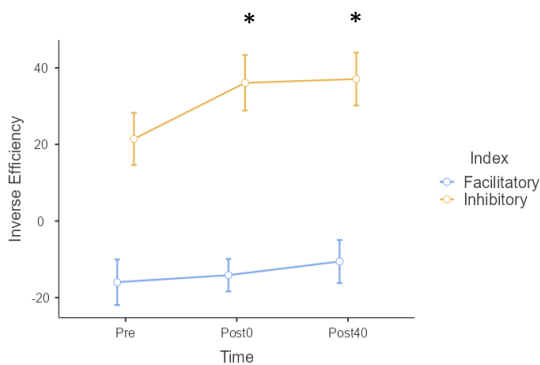
In this follow-up experiment, we extended RS1 by combining ccPAS and TMS–EEG to investigate how modulating PMv–M1 connectivity affects both motor resonance and EEG indices of action perception and conflict monitoring.

## Methods

Participants (N=24) underwent ccPAS<sub>PMV→M1</sub> and ccPAS<sub>M1→PMV</sub> with the same parameters used in Experiment 1, while performing an action observation task requiring overt finger movements that were congruent or incongruent with the observed actions (see also RS2, Experiment 1). This design allowed us to concurrently assess motor resonance and cognitive control mechanisms. TMS was applied in half of trials, allowing to analyze TMS-evoked potentials and event-related potentials (ERPs) induced by the observed action at baseline, and during the observation of a congruent / incongruent actions.

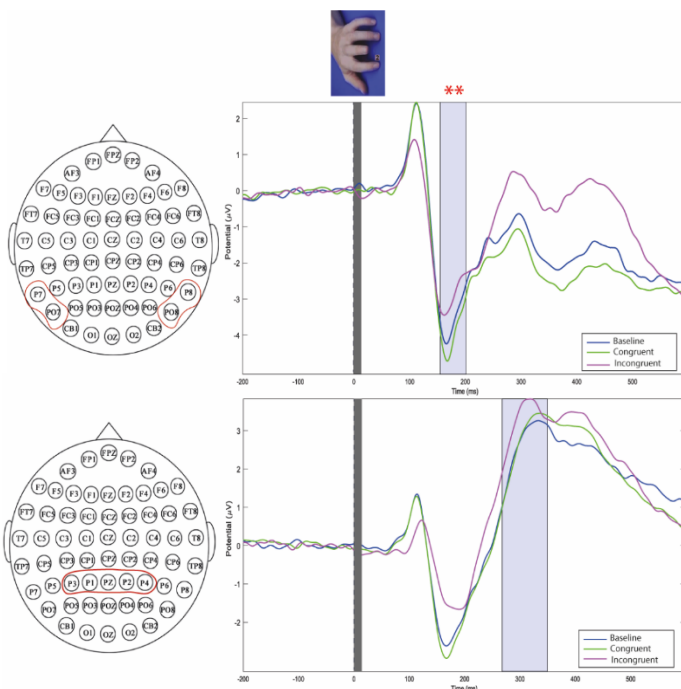
## Results

At the behavioral level, participants replicated the interference pattern observed in prior studies (e.g. Turrini et al., 2024; see RS2 Experiment 1), showing a stronger imitative tendency after ccPAS-induced strengthening of PMV–M1 projections (Figure 4; see also RS2, Experiment 1).



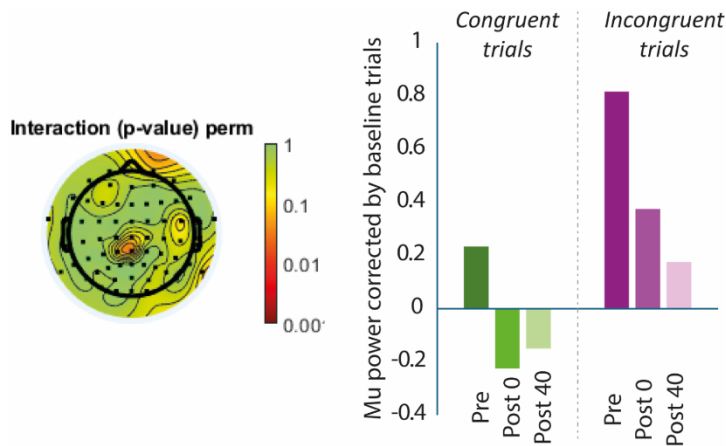
**Figure 4.** Evidence of increased motor resonance following ccPAS<sub>PMV→M1</sub>. Behavioral performance shows increased interference when participants observed actions incongruent with the movement being executed, consistent with a strengthened imitative tendency and greater motor resonance after stimulation. Error bars denote SEM.

In the EEG, we identified distinct event-related components consistent with the literature (Figure 5): N190, a parieto-occipital visual component linked to action perception, was greater for congruent than incongruent trials, confirming sensitivity to observed movements. P300b, a parietal component related to conflict processing, was higher for incongruent trials, reflecting enhanced cognitive control and conflict monitoring during imitation inhibition. These components were not affected by ccPAS<sub>PMV→M1</sub> or sham stimulation, indicating that the intervention specifically enhanced motor resonance while leaving perceptual and higher-order cognitive processes unaffected.



**Figure 5.** Influence of action congruency on visual (N190) and cognitive control (P300b) ERP components. Congruent actions elicited larger N190 responses over parieto-occipital sites, while incongruent actions evoked larger P300b amplitudes over parietal electrodes, reflecting increased perceptual encoding and conflict monitoring, respectively

TMS-evoked potentials (TEPs) were not affected by the different experimental conditions or ccPAS. On the other hand mu-rhythm (8–13 Hz) suppression over sensorimotor regions was stronger during congruent than incongruent trials. Before ccPAS (Pre), incongruent trials showed reduced mu suppression relative to neutral trials (positive bars), indicating weaker motor resonance, whereas following ccPAS<sub>PMv→M1</sub>, this pattern was markedly reversed, with a clear increase in mu suppression for incongruent trials, consistent with enhanced motor resonance. Similar, although non-significant, tendencies were observed for congruent trials (Figure 6).



**Figure 6.** Changes in mu power during action execution under congruent and incongruent conditions (expressed as difference from baseline). Positive values before ccPAS indicate reduced mu suppression, reflecting weaker motor resonance. Following ccPAS<sub>PMv→M1</sub>, this difference was markedly reduced, indicating enhanced motor resonance specifically during incongruent trials. A similar, though non-significant, trend toward increased motor resonance was observed for congruent trials.

## Conclusions

Building on Experiment 1, Experiment 2 further shows that strengthening PMv→M1 projections through ccPAS modulates neurophysiological markers of motor resonance during action observation and execution. After stimulation, mu-rhythm suppression increased for incongruent actions, indicating enhanced motor resonance specifically when executed and observed actions conflicted. In contrast, visual (N190) and cognitive (P300b) components were unaffected, suggesting that ccPAS over the PMv-M1 pathway selectively influenced motor-level processes. These findings further demonstrate that Hebbian plasticity within the PMv-M1 pathway can dynamically shape the action observation network and automatic imitation mechanisms.

**RS 2:** Building on the neurophysiological evidence of RS1, we applied the same ccPAS protocols during an automatic imitation task to test whether the same influences on motor resonance also apply at a behavioral level.

**RS 2 - Experiment 1 (published in Turrini S, Fiori F, Bevacqua N, Saracini C, Lucero B, Candidi M, Avenanti A. Spike-timing-dependent plasticity induction reveals dissociable supplementary- and premotor-motor pathways to automatic imitation. Proc Natl Acad Sci U S A. 2024;121(27):e2404925121. doi:10.1073/pnas.2404925121)**

## Background

Humans tend to spontaneously imitate others' behavior, even when detrimental to the task at hand. The action observation network (AON) is consistently recruited during imitative tasks. However, whether automatic imitation is mediated by cortico-cortical projections from AON regions to the primary motor cortex (M1) remains speculative. Similarly, the potentially dissociable role of AON-to-M1 pathways involving the ventral premotor cortex (PMv) or supplementary motor area (SMA) in automatic imitation is unclear.

In our original proposal, we hypothesized that the dorsal prefrontal cortex might play a role in controlling automatic imitation. However, a thorough review of neuroimaging meta-analyses suggests that the supplementary motor area (SMA) is a more suitable target. As we report in our article, SMA is consistently activated during both action observation and execution. Notably, invasive recording studies in both monkeys and humans have shown that SMA neurons adjust their firing rates based on the contextual appropriateness of an action. These neurons can switch from excitation during action execution to inhibition during action observation, exhibiting state-dependent properties. Therefore, in this study, we decided to use ccPAS to

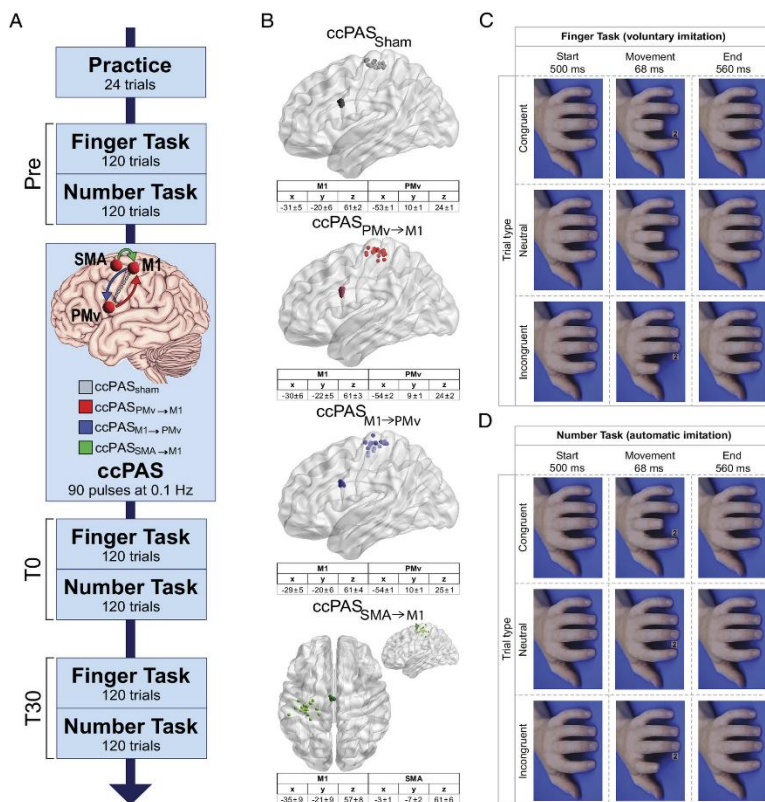
manipulate the strength of cortico-cortical connectivity of the PMv-M1 and SMA-M1 pathways to test their functional roles.

## Methods

We used ccPAS to enhance or hinder effective connectivity in PMv-to-M1 and SMA-to-M1 pathways via Hebbian spike-timing-dependent plasticity (STDP) to test their functional relevance to automatic and voluntary motor imitation.

We tested 80 participants divided in four groups undergoing different ccPAS protocols (Figure 7A, B). Two protocols aimed at strengthening PMv-to-M1 (ccPAS<sub>PMv→M1</sub> group) or SMA-to-M1 (ccPAS<sub>SMA→M1</sub> group) directional connectivity. A ccPAS<sub>M1→PMv</sub> group controlled for the protocol's directionality, and a sham ccPAS served as a control for unspecific effects (ccPAS<sub>Sham</sub> group).

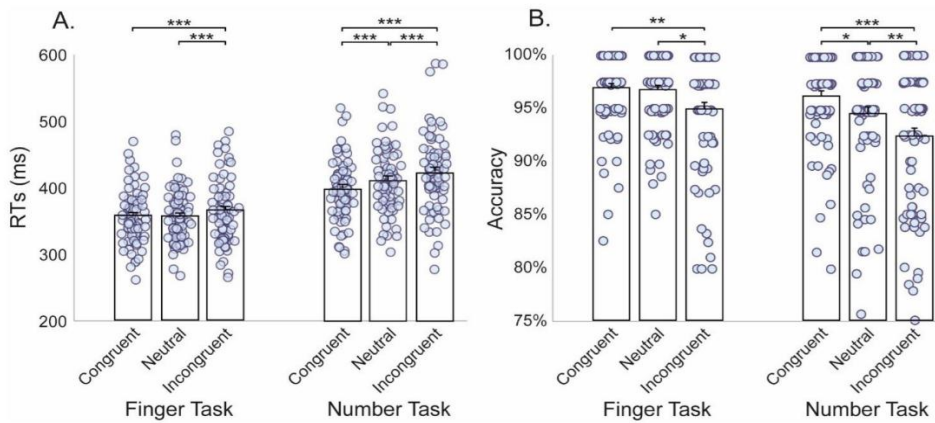
Participants completed two tasks before and after the ccPAS intervention. The two tasks were selected to tap into automatic and voluntary imitation. To assess automatic imitation, we asked participants to perform an established imitation inhibition task that uses numbers as symbolic cues dictating task rules, and finger movements as task-irrelevant cues—i.e., the Number Task (Figure 7D). To control for functional specificity, participants also performed a voluntary imitation task (Finger Task), where they were required to imitate the observed finger movements and ignore symbolic cues (i.e., numbers) (Figure 7C).



**Figure 5.** (A) General experimental procedure. (B) Individual targeted brain sites. (C) Schematic depiction of the Finger Task with finger movements as the relevant dimension, tapping into overt imitation, and the numbers as the task-irrelevant feature; (D) schematic depiction of the Number Task with symbolic cues (numbers), as the relevant dimension and the finger movements as the task-irrelevant feature, tapping into automatic imitation. Finger movements were associated with number 1 for the index finger and number 2 for the middle finger. In the congruent conditions, the number associated with the moving finger appeared, while the incongruent condition consisted of nonmatched number and finger. Congruent and incongruent trials (rows 1 and 3 of panels C and D) were identical for both tasks and differed only for the instruction given: In the Finger Task (panel C), participants were explicitly asked to imitate the finger movements they saw while ignoring the numbers, while in the Number Task (panel D), they were asked to lift the finger associated with the shown number while ignoring the movements of the fingers.

## Results

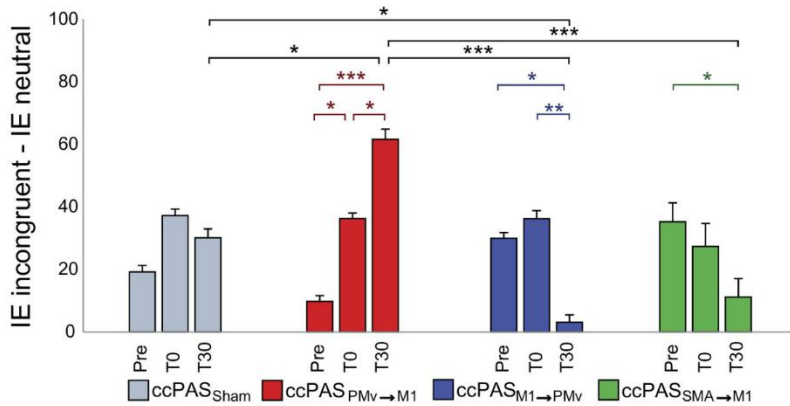
Before ccPAS, participants showed classical signs of automatic imitation, as shown by reaction times (RTs) and accuracy (% of correct response) during the Number task: compared to neutral trials, participants showed faster RTs and greater accuracy in congruent trials and slower RTs and reduced accuracy in incongruent trials. Moreover, in the Finger task (overt imitation), participants showed slower RTs and lower accuracy in incongruent trials. See Figure 8.



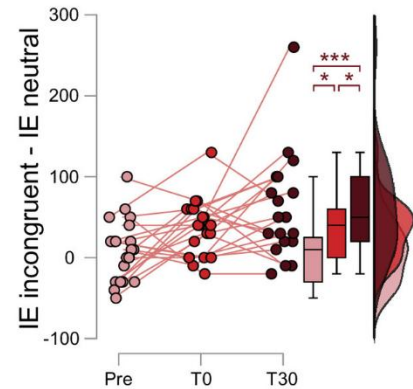
**Figure 8.** Behavioral performance in the Pre session, before ccPAS administration. **(A)** Reaction times (RTs). **(B)** Accuracy (% of correct response). \*  $p \leq 0.05$ ; \*\*\*  $p \leq 0.001$ .

Importantly, ccPAS affected behavior under competition between task rules and prepotent visuomotor associations underpinning automatic imitation (Finger task). Critically, we found dissociable effects of manipulating the strength of the two pathways (Figure 9). While strengthening PMv-to-M1 projections using ccPAS<sub>PMv→M1</sub> enhanced automatic imitation, weakening them via ccPAS<sub>M1→PMv</sub> hindered it. On the other hand, strengthening SMA-to-M1 projections reduced automatic imitation.

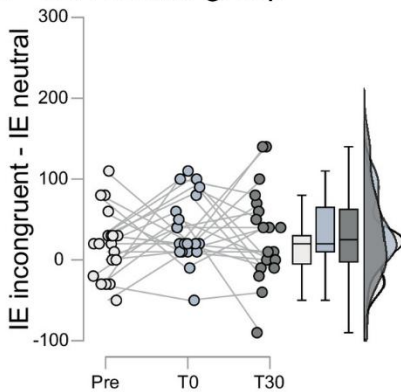
### A Interference Index - Number Task



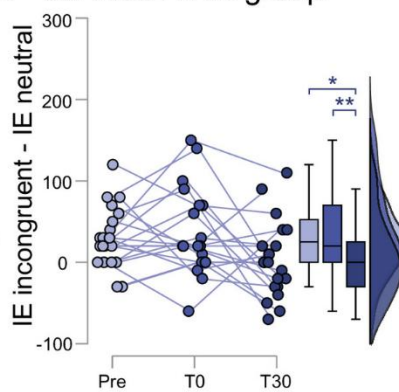
### B ccPAS<sub>PMv→M1</sub> group



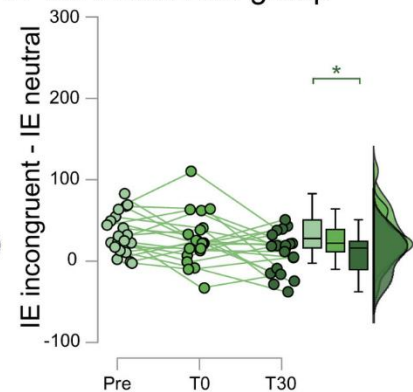
### C ccPAS<sub>Sham</sub> group



### D ccPAS<sub>M1→PMv</sub> group

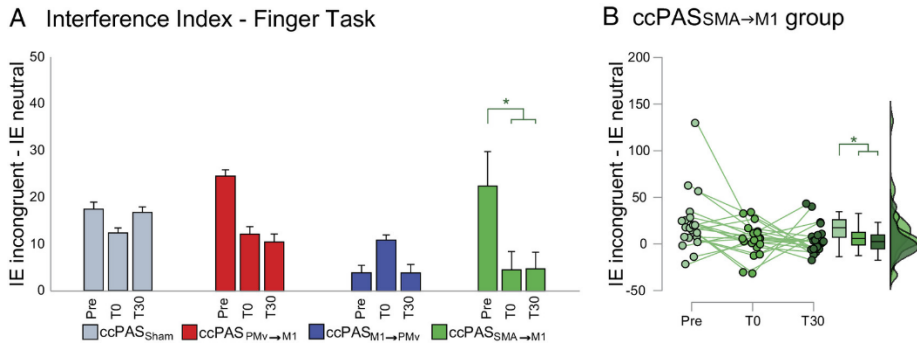


### E ccPAS<sub>SMA→M1</sub> group



**Figure 9 (A)** The interference index observed in incongruent trials of the Number Task, tapping into automatic imitation, changed as a function of time and ccPAS protocol. **(B–E)** Individual data across the four ccPAS groups. Following the ccPAS protocol, interference increased in the ccPAS<sub>PMv→M1</sub> group **(B)**, remained unchanged in the ccPAS<sub>Sham</sub> group **(C)**, and decreased in the ccPAS<sub>SMA→M1</sub> **(E)** and ccPAS<sub>M1→PMv</sub> **(D)** groups. \*  $p \leq 0.05$ ; \*\*  $p \leq 0.01$ ; \*\*\*  $p \leq 0.001$ .

Additionally, we found that strengthening SMA-to-M1 projections via  $ccPAS_{SMA \rightarrow M1}$  reduced interference from task-irrelevant cues during voluntary imitation, as shown by changes in performance in the Finger Task (Figure 10).



**Figure 10.** (A) Interference index observed in incongruent trials of the Finger Task. (B) Individual data of the  $ccPAS_{SMA \rightarrow M1}$  group. Following the  $ccPAS$  protocol, interference decreased only in the  $ccPAS_{SMA \rightarrow M1}$  group.  $*p \leq 0.05$ .

## Conclusions

Our study demonstrates that driving Hebbian STDP in AON-to-M1 projections induces opposite effects on automatic imitation that depend on the targeted pathway. Our results provide direct causal evidence of the functional role of PMV-to-M1 projections for automatic imitation, seemingly involved in spontaneously mirroring observed actions and facilitating the tendency to imitate them. Moreover, our findings support the notion that SMA exerts an opposite gating function, controlling M1 to prevent overt motor behavior when inadequate to the context.

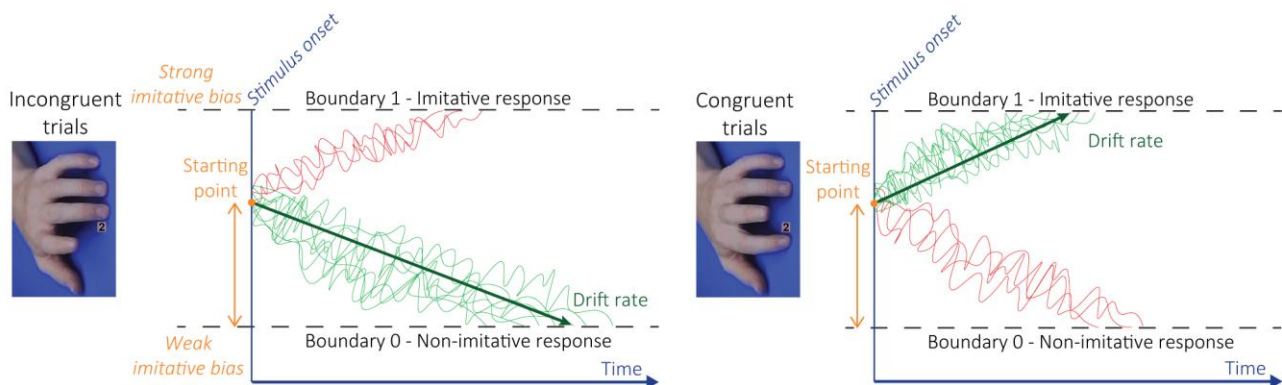
**RS 2 - Experiment 2 (published in Turrini S, Tarasi L, Bevacqua N, Fiori F, Zago S, Arcara G, Candidi M, Romei V, Avenanti A. Disentangling the functional roles of premotor-motor pathways in automatic imitation: A combined network-based transcranial stimulation and drift diffusion modeling approach. J Neurosci. 2025 Oct 2:e0340252025. doi:10.1523/JNEUROSCI.0340-25.2025)**

## Background

Building on RS2–Exp1, we asked whether strengthening or weakening premotor→M1 pathways alters latent components of automatic imitation—baseline imitative bias vs. evidence integration. To test this hypothesis, we re-analyzed data from Experiment 1 using hierarchical Drift Diffusion Modeling (HDDM).

## Methods

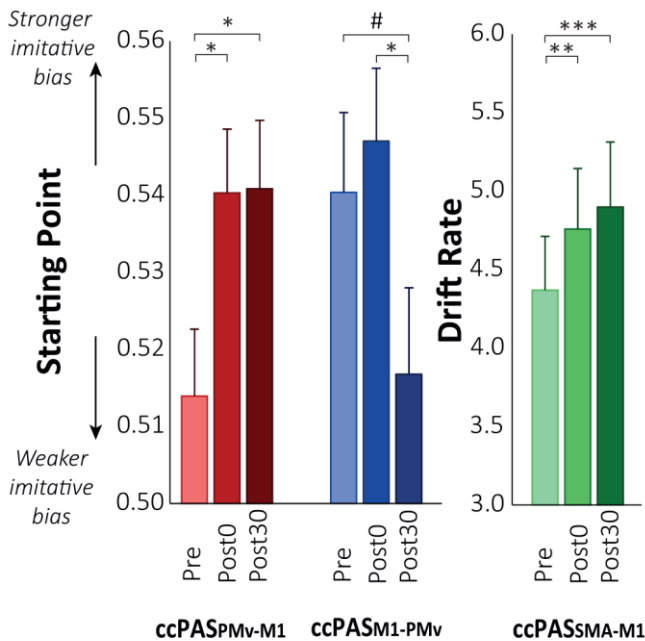
Data from sixty healthy young adults from the three key groups in Experiment 1 ( $ccPAS_{PMV \rightarrow M1}$ ,  $ccPAS_{M1 \rightarrow PMV}$ ,  $ccPAS_{SMA \rightarrow M1}$ ) were extracted. We applied HDDM to congruent/incongruent trials only, classifying responses as imitative vs. non-imitative. Two parameters indexed dissociable mechanisms: starting point ( $z$ ) = pre-decisional imitative bias; drift rate ( $v$ ) = efficiency of integrating task-relevant information.



**Figure 11.** Drift–diffusion model (DDM) schematic. Each thin line is one trial’s noisy evidence accumulation starting at  $z$  and moving toward one of two decision boundaries (imitative vs non-imitative). Crossing a boundary triggers the response (green = correct, red = error). In congruent trials the correct boundary is imitative; in incongruent trials it is non-imitative. The starting point ( $z$ ) indexes bias toward imitation ( $z = 0.5 =$  no bias). The drift rate ( $v$ ) indexes how efficiently task evidence is integrated (higher  $v \rightarrow$  faster, more accurate decisions).

## Results

Results are shown in Figure 12. At baseline, participants showed an imitative bias ( $z > 0.5$ ). ccPAS predicted pathway-specific effects on latent components of decision-making. ccPAS<sub>PMv→M1</sub> increased the starting point ( $z$ ), indicating a stronger pre-decisional bias toward imitation, whereas ccPAS<sub>M1→PMv</sub>, showed the opposite trend, reducing imitative bias. In contrast, ccPAS<sub>SMA→M1</sub> selectively increased the drift rate ( $v$ ), reflecting more efficient evidence accumulation and improved rule-based action selection. These effects confirm a double dissociation between PMv–M1 (bias modulation) and SMA–M1 (evidence integration) pathways.



**Figure 12.** Effect of the three ccPAS protocols on different components of automatic imitative behavior. Error bars represent 1SEM. # $p = .06$ ; \* $p < .05$ ; \*\* $p < .01$ ; \*\*\* $p < .001$ .

## Conclusions

ccPAS over PMv→M1 bidirectionally tunes the baseline imitative bias (starting point), whereas ccPAS over SMA→M1 enhances evidence accumulation (drift rate) for rule-guided action. These findings provide causal, parameter-level evidence for dissociable roles of premotor–motor pathways in facilitating (PMv) vs. regulating (SMA) automatic imitation.

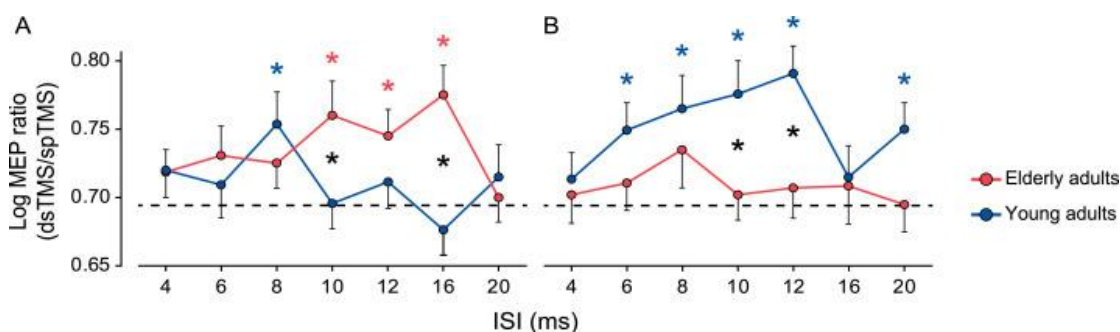
## General conclusions

Across Research Streams 1 and 2, we demonstrated that cortico-cortical projections within the AON are malleable and causally relevant for shaping how the motor system resonates with, and regulates, observed actions. Strengthening ventral premotor-to-motor (PMv→M1) projections ccPAS transiently enhanced muscle-specific motor resonance during action observation and increased mu-rhythm suppression, without affecting visual or cognitive ERP components, indicating selective modulation of motor-level mirroring processes. Extending these findings to overt behavior, we showed that PMv→M1 potentiation facilitates automatic imitation, whereas strengthening supplementary motor-to-motor (SMA→M1) projections exerts an opposite, regulatory effect, reducing imitative interference and enhancing rule-based control. Computational modeling further revealed that PMv–M1 and SMA–M1 pathways distinctly contribute to different decision components: PMv modulates the pre-decisional imitative bias, while SMA enhances evidence accumulation during deliberate action selection. Taken together, these results provide converging causal, neurophysiological, behavioral, and computational evidence that AON-to-M1 connectivity is both plastic and functionally specific, mediating the balance between spontaneous motor resonance and cognitive control over imitation. This integrated framework advances our understanding of how social perception and motor control emerge from dynamic, plastic interactions between distinct premotor–motor circuits, and lays the groundwork for targeted neuromodulatory interventions in conditions characterized by disrupted mirroring and imitation control.

## Pilot study 1-3 – Research Stream 1

### Pilot Study 1 – Timing of premotor–motor interactions (dsTMS) in young and older individuals

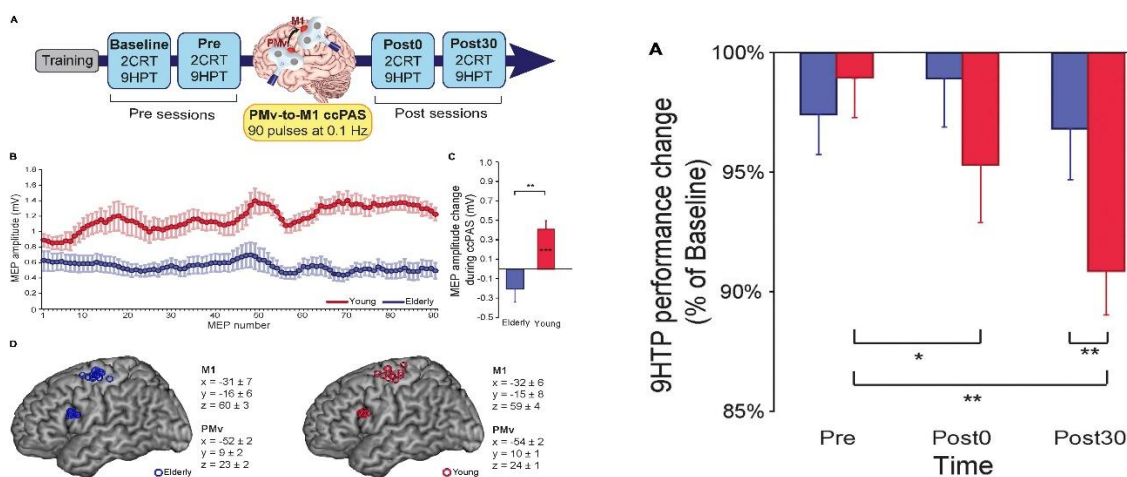
(Published in Chiappini et al., 2025 Archives of Medicine Research). In 34 participants (17 young, 17 older), we mapped PMv/IFG→M1 and preSMA/SMA→M1 effective connectivity across interstimulus intervals (ISIs) ranging 4–20 ms to identify the optimal timing for subsequent ccPAS protocols (Figure 8). In young adults, both premotor–motor pathways showed a reliable facilitation at an 8-ms ISI, whereas in older adults these interactions were delayed or absent. This experiment established 8 ms as the physiologically optimal ISI for driving Hebbian-like plasticity using ccPAS over both PMv–M1 and SMA–M1 circuits—key connections later targeted in RS1 (motor resonance) and RS2 (automatic imitation).



**Figure 8.** Changes in MEP amplitude elicited by M1 stimulation following PMv (left) and SMA (right) conditioning. The 8-ms ISI was identified as optimal only in young adults..

### Pilot Study 2 – ccPAS in young and older adults (published in Turrini et al., 2023 Frontiers in Aging Neuroscience)

We tested motor excitability in 14 young and 13 older participants during ccPAS<sub>PMv→M1</sub> (ISI = 8 ms) and the effect of ccPAS on PMv–M1 functions (Figure 9). In young adults, ccPAS induced a progressive increase in MEP amplitude and improved PMv–M1 functionality at the behavioral level (9-Hole Peg Test). No consistent physiological or behavioral effects emerged in older adults. Across participants, the magnitude of MEP modulation predicted performance gains, linking PMv–M1 potentiation to improved motor function. These findings provided proof of concept that ccPAS (8 ms) effectively induces Hebbian plasticity in the PMv–M1 pathway and highlighted young adults as the most responsive population for testing our hypotheses in RS1 and RS2.

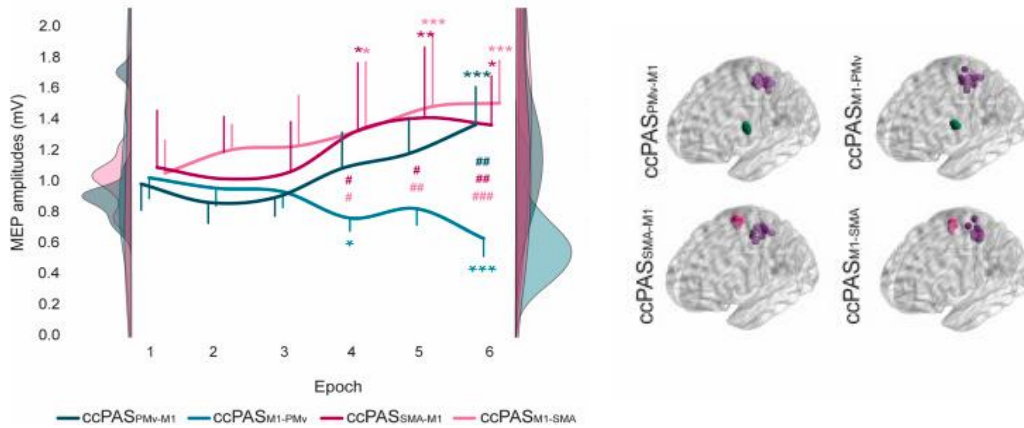


**Figure 9.** Left panel: Changes in MEP amplitude during ccPAS in the group of young (red) and older (blue) participants. Right panel: effect of ccPAS<sub>PMv→M1</sub> AS on motor performance. The protocol improved execution time only in the group of young participants.

### Pilot Study 3 – ccPAS across premotor–motor circuits in young adults at rest (published in Bevacqua et al., 2024 Brain Stimulation)

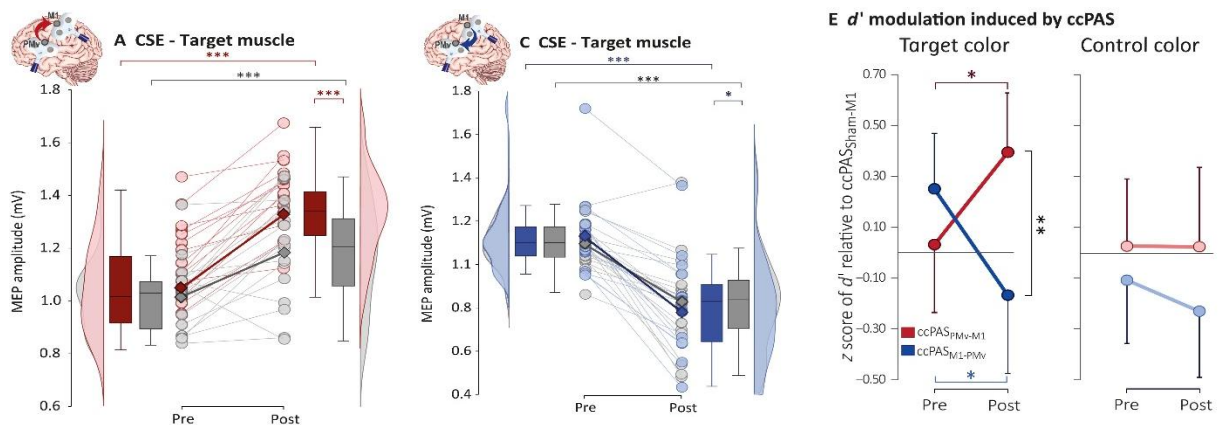
In 60 young adults, we compared four 8-ms ISI ccPAS protocols on motor excitability during the administration of the protocols. Results show that ccPAS<sub>PMv→M1</sub> induced a gradual increase in motor excitability, while the reverse order (ccPAS<sub>M1→PMv</sub>) produced motor inhibition. In contrast,

ccPAS<sub>SMA→M1</sub> and ccPAS<sub>M1→SMA</sub> elicited facilitation regardless of direction. We concluded that PMv–M1 pathway can be bidirectionally modulated (LTP-/LTD-like effects), providing a robust tool to causally probe the influence of PMv projections on M1, whereas SMA influences on M1 can be enhanced with ccPAS<sub>SMA→M1</sub>, but further investigation is required to address bidirectional influences. These findings directly informed the design of RS1 (PMv→M1 effects on motor resonance) and RS2 (PMv→M1 and SMA→M1 effects on automatic imitation).



**Figure 10.** Changes in MEPs during the administration of ccPAS over PM-M1 and SMA-M1 circuits.

**Pilot Study 4 – ccPAS across PMv–M1 in young adults during visuomotor associations (published in Turrini et al., 2025 Science Advances).** A further pilot study was designed to validate the functional and mechanistic specificity of our ccPAS approach before applying it to action observation context. Specifically, we aimed to test whether ccPAS effects depend on the functional state of the targeted network, i.e., whether pairing PMv and M1 while participants are actively engaged in a visuomotor task produces selective, task-relevant plasticity. Across four experiments in healthy young adults (total N = 72), state-dependent ccPAS<sub>PMv→M1</sub> (8 ms ISI) delivered during task performance induced LTP-like, action-specific facilitation of corticospinal excitability and improved cue–action discrimination (higher  $d'$ ). The reverse order (ccPAS<sub>M1→PMv</sub>) produced LTD-like, action-specific suppression (Figure 10). This proof-of-principle confirmed that ccPAS plasticity is state-dependent and direction-specific, strengthening the methodological rationale for applying ccPAS during action observation and imitation in RS1 and RS2.



**Figure 10.** State-dependent ccPAS<sub>PMv→M1</sub> induced LTP-like, action-specific facilitation of motor excitability and improved cue–action discrimination (higher  $d'$ ). State-dependent ccPAS<sub>M1→PMv</sub> produced LTD-like, action-specific suppression, and hindered cue–action discrimination (lower  $d'$ ).

**Overall conclusion of Pilot studies:** Across the four pilot studies, we identified 8 ms as the optimal interstimulus interval for ccPAS targeting PMv–M1 and SMA–M1 pathways, and demonstrated robust, direction-specific Hebbian plasticity, predominantly in young adults. Together, these findings validated both the timing and the population for subsequent experiments in Research Stream 1 (motor resonance) and Research Stream 2 (automatic imitation), ensuring that ccPAS interventions would be applied under the most effective physiological conditions.

## Final report

### Table highlighting the differences between expected and achieved output indicators

#### Expected and achieved output indicators (number of actions)

Output indicators	Expected (according to application)	Achieved
PhD thesis	1	2 PhD theses (1 already discussed, 1 to be discussed in early 2026)
Master's thesis	4	11 (see list below)
Organization of seminar or conference	0	0
Book	0	0
Book chapter	0	0
Conference presentation	2	6 (see list below)
Conference paper	0	0
Journal article	2	17 articles + 2 preprints (see list below)
Other (specify)	0	0

**Notes:**

**PhD Theses:**

1. Sonia Turrini. Induction of Hebbian associative plasticity through paired noninvasive brain stimulation of premotor-motor areas to elucidate the network's functional role. University of Bologna, Italy, July 2023.
2. Naomi Bevacqua. Modulating the Action Observation Network through transcranial magnetic stimulation: causal insights into automatic imitation phenomenon. PhD concluded, thesis to be discussed.

**Master degree theses:**

1. Miss Pierangela Mencarelli, registration number 000993556, Thesis title: Differenze Funzionali nella Neuroplasticità dei circuiti premotori-motori in giovani adulti e anziani
2. Miss Sara Caruso Cervera, registration number 0001039800, Thesis title: Il ruolo dei circuiti premotorio-motori sull'imitazione automatica: effetti di neuroplasticità indotta mediante ccPAS
3. Miss Laura Di Rosa, registration number 0001046269, Thesis title: Neuroplasticità delle connessioni premotorie e motorie nell'imitazione automatica: studi di ccPAS e comportamento in giovani adulti
4. Miss Giulia D'Ecclesiis, registration number 0001042728, Thesis title: Il ruolo delle aree ventrali e dorsali del network fronto-parietale nell'imitazione automatica: uno studio rTMS
5. Miss Chiara Bosi, registration number 0001088646, Thesis title: Sclerosi Multipla e connettività del sistema motorio: uno studio TMS esplorativo sul gruppo di controllo sano

6. Miss Giada Scala, registration number 0000998738, Thesis title: Just like you, just like me: il come e il perché dell'automatic imitation
7. Miss Anastasia Amadori, registration number 0001096416, Thesis title: Il protocollo di stimolazione ccPAS applicato al circuito PMv-M1: uno studio sulla coerenza temporale nell'induzione di plasticità
8. Mr Enrico Maria Orditura, registration number 0001093462, Thesis title: L'inibizione della giunzione temporo-parietale destra amplifica la percezione del dolore e la punizione di azioni accidentali: uno studio cTBS
9. Miss Angela Scarci, registration number 0001098757, Thesis title: Brain state dependent application of TMS to study the functional correlates of cortical connectivity: a movement phase locked ccPAS study
10. Miss Miriam Pia Giunta, registration number 0001080545, Thesis title: Il ruolo delle aree SMA e PMv sull'imitazione automatica: uno studio rTMS
11. Miss Olivia Giustini, registration number 0001141782, Thesis title: Contributi differenziali delle cortecce premotorie dorsale e ventrale all'apprendimento e al consolidamento di associazioni visuo-motorie arbitrarie

### **Conference presentations**

1. Sonia Turrini (2024). Spike-time-dependent plasticity induction through cortico-cortical paired associative stimulation (ccPAS) reveals dissociable neural ventral and medial motor pathways to automatic imitation. 32nd Annual congress of the Italian Society of Psychophysiology and Cognitive Neuroscience (SIPF 2024), Cesena, Italy, September 4-6 2024. [Oral communication]. Young researcher award.
2. Sonia Turrini, Naomi Bevacqua, Chiara Saracini, Boris Lucero, Matteo Candidi, Alessio Avenanti (2024). Spike-time-dependent plasticity induction through cortico-cortical paired associative stimulation (ccPAS) reveals dissociable neural ventral and medial motor pathways to automatic imitation. 30th Annual congress of the Italian Association of Psychology (AIP) Experimental section 2025, Noto, Italy, September 23-25, 2025. [Poster].
3. Sonia Turrini (2024). Spike-time-dependent plasticity induction through cortico-cortical paired associative stimulation (ccPAS) reveals dissociable neural ventral and medial motor pathways to automatic imitation. 30th Annual congress of the Italian Association of Psychology (AIP) Experimental section 2025, Noto, Italy, September 23-25, 2024. [Oral communication].
4. Sonia Turrini, Naomi Bevacqua, Chiara Saracini, Boris Lucero, Matteo Candidi, Alessio Avenanti (2025). Spike-time-dependent plasticity induction reveals dissociable premotor-motor pathways to automatic imitation. 22nd World Congress of Psychophysiology (IOP 2025), Krakow, Poland, July 8–11, 2025 [oral communication]
5. Alessio Avenanti. Dissecting the Social Brain with Neurostimulation: Mapping Connectivity, Plasticity, and Individual Differences. 31st Annual congress of the Italian Association of Psychology (AIP) Experimental section 2025, Torino, Italy, September 11-13, 2025 [oral communication]
6. Sonia Turrini, Emilio Chiappini, Alessio Avenanti (2025). Causal enhancement of feedback connectivity in the Action Observation Network improves prediction of observed human actions. 33rd Annual congress of the Italian Society of Psychophysiology and Cognitive Neuroscience (SIPF 2024), Verona, Italy [oral communication]

### **List of publications**

#### **A) Publications directly stemming from the project's topics and methodology: Research Stream 1**

1. Chiappini E, Turrini S, Zanon M, Marangon M, Borgomaneri S, Avenanti A. Driving Hebbian plasticity over ventral premotor-motor projections transiently enhances motor resonance. *BRAIN STIMULATION*. 2024;17(2):211–220. doi:10.1016/j.brs.2024.02.011
2. Chiappini E, Turrini S, Fiori F, Benassi M, Tessari A, di Pellegrino G, Avenanti A. You are as old as the connectivity you keep: Distinct neurophysiological mechanisms underlying age-related changes in hand dexterity and strength. *ARCHIVES OF MEDICAL RESEARCH*. 2025;56(1):103031. doi:10.1016/j.arcmed.2024.103031
3. Turrini S, Bevacqua N, Cataneo A, Chiappini E, Fiori F, Candidi M, Avenanti A. Transcranial cortico-cortical paired associative stimulation (ccPAS) over ventral premotor-motor pathways enhances action performance and corticomotor excitability in young adults more than in elderly adults. *FRONTIERS IN AGING NEUROSCIENCE*. 2023;15:1119508. doi:10.3389/fnagi.2023.1119508
4. Bevacqua N, Turrini S, Fiori F, Saracini C, Lucero B, Candidi M, Avenanti A. Cortico-cortical paired associative stimulation highlights asymmetrical communication between rostral premotor cortices and primary motor cortex. *BRAIN STIMULATION*. 2024;17(1):89–91. doi:10.1016/j.brs.2024.01.001

*Publication 1 reports the main study of Research Stream 1. Publications 2-4 report the results of RS1 pilot studies 1-3, respectively.*

## **B) Publications directly stemming from the project's topics and methodology: Research Stream 2**

5. Turrini S, Fiori F, Bevacqua N, Saracini C, Lucero B, Candidi M, Avenanti A. Spike-timing-dependent plasticity induction reveals dissociable supplementary- and premotor-motor pathways to automatic imitation. *PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF AMERICA (PNAS)*. 2024;121(27):e2404925121. doi:10.1073/pnas.2404925121
6. Turrini S, Tarasi L, Bevacqua N, Fiori F, Zago S, Arcara G, Candidi M, Romei V, Avenanti A. Disentangling the functional roles of premotor-motor pathways in automatic imitation: A combined network-based transcranial stimulation and drift diffusion modeling approach. *JOURNAL OF NEUROSCIENCE*. 2025 Oct 2:e0340252025. doi:10.1523/JNEUROSCI.0340-25.2025
7. Turrini S, Fiori F, Arcara G, Romei V, di Pellegrino G, Avenanti A. State-dependent associative plasticity highlights function-specific premotor-motor pathways crucial for arbitrary visuomotor mapping. *SCIENCE ADVANCES*. 2025;11(20):eadu4098. doi:10.1126/sciadv.adu4098

*Publications 5 and 6 report the main study of Research Stream 2. Publication 7 reports the results of pilot study 4.*

## **C) Publications addressing topics related to the projects**

8. Cristiano A, Finisguerra A, Urgesi C, Avenanti A, Tidoni E. Functional role of the theory of mind network in integrating mentalistic prior information with action kinematics during action observation. *CORTEX*. 2023;166:107–120. doi:10.1016/j.cortex.2023.05.009
9. Rizzo G, Martino D, Avanzino L, Avenanti A, Vicario CM. Social cognition in hyperkinetic movement disorders: A systematic review. *SOCIAL NEUROSCIENCE*. 2023;18(6):331–354. doi:10.1080/17470919.2023.2248687
10. Borgomaneri S, Zanon M, Di Luzio P, Cataneo A, Arcara G, Romei V, Tamietto M, Avenanti A. Increasing associative plasticity in temporo-occipital back-projections improves visual perception of emotions. *NATURE COMMUNICATION*. 2023;14(1):5720. doi:10.1038/s41467-023-41058-3
11. Turrini S, Avenanti A. Understanding the sources of cortico-cortical paired associative stimulation (ccPAS) variability: Unraveling target-specific and state-dependent influences. *CLINICAL NEUROPHYSIOLOGY*. 2023;156:290–292. doi:10.1016/j.clinph.2023.08.019
12. Tarasi L, Turrini S, Sel A, Avenanti A, Romei V. Cortico-cortical paired-associative stimulation to investigate the plasticity of cortico-cortical visual networks in humans. *CURRENT OPINION IN BEHAVIORAL SCIENCES*. 2024;56:101359. doi:10.1016/j.cobeha.2024.101359

## **D) Preprint addressing topics related to the projects:**

13. Di Luzio P, Tarasi L, Avenanti A, Silvanto J, Sel A, Romei V. Targeted neuromodulation of perceptual decision-making networks causally dissociates sensory and metacognitive performance. *BIORXIV* [preprint]. 2025 May 20. doi:10.1101/2025.05.15.653831 (Under review in *COMMUNICATIONS BIOLOGY*)
14. Turrini S, Amadori A, Zago S, Arcara G, Avenanti A. Brain state-dependent plasticity induction through cortico-cortical paired associative stimulation: the role of timing and circuit engagement in the human PMv-to-M1 pathway. *SSRN* [preprint]. doi:10.2139/ssrn.5412682 (Under review in *BRAIN STIMULATION*)

## **E) Other related publications in which the Bial Foundation has been thanked in acknowledgments**

15. Tortora F, Hadipour AL, Battaglia S, Falzone A, Avenanti A, Vicario CM. The role of serotonin in fear learning and memory: A systematic review of human studies. *BRAIN SCIENCES*. 2023;13(8):1197. doi:10.3390/brainsci13081197
16. Spaccasassi C, Cenka K, Petkovic S, Avenanti A. Sense of agency predicts severity of moral judgments. *FRONTIERS IN PSYCHOLOGY*. 2023;13:1070742. doi:10.3389/fpsyg.2022.1070742
17. Culicetto L, Ferraioli F, Lucifora C, Falzone A, Martino G, Craparo G, Avenanti A, Vicario CM. Disgust as a transdiagnostic index of mental illness: A narrative review of clinical populations. *BULLETIN OF THE MENNINGER CLINIC*. 2023;87(Suppl A):53–91. doi:10.1521/bumc.2023.87.suppA.53
18. Vitale F, Hernández-Sauret A, Avenanti A, de Vega M. Exploring the impact of sentential negation on inhibitory motor networks: Insights from paired-pulse TMS. *BRAIN AND LANGUAGE*. 2025;262:105536. doi:10.1016/j.bandl.2025.105536
19. Borgomaneri S, Quettier T, Ambrosecchia M, Battaglia S, Tamietto M, Avenanti A. Early changes in corticospinal excitability for subliminally presented fearful body postures. *SCIENCE REPORTS*. 2025;15(1):29088. doi:10.1038/s41598-025-13185-y