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## Distinct neural mechanisms support inner speaking and inner hearing

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## Abstract

13

14 Humans have the ability to mentally examine speech. This covert form of speech  
15 production is often accompanied by sensory (e.g., auditory) percepts. However, the  
16 cognitive and neural mechanisms that generate these percepts are still debated. According  
17 to a prominent proposal, inner speech has at least two distinct phenomenological  
18 components: inner speaking and inner hearing. We used transcranial magnetic stimulation  
19 to test whether these two phenomenologically distinct processes are supported by distinct  
20 neural mechanisms. We hypothesised that inner speaking relies more strongly on an online  
21 motor-to-sensory simulation that constructs a multisensory experience, whereas inner  
22 hearing relies more strongly on a memory-retrieval process, where the multisensory  
23 experience is reconstructed from stored motor-to-sensory associations. Accordingly, we  
24 predicted that the speech motor system will be involved more strongly during inner  
25 speaking than inner hearing. This would be revealed by modulations of TMS evoked  
26 responses at muscle level following stimulation of the lip primary motor cortex. Overall,  
27 data collected from 31 participants corroborated this prediction, showing that inner  
28 speaking increases the excitability of the primary motor cortex more than inner hearing.  
29 Moreover, this effect was more pronounced during the inner production of a syllable that  
30 strongly recruits the lips (vs. a syllable that recruits the lips to a lesser extent). These  
31 results are compatible with models assuming that the primary motor cortex is involved  
32 during inner speech and contribute to clarify the neural implementation of the fundamental  
33 ability of silently speaking in one's mind.

34 *Keywords:* inner speech, inner speaking, inner hearing, transcranial magnetic  
35 stimulation, motor evoked potential, cortical excitability

36 Word count: 6587 (excluding abstract, references, tables, and figures)

## Introduction

37

38 Rehearsing a conversation, preparing a public talk, or reading a novel are routine  
39 mental activities that are usually accompanied by an inner voice (Levine et al., 1982;  
40 Morin, 2012; Perrone-Bertolotti et al., 2012; Petkov & Belin, 2013; Sokolov, 1972). This  
41 inner voice feels like speech was produced or heard internally (e.g., Hurlburt & Heavey,  
42 2015). In other words, it involves a conscious multisensory (e.g., auditory, kinaesthetic)  
43 experience (for reviews, see Alderson-Day & Fernyhough, 2015; Perrone-Bertolotti et al.,  
44 2014). Although commonly described as a unitary construct, inner speech may be better  
45 defined as a collection of distinct but related phenomenological experiences (e.g., Hurlburt,  
46 2011; Hurlburt & Akhter, 2006). According to Hurlburt (2011), it is possible to distinguish  
47 the phenomenon of *inner speaking* from the phenomenon of *inner hearing*, whose feelings  
48 would be similar to talking in a tape recorder and hearing one's voice played back,  
49 respectively (Hurlburt et al., 2013).<sup>1</sup> The present research aims at better understanding  
50 the origins of the voice we experience during inner speaking and inner hearing, that is, the  
51 neurocognitive mechanisms through which it arises.

52 Stemming upon classical models of motor control, Lœvenbruck et al. (2018)  
53 presented a predictive model of *wilful (expanded) inner speech* production (i.e., inner  
54 speaking). In this model, the auditory and kinaesthetic sensations perceived during inner  
55 speech are thought to be the predicted sensory consequences of (a copy of) inhibited speech  
56 motor acts. More precisely, these percepts are simulated by internal forward models that  
57 use the efference copies issued from an inverse model. According to this view, the primary  
58 motor cortex would be involved during inner speech production, but its output would be  
59 inhibited by prefrontal regions involved in response inhibition, such as the right inferior

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<sup>1</sup> The distinction between inner speaking and inner hearing echoes previous distinctions such as the one between the *generative component* (i.e., the feeling of producing speech) and the *auditory component* (i.e., the feeling of hearing speech) of inner speech (e.g., MacKay, 1992) and the distinction between the *inner voice* and the *inner ear* in working memory (e.g., Baddeley et al., 1984; Buchsbaum & D'Esposito, 2019).

60 frontal cortex or the pre-supplementary motor area (Nalborczyk et al., 2022). The model  
61 put forward by Løevenbruck et al. (2018) predicts that the primary motor cortex will be  
62 involved during inner speaking, but it leaves unspecified the neural mechanisms supporting  
63 inner hearing and whether they differ from those supporting inner speaking.

64 According to the dual stream prediction model (Tian et al., 2016; Tian & Poeppel,  
65 2012, 2013), the sensory content of inner speech is provided by two distinct processes.  
66 First, the sensory content of inner speech may result from a *motor simulation prediction*  
67 *stream*. In this view, inner speech would involve the same mechanisms as overt speech  
68 production except that the speech acts should be inhibited rather than executed (this  
69 proposal is similar to the model of Løevenbruck et al., 2018, although see below differences  
70 regarding the involvement of the primary motor cortex). Second, the sensory content of  
71 inner speech may be provided by an associative memory-based process called the  
72 *memory-retrieval prediction stream* (Kosslyn et al., 1979; Moulton & Kosslyn, 2009; Tian &  
73 Poeppel, 2012). In this view, sensory percepts are motor-to-sensory associations  
74 established during past events and directly retrieved from long-term memory.<sup>2</sup>

75 The distinction between the motor simulation and memory retrieval streams has been  
76 linked to the distinction between inner speaking and inner hearing (Tian et al., 2016; e.g.,  
77 Tian et al., 2018; Tian & Poeppel, 2012, 2013). Using fMRI, Tian et al. (2016) examined  
78 the neural correlates of articulation imagery (inner speaking) and hearing imagery (inner  
79 hearing) and observed that inner speaking more strongly recruits brain areas belonging to  
80 the motor-estimation stream whereas inner hearing more strongly recruits brain areas  
81 belonging to the memory-retrieval prediction stream. Moreover, these two imagery modes

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<sup>2</sup> The distinction between these two prediction streams is reminiscent of the distinction between the prediction-by-simulation and the prediction-by-association mechanisms in speech production and perception (Pickering & Garrod, 2013) and was previously discussed in more depth in Li et al. (2020), Ma and Tian (2019), Nalborczyk (2019), Nalborczyk et al. (2021), Tian and Poeppel (2012), and Tian and Poeppel (2013).

82 have been shown to have distinct MEG correlates and distinct modulatory effects on a  
83 subsequent /ba/-/da/ auditory categorisation task (Ma & Tian, 2019).

84 Critically, in the dual stream prediction model, the primary motor cortex is  
85 considered to be “bypassed” when producing inner speech (Tian et al., 2016; Tian &  
86 Poeppel, 2012, 2013). In comparison, the model introduced in Lœvenbruck et al. (2018)  
87 and Grandchamp et al. (2019) predicts that the primary motor cortex is involved during  
88 inner speech production. In essence, at least some forms of inner speech may be  
89 accompanied by the emission of motor commands that are subsequently inhibited by  
90 cortical and subcortical mechanisms.

91 The hypothesis that inner speech involves motor inhibition is compatible with  
92 behavioural, lesional, and neuroimaging studies of motor imagery (for a review, see Guillot  
93 et al., 2012). The involvement of the primary motor cortex during inner speech and its  
94 partial inhibition by cortical and subcortical mechanisms may explain the residual  
95 peripheral muscular activity that is sometimes observed during inner speech production  
96 (Jeannerod, 2006; Lœvenbruck et al., 2018; Nalborczyk, 2019). Another example is the  
97 observation that inner speech is accompanied by an increase in tongue motor excitability,  
98 compared to rest or to an auditory speech perception condition (Maegherman et al., 2020).  
99 If the dual stream prediction model is correct in that the primary motor cortex is  
100 “bypassed” during inner speech, neither inner speaking nor inner hearing should increase  
101 the involvement of the primary motor cortex. In contrast, if the motor control view is  
102 correct in that the primary motor cortex is involved (but actively inhibited by cortical and  
103 subcortical mechanisms), inner speaking should be accompanied by an increase in motor  
104 cortex excitability.

105 Transcranial magnetic stimulation (TMS) has been used to probe the involvement of  
106 the motor system in speech production and perception (for a review, see Devlin & Watkins,  
107 2007). Single TMS pulses can be applied to the primary motor cortex to elicit motor

108 evoked potentials (MEPs) in the orofacial muscles. MEPs can be recorded using surface  
109 electromyography (EMG). Their amplitude depends on the state of the motor system: it is  
110 greater during muscle contraction compared to rest. Therefore, MEP amplitude provides a  
111 direct measure of motor excitability that can be used to examine the involvement of the  
112 motor system during speech production and perception (Möttönen et al., 2014). For  
113 instance, seeing or hearing speech increases motor excitability in a content- and  
114 effector-specific manner, in the same way that observing hand movements does (e.g.,  
115 Fadiga et al., 2002; Watkins et al., 2003). Sub-cortically, however, MEPs recorded from the  
116 orofacial muscles and those recorded from the hand muscles originate from different motor  
117 pathways. Whereas muscles from the face are innervated by the corticobulbar pathway,  
118 muscles from the hand are innervated by the corticospinal pathway, with direct  
119 consequences on the shape and latency of MEPs. Indeed, MEPs recorded over the orofacial  
120 muscles peak around 10-15ms after the pulse, whereas MEPs recorded over the hand  
121 muscles peak around 20-25ms after the pulse, because (amongst other things) of the  
122 different lengths of the corticobulbar vs. corticospinal tracts (Adank et al., 2018;  
123 Maegherman et al., 2020; Möttönen et al., 2014).

124 We hypothesise that during inner speaking, sensory (e.g., auditory) percepts are  
125 mostly provided by a motor-to-sensory simulation, whereas during inner hearing, sensory  
126 percepts are mostly reconstructed from stored perceptual information. Therefore, inner  
127 speaking should be accompanied by an increase in motor cortex excitability evidenced by  
128 larger MEPs recorded over orofacial muscles. Conversely, inner hearing should be  
129 accompanied by a much reduced or even absent increase in motor cortex excitability  
130 (**hypothesis #1**). Our secondary hypothesis is that this increase in motor cortex  
131 excitability is content-specific, as it is usually observed in TMS studies of speech perception  
132 (e.g., Fadiga et al., 2002; Watkins et al., 2003). We predict that lip motor cortex  
133 excitability increases should be more pronounced when covertly speaking a syllable that  
134 should strongly recruit the lips (i.e., /bu/, hereafter referred to as a “rounded” syllable)

135 than when covertly speaking a syllable that should recruit the lips to a lesser extent (i.e.,  
136 /gi/, hereafter referred to as a “spread” syllable) (**hypothesis #2**).

## 137 **Methods**

138 In the *Methods* and *Data analysis* sections, we report how we determined our sample  
139 size, all data exclusions, all manipulations, and all measures in the study (Simmons et al.,  
140 2012). A pre-registered version of our protocol can be found on OSF:  
141 <https://osf.io/7kwv6/>.

## 142 **Ethics information**

143 The present research complies with the French national ethics regulation (agreement  
144 of CPP Sud Méditerranée I, ANSM national number 2017-A03614-49) and the Declaration  
145 of Helsinki. All participants provided informed consent and received a monetary  
146 compensation of 40€.

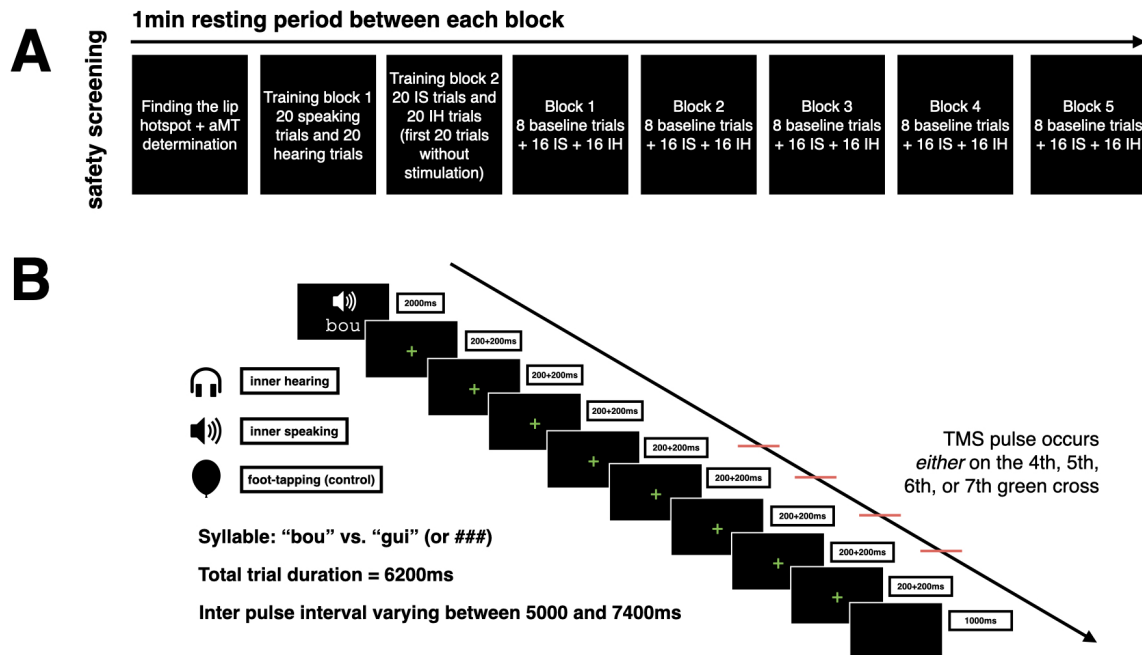
## 147 **Design**

148 The experimental design was fully within participants, with two crossed two-level  
149 factors: inner speech mode (inner speaking vs. inner hearing) and articulatory features  
150 (rounded vs. spread syllables). The experiment also included control trials during which we  
151 applied single-pulse TMS to assess motor cortex excitability while participants were  
152 performing an unrelated task. In these control trials, participants were asked to imagine  
153 tapping with their ipsilateral foot (i.e., to perform motor imagery of the foot; Figure 1).

## 154 **Procedure**

155 **Transcranial magnetic stimulation.** Participants were familiarised with TMS  
156 before the training session. We asked the participants to fill out a safety screening form.  
157 Participants with contraindications for TMS (e.g., neurological disorders, medication, a  
158 family history of epilepsy) were not included in the experiment. Afterwards, we explained





*Figure 1. A.* Experimental procedure. The main experimental part (post-training) involves 5 blocks of 40 trials each. aMT: active motor threshold, IS: inner speaking, IH: inner hearing. *B.* Illustrated timecourse of a single trial.

159 the details of the experiment to the participant and obtained their written informed  
160 consent.

161 To ensure similar levels of muscle activation throughout the experiment, we trained  
162 the participants to maintain a baseline activity between 20 and 30% of their maximum  
163 voluntary contraction. To find the location of the motor lip representation on the  
164 contralateral primary motor cortex, we followed the protocol described in Möttönen et al.  
165 (2014). We first localised the motor hand representation with the hotspot method, that is,  
166 by looking for the cortical site that elicits the maximal MEPs at a given intensity. Then,  
167 we localised the lip hotspot from the hand one, by keeping a minimal 5-sec break between  
168 TMS pulses.

169 The active motor threshold was defined as the minimal intensity necessary to elicit a

170 lip MEP with a minimum peak-to-peak amplitude of approximately  $50\mu\text{V}$  (as in Watkins  
171 et al., 2003; Watkins & Paus, 2004) in five stimulations out of ten (Rothwell et al., 1999).  
172 The intensity of the stimulator was then set to approximately 120% of the active motor  
173 threshold during the experimental session (this threshold was adjusted on a per-participant  
174 basis to maintain comfort throughout the experiment, cf. supplementary materials). As in  
175 Maegherman et al. (2020), we used a figure-of-eight coil placed around a  $45^\circ$  angle relative  
176 to the sagittal plane, inducing a posterior-to-anterior current flow, approximately  
177 perpendicular to the lateral fissure. The position of the TMS coil relative to cortex was  
178 continuously tracked and maintained throughout the experiment using a neuronavigation  
179 system (Navigation Brain System, Nexstim, Helsinki, Finland). A standard MRI image  
180 was used for the neuronavigation. All TMS pulses were monophasic and generated by a  
181 Magstim 200 device. The average active motor threshold was of 59.87% (ranging from 46%  
182 to 74%,  $\text{SD} = 7.83$ ) (expressed as a percentage of maximum stimulator output).

183 **Surface electromyography.** EMG activity of the (right section of the) orbicularis  
184 oris (OO) muscle and the abductor pollicis brevis (APB) muscle of the right hand was  
185 recorded bipolarly with surface electrodes connected to a 16-channel amplifier (BrainAmp  
186 ExG with eight bipolar and eight auxiliary channels, Brain Products Company, Gilching,  
187 Germany) at a sampling rate of 5kHz. Using alcohol, we cleaned the skin on the right  
188 section of the OO, above the belly of the APB muscle, as well as the skin around the site of  
189 the ground electrode placed on the right temple. We then attached electrodes on these  
190 sites. We visually checked the recorded EMG signals. If the signals appeared to be noisy,  
191 we re-cleaned the skin and re-attached the electrodes. After data collection, we computed  
192 the area-under-the-curve (AUC) of the rectified EMG signal. The covered area was taken  
193 as an index of the MEP size (Maegherman et al., 2020). In each trial, the MEP AUC was  
194 computed in a window spanning from 8 to 35ms after the TMS pulse. As in Maegherman  
195 et al. (2020), a pre-pulse section of 27ms (i.e., from 35 to 8ms before the pulse) was  
196 extracted to allow a posteriori checks of equivalent baseline contraction across conditions.

197       **Training blocks.** Training was composed of two blocks. In the first block,  
198 participants were trained to either produce (overtly) or to listen to a syllable for a 2-sec  
199 period at a 2.5Hz pace (20 hearing and 20 speaking trials; Figure 1). First, a syllable was  
200 presented on screen for 1 sec. In speaking trials, participants had to repeatedly utter this  
201 syllable at 2.5Hz for 2 sec. The 2.5Hz pace was cued by the display of a green cross on the  
202 screen. In hearing trials, participants were asked to listen to recordings of two native French  
203 speakers uttering the target syllables at a 2.5Hz pace. The syllables were chosen to induce  
204 a stronger involvement of the orbicularis oris muscle or of the zygomaticus muscle (i.e.,  
205 /bu/ vs. /gi/, respectively). At the end of this first training block, participants were given  
206 earplugs to reduce the discomfort caused by the TMS click sound (Counter et al., 1991).

207       In the second training block, participants were familiarised with the  
208 phenomenological contrast between inner speaking and inner hearing conditions. In the  
209 inner speaking conditions, participants had to imagine speaking the syllables “in their  
210 mind” without moving the speech effector and without producing any sound (as in Tian et  
211 al., 2016). In the inner hearing conditions, participants had to recreate in their minds the  
212 voice from the hearing trials used in the first training block, while also minimising the  
213 feeling of movement in their speech effectors (as in Tian et al., 2016).

214       It should be noted that Tian and Poeppel (2012), Tian and Poeppel (2013), and Tian  
215 et al. (2016) use different definitions of inner speaking and inner hearing than Hurlburt et  
216 al. (2013). For the former, inner speaking refers to the act of silently (mentally) talking to  
217 oneself from the first perspective, with one’s own perceived voice, whereas inner hearing  
218 refers to the act of imagining hearing speech, produced with the voice of someone else  
219 (sometimes designated as auditory verbal imagery). When defined in these terms, the  
220 distinction between inner speaking and inner hearing may be considered as the “speech  
221 analogue” of the distinction between first-person and third-person motor imagery in the  
222 motor imagery literature. As noted by Alderson-Day and Fernyhough (2015), however, this

223 operationnalisation of the phenomena of inner speaking and inner hearing makes it difficult  
224 to distinguish between the influence of the perspective and the distinction between inner  
225 speaking and inner hearing per se. While we acknowledge this limitation, we decided to use  
226 this operationnalisation of inner speaking and inner hearing in order to compare the results  
227 of the present study to those of previous studies (Tian et al., 2016; e.g., Tian & Poeppel,  
228 2012, 2013).

229 **Experimental protocol.** The timing of trials was identical across conditions  
230 (Figure 1). In each trial, the target syllable (i.e., /bu/ vs. /gi/, written as “bou” vs. “gui”),  
231 together with a pictogram indicating whether the participant should generate inner  
232 speaking, inner hearing, or foot-tapping imagery, was displayed for 2000ms. We used a  
233 pictogram that does not directly refer to the effector targeted by the TMS pulse to avoid  
234 automatic activation of the corresponding effector (see Figure 1). Then, a green cross  
235 appeared at the center of the screen every 400ms (i.e., at a 2.5Hz pace) for 3200ms.  
236 Participants were instructed to start producing the syllable at the appearance of the first  
237 green cross, and to keep repeating it in rhythm with the appearance of the green cross (i.e.,  
238 every 400ms). In all three task conditions, the TMS pulse exactly occurred exactly on the  
239 fourth, fifth, sixth, or seventh green cross. We decided to vary the occurrence of the TMS  
240 pulse to maintain the participant’s attention on the task throughout the experiment and to  
241 reduce the predictability of the pulse’s occurrence. At the end of each trial, the last (i.e.,  
242 eighth) green cross was followed by a blank screen presented for 1000ms. This procedure  
243 resulted in inter-pulse-intervals varying between 5000ms and 7400ms.

244 The main experimental part (post-training) consisted of five blocks of 40 trials each,  
245 yielding a total of 200 trials/MEPs per participant (40 control trials, 80 inner speaking  
246 trials: 40 /bu/ and 40 /gi/, and 80 inner hearing trials: 40 /bu/ and 40 /gi/). The order of  
247 trials within each experimental block was randomised across participants. Each block was  
248 followed by a 1min resting period. Finally, participants had to fill out the Movement  
249 Imagery Questionnaire-3 Second French version (Robin et al., 2020). The experimental

250 procedure was developed using the PsychoPy software (Peirce et al., 2019). Participants  
251 were then fully informed about the theoretical rationale for the study and compensated for  
252 their participation.

### 253 **Sampling plan**

254 To define the number of participants, we conducted a Bayesian a priori power  
255 analysis, where “statistical power” is to be understood in its general meaning, that is, the  
256 probability of achieving some statistical goal (Kruschke, 2015). We simulated data (see the  
257 supplementary materials for more details) by varying the number of participants (from 20  
258 to 50) and the number of trials in each condition per participant (30 vs. 60). We were  
259 interested in the probability of detecting two effects: i) the main effect of the inner speech  
260 mode, that is, the difference between the average MEPs in the inner speaking vs. inner  
261 hearing conditions (60 trials in each condition), and ii) the effect of the type of syllable,  
262 that is, the difference between the average MEPs for /bu/ vs. /gi/ trials *within* the inner  
263 speaking condition (30 trials in each condition).

264 The null hypothesis (i.e., no difference between conditions) requires more  
265 observations to be corroborated than the alternative hypotheses of small, medium, or large  
266 effects (e.g., Schönbrodt et al., 2017; Schönbrodt & Wagenmakers, 2018). Therefore, we  
267 decided to plan for a null effect to obtain a conservative estimate of the number of  
268 observations and participants needed to detect all sorts of effects, from null to large. This  
269 analysis revealed that, with 30 or more observations per participant and per condition, we  
270 needed at least 30 participants to reach a probability equal or superior to 0.9 of obtaining a  
271 Bayes factor (BF) equal or superior to 10 in favour of the null hypothesis. The detailed  
272 resulting power curve and the reproducible code used to conduct this analysis are available  
273 in the online supplementary materials.

## 274 **Participants**

275 In accordance with our power analysis, we recruited 31 French-speaking  
276 undergraduate students in Psychology from Aix-Marseille University, ranging in age from  
277 18 to 27 years ( $M = 20.42$ ,  $SD = 1.85$ , 29F, 2M), with no reported history of psychiatric or  
278 neurological disorder, speech disorder, or hearing deficit.

## 279 **Analysis plan**

280 All analyses were conducted in R (R Core Team, 2021). We fitted several Bayesian  
281 multilevel models using the `brms` package (Bürkner, 2017) with two categorical predictors  
282 (recoded using sum contrasts as -0.5 vs. 0.5): the inner speech mode (i.e., inner speaking  
283 vs. inner hearing), the syllable (i.e., /bu/ vs. /gi/), and the AUC of the lip MEP as a  
284 continuous dependent variable (for an introduction to Bayesian multilevel modelling, see  
285 Nalborczyk et al., 2019). For each effect of interest, we report the mean of the posterior  
286 distribution along with its 95% credible interval, as well as the mean of the posterior  
287 distribution of the standardised mean difference (Cohen's  $d$ ) with its 95% credible interval,  
288 when appropriate (i.e., when comparing two conditions). We also report the Bayes factor  
289 (BF), which quantifies the relative support (evidence) for either the null or the alternative  
290 hypothesis (e.g., Wagenmakers, 2007). Data collection and analysis were not performed  
291 blind to the conditions of the experiments.

292 We tested for differences in baseline background contraction across conditions per  
293 participant. If this test revealed a difference (i.e., a  $BF_{10} > 10$ ), we applied the common  
294 range correction (as in Spieser et al., 2013; method adapted from Schieppati et al., 1996).  
295 This method consists in removing trials below the maximum value of minimum values  
296 across conditions and trials above the minimum value of maximum values across conditions,  
297 per participant. After removing these trials, we then tested again for a difference. If there  
298 still was a difference in baseline across conditions, we removed this participant from the  
299 subsequent analyses. If there was no difference across conditions, and if there were at least

300 20 trials per condition left for this participant, we kept it in the subsequent analyses. If the  
301 common range procedure removed more than 20 trials per condition and participant, we  
302 applied another procedure in which all trials are kept, consisting in including the  
303 background contraction level as a continuous predictor in the statistical models.

304 It should be noted that we finally opted for the latter approach, which gave similar  
305 results as the former one, with the advantage of not discarding the data crossing an  
306 arbitrary threshold, and therefore resulting in more precise estimates. More precisely, we  
307 added the (per-participant) level of background EMG activity (its standardised AUC) as  
308 both a fixed and a random effect in the model (cf. the model formula on page 5 of the  
309 supplementary materials, reproduced in a simplified format below) (the detailed code is  
310 also available in the Rmarkdown file of the manuscript): `post ~ 1 + mode * syllable +`  
311 `pre + (1 + mode * syllable + pre | participant)`. Given this formulation, estimates  
312 of the model should be interpreted at the per-participant average level of background EMG  
313 contraction (i.e., the `pre` variable). In other words, estimates from this model can be  
314 interpreted in the following way: “Given (conditionally on) the level of background EMG  
315 contraction, what is the (additional/remaining) effect of `mode`, `syllable`, and their  
316 interaction `mode:syllable`?”. For completeness, analyses performed using the common  
317 range procedure are also reported in the online supplementary materials.

318 The first MEP in each block was removed because it is usually much larger than  
319 those that follow. As noted by Möttönen et al. (2014), it is not always possible to record  
320 robust lip MEP when the lips are “relaxed”. Therefore, we pre-registered that we would  
321 report the number of participants in which the experiment could not be carried out (i.e.,  
322 participants for which the stimulation was uncomfortable and participants in which we  
323 could not elicit MEPs). Fortunately, this did not happen and no participant was discarded  
324 from the analyses for this reason.

## Results

This section is divided into two parts. First, we present results from confirmatory (preregistered) analyses, aiming to test the difference in MEP amplitude between inner speaking and inner hearing (**hypothesis #1**) and the difference between the /bu/ and /gi/ syllables in the inner speaking conditions (**hypothesis #2**). Second, we present results from exploratory (non-preregistered) analyses, including an assessment of inter-individual differences in the two effects of interest, analyses of the relation between these effects and self-reported motor imagery skills, and analyses of the cortical silent period.

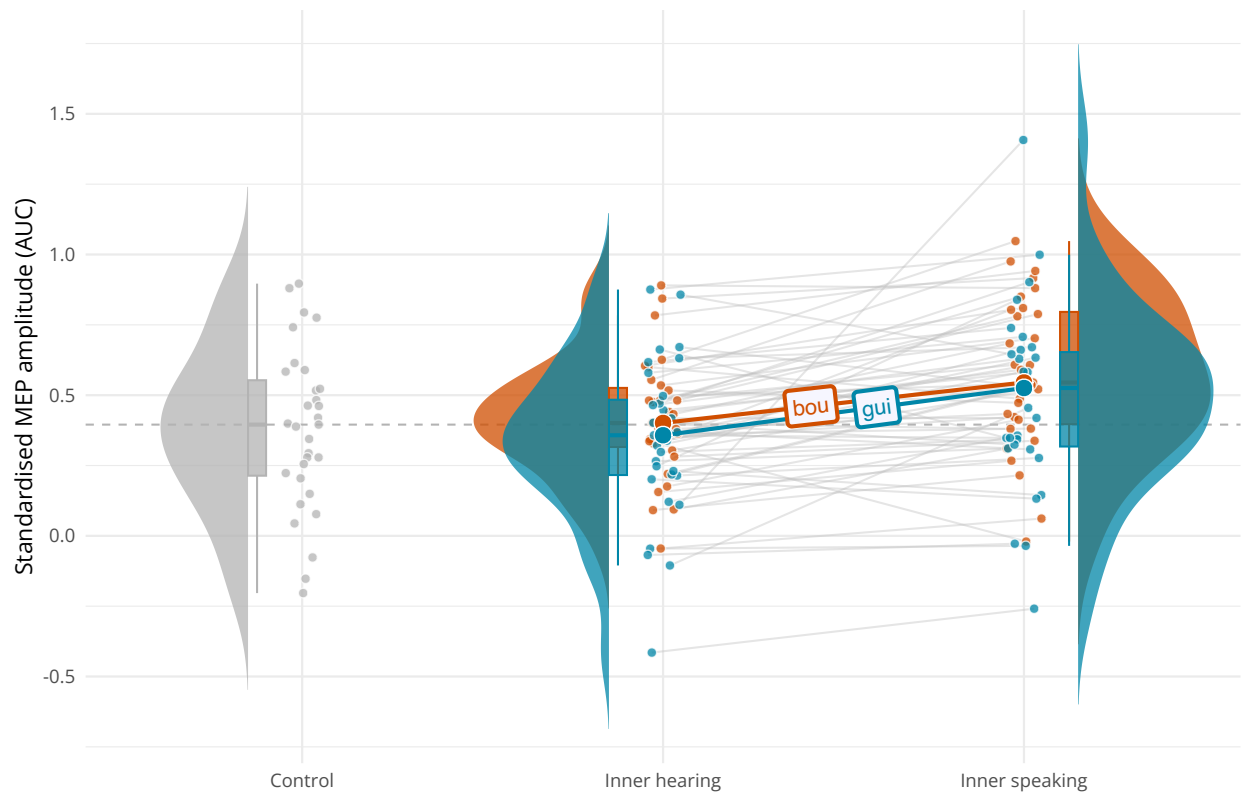
As predicted, inner speaking was associated with larger MEPs than inner hearing (hypothesis #1), and the mental production of the /bu/ syllable was associated with larger MEPs than the mental production of the /gi/ syllable in the inner speaking condition (hypothesis #2). However, this syllable effect was not specific to inner speaking (i.e., there was no evidence for an interaction effect between inner speech mode and syllable). Using a model comparison approach, we further demonstrated that these two effects were in the same direction in *all* participants.

### Confirmatory (preregistered) analyses

Before moving to the statistical results, we represent the distribution of standardised MEP amplitudes across conditions in Figure 2. This figure shows that the MEP amplitude recorded in the two inner hearing conditions was at similar levels as those recorded in the control condition. Conversely, MEPs were larger in the two inner speaking conditions. Moreover, MEPs were larger for the /bu/ syllable than for the /gi/ syllable.

To estimate these effects while accounting for the skewness of the collected data (for more details, see the online supplementary materials), we fitted a multilevel Skew-Normal model. The Skew-Normal distribution is a generalisation of the Normal distribution with three parameters  $\xi$  ( $\xi$ ),  $\omega$  ( $\omega$ ), and  $\alpha$  ( $\alpha$ ) for location, scale, and skewness





*Figure 2.* Average standardised MEP amplitude across conditions. Each dot represents a participant’s median AUC computed across 40 trials per condition. The dashed grey horizontal line represents the group’s median AUC in the control (imagined foot-tapping) condition.

350 (shape), respectively. Estimates from this model regarding the location parameter are  
 351 reported in Table 1.

352 First, notice that the effect of background (i.e., pre-pulse) EMG activity (i.e., the **pre**  
 353 variable) was strongly positive ( $\beta = 0.799$ , 95% CrI [0.695, 0.907],  $BF_{10} = 10 \times 10^{17}$ ),  
 354 indicating that, on average, higher levels of background EMG activity were associated with  
 355 larger MEPs. This phenomenon is well known and stresses again the importance of  
 356 including the level of background EMG activity in the model when estimating the effect of  
 357 the other variables of interest (here, the effect of inner speech mode, the effect of the

Table 1

*Estimates from the multilevel Skew-Normal model regarding the location parameter*

Predictor	Estimate	SE	Lower	Upper	Rhat	BF <sub>10</sub>	BF <sub>+</sub>
mode	0.103	0.025	0.054	0.151	1.000	53.476	59999.000
syllable	0.078	0.024	0.032	0.124	1.000	5.645	1845.154
pre	0.799	0.054	0.695	0.907	1.000	$10 \times 10^{17}$	$\infty$
mode:syllable	0.033	0.052	-0.070	0.136	1.000	0.064	2.861

*Note.* The 'Estimate' column represents the estimated group-level effect (slope) of each predictor included in the model (in terms of standardised AUCs). The 'Lower' and 'Upper' columns contain the lower and upper bounds of the 95% CrI, whereas the 'Rhat' column reports the Gelman-Rubin statistic. The last two columns report the BF in favour of the alternative hypothesis (relative to the null) and the directional (i.e., one-sided) BF, respectively.

358 syllable, and their interaction).

359 Regarding hypothesis #1 (i.e., the difference between inner speaking and inner  
 360 hearing), this analysis revealed that MEPs were larger in the inner speaking than in the  
 361 inner hearing conditions ( $\beta = 0.103$ , 95% CrI [0.054, 0.151],  $\text{BF}_+ = 59999$ ) and larger for  
 362 the /bu/ than for the /gi/ syllable ( $\beta = 0.078$ , 95% CrI [0.032, 0.124],  $\text{BF}_+ = 1845.154$ ).<sup>3</sup>  
 363 There was weak evidence in favour of a null interaction effect ( $\beta = 0.033$ , 95% CrI [-0.07,  
 364 0.136],  $\text{BF}_{10} = 0.064$ ).

365 Regarding hypothesis #2 (i.e., the difference between the /bu/ and /gi/ syllables in  
 366 the inner speaking conditions), a contrast analysis revealed that there was strong evidence

<sup>3</sup> For one-sided hypotheses,  $\text{BF}_+$  represents the ratio of the posterior probability of the effect being positive and the posterior probability of the effect being negative.

367 for larger MEPs during inner speaking of the /bu/ syllable than during inner speaking of  
368 the /gi/ syllable ( $\beta = 0.094$ , 95% CrI [0.035, 0.154],  $\text{BF}_+ = 213.286$ ).

### 369 **Exploratory (non preregistered) analyses**

370 In this section, we report the results of exploratory (i.e., non-preregistered) analyses.  
371 With these analyses, we aimed at assessing i) the variability of the observed effects across  
372 participants, ii) the impact of self-reported motor imagery abilities, and iii) potential  
373 differences between conditions in the duration of the cortical silent period (CSP), taken as  
374 an index of intracortical inhibition.

375 **Inter-individual differences.** Although group-level effects were small, they were  
376 remarkably stable across participants. We followed a model comparison approach that  
377 incorporates various constraints into Bayesian multilevel models (Haaf & Rouder, 2017;  
378 Rouder & Haaf, 2019) to test whether the estimated (true) effects were in the same  
379 direction for all participants (see also Van Geert et al., 2022, for a recent application).  
380 More precisely, we compared the evidence for a model that does not place any constraints  
381 on the participants' true effect (hereafter the "unconstrained" model) with the evidence for  
382 a model that constrains true participants' effect to have a particular sign (hereafter the  
383 "positive effects" model). The Bayes factor comparing the likelihood of the observed data  
384 under these two models was 127.25 (inverse  $\text{BF} = 0.008$ ) for the effect of inner speech  
385 mode, indicating that the observed data were 127.25 more likely under the positive effects  
386 model than under the unconstrained model.

387 The Bayes factor comparing the likelihood of the observed data under the positive  
388 effects model and under the unconstrained model was 132.47 (inverse  $\text{BF} = 0.008$ ) for the  
389 effect of the syllable, indicating that the observed data were 132.47 more likely under the  
390 positive effects model than under the unconstrained model. The Bayes factor comparing  
391 the likelihood of the observed data under the positive effects model and under the  
392 unconstrained model was 9.426 (inverse  $\text{BF} = 0.106$ ) for the interaction effect, indicating

393 that the observed data were 9.426 more likely under the positive effects model than under  
394 the unconstrained model.

395 Figure 3 illustrates individual-level estimates of each effect based on the previously  
396 described Skew-Normal model. Overall, these analyses suggest that *all* individuals show  
397 the same effects of inner speech mode and syllable: inner speaking led to larger MEPs than  
398 inner hearing, and mentally producing the /bu/ syllable led to larger MEPs than mentally  
399 producing the /gi/ syllable.

400 **Impact of self-reported motor imagery abilities.** To assess the impact of  
401 self-reported motor imagery abilities, we created a set of additional regression models  
402 containing either the score on each of the MIQ subscale (i.e., the internal perspective score,  
403 the external perspective score, or the kinaesthetic score) or the total score. We then  
404 compared these models using the Widely Applicable Information Criterion (WAIC,  
405 Watanabe, 2010), a generalisation of the Akaike information criterion (Akaike, 1974). The  
406 WAIC provides a relative measure of predictive accuracy of the models (it is an  
407 approximation of the out-of-sample deviance) and balances underfitting and overfitting by  
408 sanctioning models for their complexity (Burnham et al., 2011; Burnham & Anderson,  
409 2002; Hegyi & Garamszegi, 2011). These analyses revealed that the model with the lowest  
410 WAIC (i.e., the most parsimonious model) was the model without any MIQ score,  
411 suggesting that self-reported motor imagery abilities did not affect the previously described  
412 effects (see the online supplementary materials for code details).

413 **Analyses of the cortical silent period.** The cortical silent period (CSP) refers  
414 to a period of TMS-induced reduction in the EMG activity of a voluntarily contracting  
415 muscle (for review, see Hupfeld et al., 2020). The duration of the CSP is obtained by  
416 measuring the time interval between the offset of the MEP and the restoration of EMG  
417 activity. Overall, the duration of the CSP is considered to reflect the levels of slow  
418 metabotropic postsynaptic GABA<sub>b</sub>-mediated inhibition, occurring within the primary  
419 motor cortex (Cardellicchio et al., 2020; Hallett, 2007; Moezzi et al., 2018; Werhahn et al.,

420 1999). Crucially, intracortical inhibition has been suggested as one of the mechanisms  
 421 preventing motor execution during motor imagery (for review, see Guillot et al., 2012).

422 To examine whether our different manipulations induced different levels of  
 423 intracortical inhibition, we analysed the effect of inner speech mode (inner speaking  
 424 vs. inner hearing) and the effect of the syllable to be produced mentally (/bu/ vs. /gi/) on  
 425 the duration of the CSP (for more details on the determination of the CSPs' duration, see  
 426 the online supplementary materials). To estimate these effects, we fitted a multilevel  
 427 Log-Normal regression model to the CSPs' durations. Estimates from this model are  
 428 reported in Table 2. Overall, all effects were small and more likely to appear under the null  
 429 hypothesis than under the alternative hypothesis, suggesting that levels of intracortical  
 430 inhibition did not differ across conditions.

Table 2

*Estimates from the multilevel Log-Normal model*

Predictor	Estimate	SE	Lower	Upper	Rhat	BF <sub>10</sub>
mode	0.203	0.195	-0.171	0.590	1.003	0.142
syllable	0.132	0.189	-0.232	0.515	1.003	0.099
mode:syllable	0.025	0.377	-0.714	0.767	1.003	0.172

*Note.* The 'Estimate' column represents the estimated effect (slope) of each predictor included in the model. The 'Lower' and 'Upper' columns contain the lower and upper bounds of the 95% CrI, whereas the 'Rhat' column reports the Gelman-Rubin statistic. The last column reports the Savage-Dickey BF in favour of the alternative hypothesis (relative to the null).

## Discussion

431  
432 We investigated the neural processes involved in two forms of inner speech: inner  
433 speaking and inner hearing. Based on previous observations and on predictions of the dual  
434 stream prediction model (Tian et al., 2016; Tian & Poeppel, 2012, 2013), we assumed that  
435 inner speaking relies more strongly on a motor simulation mechanism than inner hearing.  
436 Based on the neurocognitive model of inner speech production developed in Løevenbruck et  
437 al. (2018) and Grandchamp et al. (2019), we hypothesised that the motor simulation  
438 mechanism underlying inner speaking would be indexed by increased levels of cortical  
439 excitability during inner speaking as compared to inner hearing (hypothesis #1). Given  
440 the involvement of motor simulation during inner speaking, we further hypothesised that  
441 the increase in cortical excitability during inner speaking should reflect the phonetic  
442 features of what is said (simulated) mentally (hypothesis #2).

443 Overall, these predictions were corroborated by our data. Even when controlling for  
444 pre-pulse EMG activity, we observed that inner speaking was associated with larger MEPs  
445 than inner hearing (hypothesis #1), and that the mental production of the /bu/ syllable  
446 was associated with larger lip MEPs than the mental production of the /gi/ syllable in the  
447 inner speaking condition (hypothesis #2). However, this syllable effect was also present  
448 during inner hearing (it was only slightly stronger in the inner speaking condition, cf. Table  
449 1), although the average MEP amplitude during inner hearing was not different from the  
450 average MEP amplitude in the control condition. We further observed that the two effects  
451 of interest pointed in the same direction for *all* participants (Figure 3). These results  
452 provide explicit constraints for current models of inner speech production.

453 Our results are compatible with the distinction between inner speaking and inner  
454 hearing, as postulated by the dual stream prediction model. The stronger increase in  
455 cortical excitability during inner speaking than inner hearing supports the involvement of a  
456 motor simulation mechanism during inner speaking, whose role would be to provide the

457 sensory content of inner speech (e.g., the inner voice). The stronger involvement of the  
458 primary motor cortex during inner speaking is consistent with the observation that inner  
459 speaking is associated with a stronger perceptual reactivation in auditory cortices (Tian et  
460 al., 2016). Whereas the syllable effect was stronger during inner speaking, it was  
461 nonetheless present (non-null) during inner hearing ( $\beta = 0.061$ , 95% CrI [0.005, 0.117],  
462  $BF_+ = 25.625$ ). This result suggests that the motor simulation stream may also be  
463 solicited during inner hearing, but to a lesser extent than during inner speaking (consistent  
464 with the results obtained by Tian et al., 2016).<sup>4</sup> By contrasting inner speaking and inner  
465 hearing of non-speech sounds, Chu et al. (2023) recently showed a clear dissociation in the  
466 involvement of the motor-based and memory-based networks, further supporting the  
467 distinction between these two processes. Overall, our results are also compatible with the  
468 framework recently provided by Pratts et al. (2023), in which inner speech can be  
469 generated by two separate mechanisms similar to those postulated by the dual stream  
470 prediction model, according to the intentionality and egocentricity constraints of the task.

471 The dual stream prediction model grants a secondary role to the primary motor  
472 cortex for inner speech, as it is considered to be “bypassed” during inner speech (Tian et  
473 al., 2016; Tian & Poeppel, 2012, 2013). Our results contradict this view by showing that  
474 the primary motor cortex is involved during inner speaking and that this involvement is  
475 modulated by phonetic features. In contrast, our results are compatible with the models  
476 developed in Løevenbruck et al. (2018) and Grandchamp et al. (2019), as well as other  
477 models of motor imagery, such as Grush (2004), in which the role of the primary motor  
478 cortex during inner speech is to issue motor commands from which predictions of sensory  
479 consequences can be subsequently computed (leading to the rich multisensory content of  
480 inner speech). However, the observed increase in M1 excitability could be due to various

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<sup>4</sup> A more trivial but unverifiable interpretation of this result is that some trials contained inner speech performed in the incorrect mode. This would, as per our hypotheses, increase the average AUC in inner hearing trials, or decrease it in inner speaking trials, or both.

481 reasons, for example strategically performing the imagery tasks by subliminally executing  
482 the action. Moreover, our observations cannot exclude the possibilities of upper motor  
483 pathways mediating inner speaking. These questions can not be answered from the present  
484 data and could be targeted in subsequent studies. Nevertheless, the observed increase in  
485 M1 excitability revives a classic crucial issue referred to as “the problem of inhibition of  
486 execution” by Jeannerod (2001): Given the involvement of the motor system in providing  
487 the multisensory content of inner speech, how is it possible for inner speech not to lead to  
488 motor execution?

489         It has been suggested that the subthreshold involvement of the primary motor cortex  
490 may result from either a subliminal activation or from active inhibitory mechanisms  
491 counteracting a supraliminal activation. Crucially, both options require an explanation of  
492 how activity within the primary motor cortex is maintained under the execution threshold.  
493 Regarding the first mechanism, Bach et al. (2021) suggested that the motor (execution)  
494 threshold may be “upregulated” during motor imagery to prevent execution. How this  
495 would be achieved or implemented however, is not specified (Nalborczyk et al., 2023).  
496 Regarding the second mechanism, supraliminal activation of the motor system could be  
497 counterbalanced by parallel inhibitory signals (e.g., Berthoz, 1996; Bonnet et al., 1997;  
498 Jeannerod, 1994, 2001). Recent behavioural results obtained using an action-mode  
499 switching paradigm support the hypothesis of parallel inhibitory mechanisms operating  
500 during motor imagery (Rieger et al., 2017). By asking participants to rapidly alternate  
501 between imagined and executed movements, it is possible to measure switching costs or  
502 benefits when switching from imagery to execution or from execution to imagery. Overall,  
503 results from such studies show that motor imagery of hand movements slows down  
504 performance in the subsequent trial (Bart et al., 2021a, 2021b, 2021c; Rieger et al., 2017;  
505 Scheil & Liefoghe, 2018). Accordingly, we previously proposed that similar inhibitory  
506 mechanisms may also be at play during inner speech production to prevent the execution of  
507 speech acts (Grandchamp et al., 2019; Løevenbruck et al., 2018; Nalborczyk et al., 2022).



508       Speech production differs considerably from the simple hand movements which are  
509 often assessed in motor imagery studies. In particular, it requires the coordination and  
510 sequencing of many articulators in short timescales. Zhao et al. (2023) suggested that  
511 these peculiarities may require an additional (or alternative) cerebral network for inhibiting  
512 speech. Using high-density ECoG, they observed activity in the premotor cortex associated  
513 with speech stopping. Moreover, electrocortical stimulation over this area caused  
514 involuntary speech arrests, interpreted as an engagement of the inhibitory mechanisms  
515 implemented within this area (see also Silva et al., 2022). This would be consistent with  
516 recent models of inner speech production, in which parallel inhibitory mechanisms are  
517 assumed to be issued by the rostral part of the precentral gyrus (Løevenbruck et al., 2018)  
518 or the orbitofrontal cortex (Grandchamp et al., 2019). Although our data cannot decide  
519 between these possibilities, they provide preliminary evidence regarding the role of  
520 intracortical inhibition during inner speech. Our analyses of the CSP durations revealed  
521 that levels of intracortical inhibition did not differ across inner speech modes (i.e., inner  
522 speaking vs. inner hearing) nor across syllables (i.e., bou vs. gui). This result goes against  
523 the hypothesis of an increased *intracortical* (GABA<sub>b</sub>-mediated) inhibition during inner  
524 speaking. However, it should be stressed that it does not allow ruling out the involvement  
525 of other forms of inhibition, such as those involving GABA<sub>a</sub>-mediated intracortical  
526 inhibition or cortico-subcortico-cortical circuits. Further research should aim at clarifying  
527 how these multiple processes interact together to maintain the activity of the primary  
528 motor cortex below the execution threshold during inner speech. Examining how the  
529 interplay between excitatory and inhibitory inputs to the primary motor cortex is  
530 modulated in different forms of inner speech (for instance in dysfunctional inner speech  
531 such as rumination or auditory verbal hallucinations) and their precise neural  
532 implementation are important future directions.

533       In summary, the results we describe establish the differential involvement of the  
534 primary motor cortex in two different phenomenological experiences of inner speech,

535 suggesting that distinct neural processes can support the mental production of speech.  
536 Various forms of inner speech, such as inner speaking or inner hearing, selectively engage  
537 these processes, and their involvement can be probed using transcranial magnetic  
538 stimulation. These results stress the importance of examining different forms of inner  
539 speech to account for its variety.

#### 540 **Data availability**

541 Data are available via the Open Science Framework: <https://osf.io/7kwv6/>.

#### 542 **Code availability**

543 Supplementary materials are available via the Open Science Framework:  
544 <https://osf.io/7kwv6/>.

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#### 554 **Author contributions**

555 Conceptualisation: LN, ML, MB, LS, FXA; Data curation: LN, MB, VS, LS; Formal  
556 analysis: LN, LS; Funding acquisition: LN, ML, LS, FXA; Investigation: LN, LS;  
557 Methodology: LN, ML, MB, VS, LS, FXA; Project administration: LN, ML, MB, LS,  
558 FXA; Resources: LN, ML, MB, LS, FXA; Software: LN, LS, VS; Supervision: ML, MB,

559 LS, FXA; Validation: ML, FXA; Visualisation: LN; Writing - original draft: LN; Writing -  
560 review and editing: LN, ML, MB, LS, FXA.

561

### **Competing interests**

562

The authors declare no competing interests.

563

Table 3. Design table

Question	Hypothesis	Sampling plan	Analysis plan	Interpretation given to different outcomes
Does inner speaking induce a stronger increase in motor cortex excitability than inner hearing?	Hyp1: Inner speaking will be associated with larger MEPs (i.e., higher AUCs) than inner hearing.	We determined the sample size based on an a priori power analysis (cf. main text).	The outcome is the mean AUC, computed from the rectified MEP. We will fit a Bayesian multilevel linear regression model with inner speech mode as a binary predictor.	Bayes factors will be interpreted in a continuous way. However, to facilitate interpretation, the results will also be considered consistent with the hypothesis if $BF_{10} > 3$ . The results will be considered consistent with the null hypothesis if $BF_{10} < 1/3$ .
Is this increase specific to the content produced in inner speaking?	Hyp2: The inner production of the /bu/ syllable will be associated with larger MEPs than the inner production of the /gi/ syllable in the inner speaking condition.	Same as above.	The outcome is the mean AUC, computed from the rectified MEP. We will fit a Bayesian multilevel linear regression model with inner speech mode and the linguistic material as binary predictors. We will then inspect the difference between /bu/ and /gi/ syllables within the inner speaking condition.	Same as above.

564

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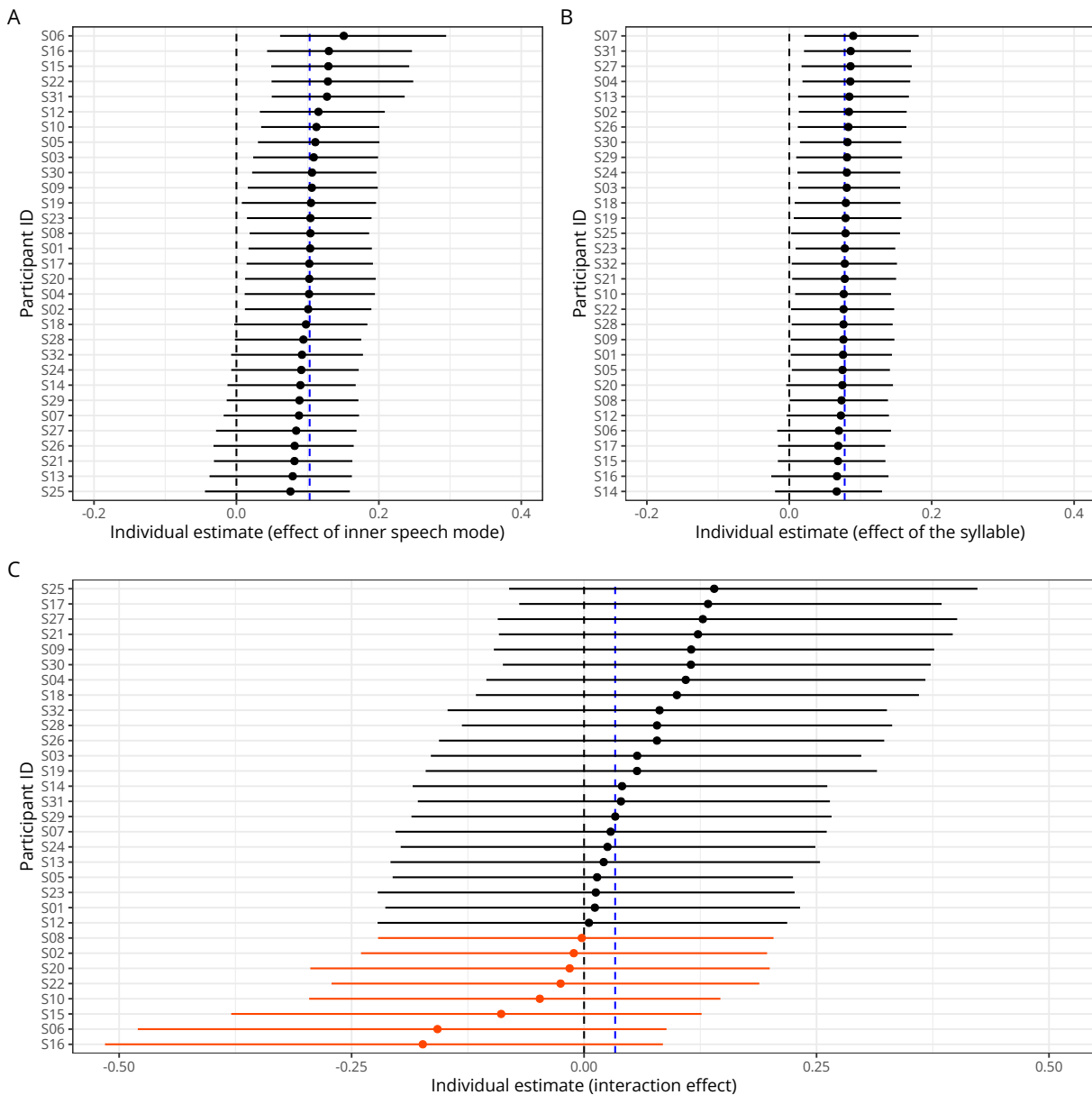
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*Figure 3.* Model-based estimates of each individual-level (i.e., 'random' or 'varying') effects, in descending order. Negative estimates are highlighted in orange. The vertical blue dashed line represents the average effect. Panel A: Individual-level effects of the inner speech mode (positive values being associated with larger MEPs during inner speaking). Panel B: Individual-level effects of the syllable (positive values being associated with larger MEPs when producing the /bu/ syllable). Panel C: Individual-level interaction effects between inner speech mode and syllable. Positive values are associated with a stronger syllable effect in the inner speaking condition (or symmetrically, a stronger effect of inner speech mode for the /bu/ syllable).