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TITLE PAGE

Title: “Inducing oculomotor plasticity to disclose the functional link between voluntary saccades and endogenous attention deployed peripherally”

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1 *ABSTRACT*

2 To what extent oculomotor and attention systems are linked remains strongly debated. Previous
3 studies suggested that saccadic adaptation, a well-studied model of oculomotor plasticity, and
4 orienting of attention rely on overlapping networks in the parietal cortex and can functionally
5 interact. Using a Posner-like paradigm in healthy human subjects, we demonstrate for the first
6 time that saccadic adaptation boosts endogenous attention orienting. Indeed, the discrimination of
7 perifoveal targets benefits more from central cues after backward adaptation of leftward
8 voluntary saccades than after a control saccade task. We propose that the overlap of underlying
9 neural networks actually consists of neuronal populations co-activated by oculomotor plasticity
10 and endogenous attention deployed perifoveally. The functional coupling demonstrated here
11 plaid for conceptual models not belonging to the framework of the premotor theory of attention
12 as the latter has been rejected precisely for this voluntary/endogenous modality. These results
13 also open new perspective for rehabilitation of visuo-attentional deficits.

14 1. INTRODUCTION

15 As much as we would like to, our brain is not able to deal with the huge amount of information
16 brought up by our senses. Especially when it comes to vision, albeit the dominant sense of
17 primates, our brain resources are too limited to efficiently handle visual information sensed by
18 the millions of photoreceptors of our eyes. Therefore, we need to select what part of space we
19 want to pay attention to. Visuospatial attention is a cognitive process which plays a critical role in
20 this selection by facilitating the visual processing of objects and features falling in the area of
21 space where it is focused on, at the expense of those situated outside^{1,2}. To get a refined and
22 homogenous analysis of our entire visual field, this attentional focus must be frequently re-
23 oriented either automatically, in response to the sudden appearance of a stimulus (exogenous
24 attention) or voluntarily, being driven by internal goals (endogenous attention)¹. These two
25 attention-shifting mechanisms are partially distinct, relying respectively on the ventral and dorsal
26 streams of attention^{3,4}, and both can either or not be accompanied by eye movements (overt and
27 covert shifts, respectively).

28 Saccadic eye movements are also of utmost importance to explore our visual environment and
29 select meaningful information therein. Indeed, as visual acuity is highest in the narrow central
30 zone of the visual field processed by the fovea, gaze shifts are mandatory to explore a visual
31 scene. Like attention shifts, gaze shifts are either exogenously or endogenously triggered,
32 corresponding to so-called reactive (RS) or voluntary saccades (VS), respectively. Shifts of
33 attention and saccadic eye movements share several other features, up to the point that, in the
34 framework of the premotor theory of attention, attention shifts are considered to be unexecuted
35 saccades inhibited at the oculomotor output level⁵.

36 Saccadic adaptation (SA) is a well-studied sensorimotor adaptation process ^{see 6,7 for reviews} and
37 therefore constitutes a convenient tool to assess the role of the oculomotor system on spatial
38 attention. Interestingly, in human, the neural substrates of SA and of visuospatial attention
39 overlap. Indeed, the intraparietal sulcus (IPS) has been involved in both adaptation of VS^{8,9} and
40 endogenous attention ^{see 3 for review} while the right temporo-parietal-junction (rTPJ) has been
41 involved in adaptation of RS^{8,10,11,12} and exogenous attention³. Moreover, two behavioral studies
42 have suggested that this overlap might have functional consequences: the first reports increased
43 performances in a visual detection task performed after adaptation of RS¹³, and conversely, the
44 second shows that RS adaptation efficiency is increased when subjects are simultaneously
45 engaged in an attention-demanding task directed to the saccade target¹⁴. Note, however, that the
46 visual detection task used by Habchi et al¹³ did not allow to specifically isolate covert attention
47 shifts from the other cognitive or motor components involved. In addition, and to the best of our
48 knowledge, the coupling between SA and attention has never been investigated in the
49 endogenous modality. As the premotor theory of attention has been challenged for the
50 voluntary/endogenous modality ^{see 15 for review}, highlighting a functional link between oculomotor
51 plasticity and endogenous attention would have strong theoretical implications.

52 Therefore, the present study aimed at investigating the coupling between saccades and visuo-
53 spatial attention in the endogenous modality, using a Posner-like paradigm allowing to
54 specifically assess pure covert attention shifts before and after the development of voluntary
55 saccades adaptation.

56

57 2. MATERIALS & METHODS

58 2.1. Subjects

59 The experiment adheres to the code of ethics of the World Medical Association – Declaration of
60 Helsinki (2008) and received the approval of the Ethics Committee of INSERM (CEEI - IRB
61 00003888, n°16-305). Forty-one subjects provided a written informed consent before performing
62 the tasks and received a compensation for their participation. Among those subjects, four were
63 excluded because they did not show significant saccadic gain modulation in one of the two
64 adaptation exposures and one was excluded because of poor discrimination performances (for
65 details see paragraphs 2.3.1.2 and 2.3.2.1)The remaining subjects were all right-handed except
66 one, comprised 17 males and 19 females, with a mean age of 25.5 +/- 4.53 SD (Standard
67 Deviation). Their vision was normal or corrected-to-normal. Criteria of exclusion were:
68 neurological or psychiatric disorders history; cognitive disorders preventing the comprehension
69 of the instructions; severe sleep deprivation during the last 24 hours; consumption of
70 psychotropic drugs, substances, or alcohol during the last 24 hours; participation to other
71 experiments involving sensorimotor adaptation during the last week. After written consents
72 obtained, each subject was assigned pseudo-randomly to one of the six sub-groups of each
73 experiment, corresponding to the 6 possible orders of testing in the three sessions (within-subject
74 design, see General Design section). The number of subjects was determined from a power
75 analysis performed through the G*Power software¹⁶ and based on parameters established from
76 the literature and from pilot data (see Power analysis in the Supplementary Methods).

77

78 2.2. Apparatus, stimuli and procedure

79 2.2.1. Apparatus

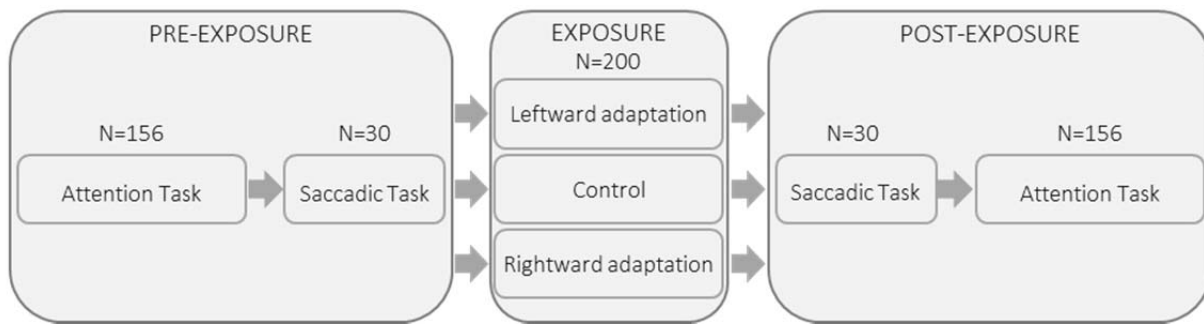
80 Experiments were carried out in a dimly lit room. Subjects were installed in a comfortable
81 position with the head stabilized by a chin-rest, cheekbone rests, and forehead support; they faced
82 a computer screen (1920 x 1080 pixels; 53.5 x 34.5 cm; 144 Hz refresh rate) at 57 cm from their
83 eyes. Experiments are timed based on the 144 Hz refresh rate of the computer display (frame
84 duration approximately 7 ms), therefore all time-intervals reported in the following represent
85 multiple of the frame duration and are rounded to the nearest value in milliseconds. Psychopy¹⁷,
86 an open-source software, was used for the stimuli presentation and data collection in all different
87 tasks. Movements of the right eye were recorded at a frequency of 1000 Hz using the remote
88 configuration of the EyeLink 1000 infrared eye-tracker (SR research, Canada). Each task started
89 with the calibration of the eye-tracker by asking subjects to fixate a series of 5 targets displayed
90 near the borders and at the center of the screen.

91 2.2.2. General design

92 Experiments 1 and 2 were carried out separately in two different experimental groups. The two
93 experiments were identical except for the eccentricity of the target in the attention task (see
94 paragraph 2.2.4). In each experiment, subjects were submitted to three experimental sessions
95 (within-subjects design), each of which ('leftward adaptation', 'rightward adaptation' and
96 'control') comprising identical pre-exposure and post-exposure phases as well as a specific
97 exposure phase (Fig. 1). During all three exposures, saccades in both directions were performed.
98 In the leftward adaptation, only leftward saccades were adapted; conversely in the rightward
99 adaptation, only rightward saccades were adapted; finally in the control, no saccades were
100 adapted. This control session allowed assessment of unspecific effects of exposure to a saccadic
101 task. The effects on attention were measured by comparing, between the pre- and post-exposure
102 phases of each session, subjects' performance in a visual discrimination attention task; in

103 addition, comparing the gain of saccades measured during a test saccade task performed before
 104 and after exposure allowed us to check for successful saccadic adaptation in the respective
 105 hemifields. The delay between each session was at least 14 days in order to avoid any retention of
 106 saccadic adaptation between sessions, based on a previous study disclosing a significant retention
 107 of adaptation up to 5 days after exposure but not 11 days after¹⁸.

108



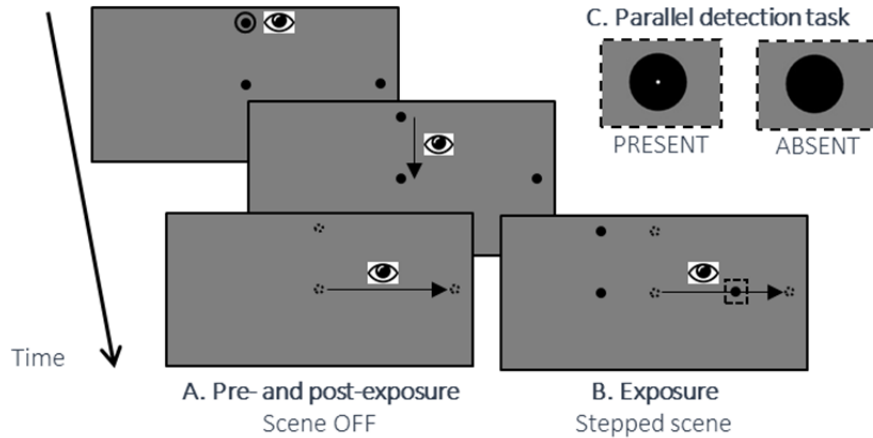
109

110 **Figure 1:** Study general design. In both Experiments 1 and 2, each subject underwent 3 experimental
 111 sessions -composed of a pre-exposure, an exposure and a post-exposure phases - differing only by the
 112 exposure phase (either leftward adaptation, rightward adaptation or control). N = number of trials.

113

114 2.2.3. Saccadic tasks

115 The saccadic adaptation was performed by a modified version of the double-step paradigm
 116 introduced by McLaughlin (1967). This paradigm consists in displacing the visual scene while
 117 the subject is executing a saccade towards a peripheral target. Thanks to the saccadic suppression
 118 phenomenon, this intra-saccadic visual displacement is usually not consciously perceived by
 119 subjects and leads to a mismatch between post-saccadic eye fixation and target location which is
 120 interpreted by the central nervous system as a saccade aiming error.



121

122 **Figure 2: Time-line of a trial in the saccadic tasks (not to scale).** After the circle around the fixation
 123 point turns off, subjects had to make, at their own pace, a downward saccade to the central point and then
 124 a horizontal –voluntary– saccade to the peripheral target. **A.** In the pre- and post- saccadic phases, the
 125 visual scene was turned off as soon as the voluntary saccade was detected. Subjects were instructed to
 126 keep looking at the peripheral target position for ~ 1 sec and then look back to the upper location in
 127 anticipation of the fixation point re-appearance, using that time period to blink if necessary. **B.** In the
 128 exposure phase, the visual scene was shifted backward immediately at the voluntary saccade onset
 129 (adapted saccades) or after 805 ms (unadapted saccades). The scene remained for 1610 ms in total in
 130 both conditions. The size of the shift increased progressively across blocks 1-4 (respectively 1°, 2°, 3° and
 131 3°). **C.** Enlarged view of peripheral targets during the exposure phase: subjects additionally performed a
 132 simple detection task to favor a sustained motivation: they had to report by a push button the presence of
 133 a small white dot inside the peripheral target (visible only in perifoveal vision after the saccadic response:
 134 see enlarged views of a dot-present target and of a dot-absent target). Feedback regarding this simple
 135 detection task was given at the end of each block.

136

137 Sequence of events for adapted saccade trials (Fig. 2B). Three dots of 0.3° of visual angle were
 138 displayed on the computer screen. The first dot was located 4° above the center of the screen, and

139 was surrounded by a small circle. The second dot was at the center of the screen. The third dot,
140 the peripheral target, was at 9° of eccentricity aligned with the horizontal meridian, either to the
141 left or to the right. The side of the peripheral target was blocked with 12 trials in the adapted
142 direction, 12 in the opposite direction, repeated 2 times for each block. The subject had to fixate
143 the upper dot during a pseudo randomized delay between 301 ms and 701 ms after which the
144 disappearance of the surrounding circle ('go signal') indicates that he/she had to look
145 successively at the other two targets. Correct eye fixation of the upper dot was ensured by
146 continuous monitoring of the eye-tracker signal. In the next 2000 ms, the subject had to make at
147 her/his own pace, a first saccade towards the central dot (vertical saccade) and then a second
148 saccade from there towards the peripheral target (horizontal voluntary saccade). The voluntary
149 saccade was detected when the eye velocity was higher than $70^\circ/\text{s}$ ²⁰. This event immediately
150 triggered the shift of the visual scene when the peripheral target was in the adapted hemifield
151 (Fig. 2B). The visual scene shift was progressively increased through the blocks (1° for the first
152 block, 2° for the second, 3° for the third and fourth blocks) leading to a progressive decrease of
153 the target final eccentricity (8° , 7° and 6° respectively). The visual scene remained visible for a
154 total of 1610 ms after the detection of the voluntary saccade. The subject then had a delay of
155 1000 ms to blink and look back to the upper dot. The next trial started as soon as correct fixation
156 of the upper dot location was detected.

157 Sequence of events for unadapted saccade trials. These trials were identical to the adapted
158 saccade trials except that the jump of the visual scene occurred 805 ms after the detection of the
159 voluntary saccade. These trials correspond to the saccades toward the unadapted hemifield for the
160 leftward and rightward exposure and for the saccades toward both hemifields in the control
161 exposure.

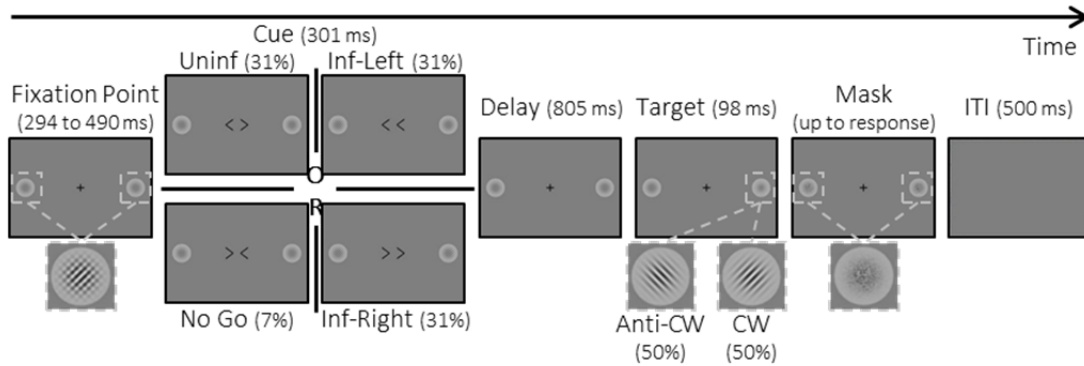
162 The total exposure phase consisted of 196 trials distributed in 4 blocks of 48 trials each (24 with a
163 right target and 24 with a left target). Between each block, the subject was allowed to rest with
164 the head still as long as needed.

165 To maximize subjects' involvement and motivation throughout the saccadic tasks, they were
166 requested to perform in parallel an easy detection task: in random trials (from 5 to 20 per block),
167 the peripheral target contained a white dot of 0.008° of visual angle (not detectable in peripheral
168 vision but easy to detect after the saccade to the target), and subjects had to push a button after
169 each trial in which they detected the white dot. Performance feedback was provided to subjects
170 during the rest period between the blocks but was not further analyzed.

171 Pre- and post-exposure saccadic tasks (Fig. 2A). These tasks were identical to the exposure tasks
172 except that the visual scene did not jump but instead was turned off at the initiation of the
173 voluntary saccade. Each task consisted in one block of 30 trials (15 with a right target and 15
174 with a left target, randomly ordered). Comparison between pre- and post-exposure tasks allowed
175 determination of the SA after-effect (change of saccade amplitude in post- versus pre-exposure)
176 and thus quantitative assessment of the adaptation strength.

177 2.2.4. Attention task: visual discrimination

178 A variant of the Posner task¹ was designed with the main features (a central cue, and a long SOA)
179 chosen to evoke shifts of endogenous attention. Contrasting between informative trials (cue
180 always valid) and uninformative trials (uninformative cue) allowed us to measure the pure benefit
181 of endogenous attention orienting. This approach was preferred over that used in many
182 endogenous attentional studies, consisting of contrasting between valid and invalid cues, which
183 rather yields the cumulated effect of exogenous costs and endogenous benefits^{21,22}.



185

186 **Figure 3: Time-line of a trial in the attention task.** A central fixation cross and 2 lateral placeholders
 187 (eccentricity: 7.5° in Experiment 1; 3° in Experiment 2) each containing 2 orthogonal gabors, were
 188 present at the beginning of the trial. Then central cues appeared for 301 ms, either indicating the side of
 189 the upcoming target (100% valid informative cue: Inf-Left or Inf-Right), or providing no spatial
 190 information (uninformative cue: Uninf), or indicating to restrain the response (No Go cue). The target
 191 presented after 805 ms of delay (SOA = 1106 ms) consists in the brief disappearance of one gabor on one
 192 side (left 50% or right 50%), followed after 91 ms by a mask. Using a push / pull device, subjects had to
 193 respond as fast and as accurately as possible whether the target was tilted clockwise (CW) or anti-
 194 clockwise (anti-CW). Experiments 1 and 2 differed only according to the eccentricity of the discrimination
 195 target and associated place-holder.

196

197 Sequence of events in the attention task trials (Fig. 3). A fixation cross subtending 1° of visual
 198 angle appeared at the center of the screen (grey 50%) at the beginning of the trial and, except
 199 during the cue period, remained visible until the subject's response. Subjects had to keep eye
 200 fixation on that location all throughout the trial. Two light grey (35%) placeholders (circles of
 201 2.5° of visual angle in Experiment 1; 1.5° in Experiment 2) were also presented along the
 202 horizontal meridian, on the left and on the right, at 7.5° of eccentricity in Experiment 1, and at 3°

203 of eccentricity in Experiment 2. Each placeholder initially contained two gabor patches
204 (Experiment 1: 4 cycles per degree (cpd) of spatial frequency and 2.5° of visual angle;
205 Experiment 2: 4 cpd of spatial frequency and 1.5° of visual angle) presented with a Gaussian
206 mask and superimposed orthogonally (one gabor tilted at 45° and the other at -45° relative to the
207 vertical, leading to the perception of a grid). The contrast of the gabor patches was previously
208 determined for each individual by a staircase procedure to achieve a 80% level of correct
209 discrimination (see Staircase procedure in Supplementary methods). After a pseudo-randomized
210 (294 to 490 ms) delay from the beginning of the trial, a cue appeared for 301 ms. This cue was
211 composed of two empty arrows (1.5° vertically x 1° horizontally) flanking the center of the
212 screen (1.0° of horizontal spacing). For ~ two thirds of the trials (32 ‘informative trials’ out of 52
213 trials for each block) the cue validly informed the future target location: the two arrows both
214 pointed either toward the left or toward the right of the screen to indicate the placeholder in
215 which the target will appear. In 16 ‘uninformative’ trials (~one third), the cue did not provide any
216 spatial information about the upcoming target, the two arrows pointing outwards. The 1:2 ratio of
217 uninformative versus informative trials was meant to potentiate the cueing effect²³. In the four
218 remaining trials of each block, a ‘no-go cue’ represented by the two arrows pointing inwards
219 instructed subjects to refrain from answering. These ‘no-go’ trials were meant to enforce subjects
220 to use the cue to perform the task correctly, and thus favoring the conscious interpretation and
221 increasing the benefit of the cue. However, they were not analyzed. In all trials, the cue period
222 was followed first by displaying again the fixation point and then 805 ms after cue offset by a
223 brief extinction (98 ms) of one of the two gabor patches either in the left placeholder (50%) and
224 or in the right placeholder (50%): the remaining gabor patch thus constitutes the target (SOA =
225 1106 ms) which orientation had to be discriminated. Immediately after this target presentation, a
226 mask was displayed in the two placeholders until the subject’s response was made or for a

227 maximum duration of 1500 ms. Subjects had to discriminate as fast and as accurately as possible
228 whether the target gabor patch was tilted clockwise or anticlockwise (45° or -45° with respect to
229 the vertical, respectively). Subjects answered with their index finger through a double switch
230 device oriented in their mid-sagittal axis, with a response assignment randomized between
231 subjects: half of them pushed the switch for a “clockwise” target and pull it for an
232 “anticlockwise” target, the other half was instructed with the opposite assignment. Eye fixation
233 was continuously monitored all throughout the trial and whenever the subject broke fixation
234 (gaze deviating in any direction more than 1.5° from the fixation cross), the fixation cross
235 immediately turned red and the trial was aborted. Aborted trials were replayed back during the
236 same block of trials. We chose a SOA duration of 1106 ms in order to minimize any involvement
237 of attention oriented exogenously²⁴. Moreover, the pilot data reported in Supplementary data
238 showed that the duration of the SOA does not affect the validity effect in our discrimination task.

239 The task consisted of 3 blocks of 52 trials each (156 in total): 16 ‘informative - left target’ and 16
240 ‘informative - right target’, 8 ‘uninformative - left target’ and 8 ‘uninformative - right target’, and
241 4 ‘no-go cue’. Between each block, subjects received standardized feedback about their
242 performance (see Instructions and feedback in Supplementary methods).

243

244 2.3.Data analyses

245 Data analyses were performed with the open-source software R (The R Core Team, 2013). These
246 analyses concerned the saccadic behavior during the pre- and post-exposure saccadic tasks as
247 well as the performances in the attention tasks measured by cue benefit (relative change of
248 reaction time – RT – between informative and uninformative trials). Any exclusion of a subject

249 due to criteria described in the following paragraphs led to his/her replacement. All the group
250 analyses have been carried out separately for the two experiments.

251 2.3.1. Saccadic tasks

252 2.3.1.1. Preprocessing

253 Eye movement data were analyzed off-line using custom software developed in Matlab (Math
254 Works Inc., Natick, MA, USA). The beginning of the primary horizontal saccade was identified
255 offline based on a velocity threshold of 30°/s. Saccadic amplitude was measured as the difference
256 between eye positions 50 ms before the saccade onset and 50 ms after the saccade offset. The
257 gain of a saccade was used as the dependent variable in the saccadic tasks: it was computed as the
258 ratio between saccadic amplitude and initial target eccentricity (difference between target
259 position and starting position of the saccade). Saccades with a gain less than 0.5 or outside the
260 mean $\pm 2SD$ interval were discarded from further analysis.

261 2.3.1.2. Statistical analysis

262 Since the saccadic adaptation was critical to test our hypothesis, we excluded from the main
263 analysis subjects who did not show the expected decreased gain of saccades in the adapted
264 hemifield. To this aim, we first performed, separately for each subject and each hemifield, a
265 unilateral Student t-test comparing the gain of the saccades between the pre- and the post-
266 saccadic tasks and used a threshold p-value of 0.05 after FDR (False Discovery Rate²⁵) correction
267 for 6 multiple comparisons. Moreover, for representational purposes, we computed the exposure
268 after-effect for each hemifield and each exposure condition as follow:

$$\text{Exposure after - effect}_{\text{exposure of interest}} = \frac{\text{mean gain}_{\text{post-exposure}} - \text{mean gain}_{\text{pre-exposure}}}{\text{mean gain}_{\text{pre-exposure}}}$$

269 A negative exposure after-effect reflects a decrease of the saccadic gain between the pre- and the
270 post-exposure phases.

271 Finally to calculate the effect size of the exposure after-effect in the exposure sessions, we
272 computed the mean of the gain for each subject, in the adapted hemifield for the pre-exposure and
273 the post-exposure phase separately. These values were used to calculate the Cohen's *d* effect size
274 for Student t test.

275 As the results to the attentional task in Experiment 2 revealed a significant effect of leftward
276 backward SA (see paragraph 3.2.2.2), we performed two supplementary analyses on saccadic
277 data of the leftward adaptation exposure.

278 First, to assess the effect of SA on voluntary saccades preparation time, we computed the fixation
279 time (FT) as the period of time between the end of the first saccade and the beginning of the
280 second (voluntary) saccade. In each subject, we computed the median FT for each saccade
281 direction and each phase of the exposure of interest, namely the backward adaptation of leftward
282 saccades. We then performed a rMANOVA on median FT with the saccade direction (leftward or
283 rightward), and the phase (pre- or post-exposure) as within-factors.

284 Second, since in Experiment 2, the eccentricity of the discrimination target corresponds to the
285 final size of the intra-saccadic step (ISS, 3°), we searched for any correlation between the cue
286 benefit and the post-saccadic error after leftward, adapted, saccades. Post-saccadic error was
287 measured as the distance between the saccade landing position and the jumped target. The
288 correlations were then computed separately for each block of exposure and one global correlation
289 on post-saccadic error across all blocks

290

291 2.3.2. Attention task

292 2.3.2.1. Preprocessing

293 To ensure that the level of involvement of each subject was high, subjects with low global
294 performance or with high fluctuations were excluded. To this aim, each session were divided in 8
295 experimental cells of conditions (2 cue types x 2 target hemifields x 2 phases, smallest cell = 24
296 trials). We excluded subjects with a number of correct trials inferior to 8 for any of these cells.
297 Then, trials with outlier RT were excluded using the John Tukey's method of leveraging the
298 Interquartile Range, and the median RT of the remaining trials was computed in each of these
299 cells. If one cell's median RT lies outside ± 3 SD (Standard Deviation) from the subject's average
300 of median RTs computed across the 8 cells, the subject was excluded.

301 We emphasize that only correct responses were considered in this analysis.

302 2.3.2.2. Outcome neutral criteria

303 First of all, a significant difference of RT between the informative trials and uninformative trials
304 in the pre-exposure phase was a prerequisite to demonstrate that, at the group level, our attention
305 task readily engaged the orienting of endogenous attention. For that purpose, a 2-way rmANOVA
306 was performed on RT of the pre-exposure phases only, with cue type as 2-level factor
307 (informative / uninformative) and exposure as 3-level factor (control, leftward and rightward
308 adaptation). The critical outcome neutral criterion was a main cue type effect and an absence of
309 significant interaction between cue type and exposure factors, which would allow us to
310 demonstrate a significant difference of RT during pre-exposure between informative trials and
311 uninformative trials, irrespective of the exposure session.

312 2.3.2.3. Statistical analysis

313 For this analysis, the dependent variable was the subjects' cue benefit on discrimination RT,
314 which was computed as follows:

$$Cue\ benefit_{exposure\ of\ interest} = \frac{RT_{Uninformative} - RT_{Informative}}{RT_{Informative}}$$

315 This dependent variable was averaged in each of the 12 experimental cells defined from the
316 factors of the following rmANOVA, and then submitted to this rmANOVA, with the target
317 hemifield (left or right), the phase (pre- or post-exposure) and the exposure (leftward adaptation,
318 rightward adaptation, or control) as within-factors.

319 Post Hoc analyses of significant interaction was performed using paired Student t-tests separately
320 for each of the three exposure conditions. The three p-values were then FDR corrected.

321 After highlighting an effect of leftward adaptation on cue benefit in both hemifields in
322 Experiment 2 (see paragraph 3.2.2.2), we wanted to address whether the beneficial effect of SA
323 on orienting of attention was due to a specific change in RT for informative trials relative to
324 uninformative trials. We thus performed, for the leftward adaptation exposure and separately for
325 informative and uninformative trials, a one-way rmANOVA on median RT with phase (pre- or
326 post-exposure) as the within subject factor.

327 Finally, we sought for a correlation (Pearson's product-moment correlation) between the after-
328 effect of leftward saccades adaptation (see formulae above) and the relative change of cue benefit
329 between the pre- and the post-exposure of leftward adaptation, calculated as follow:

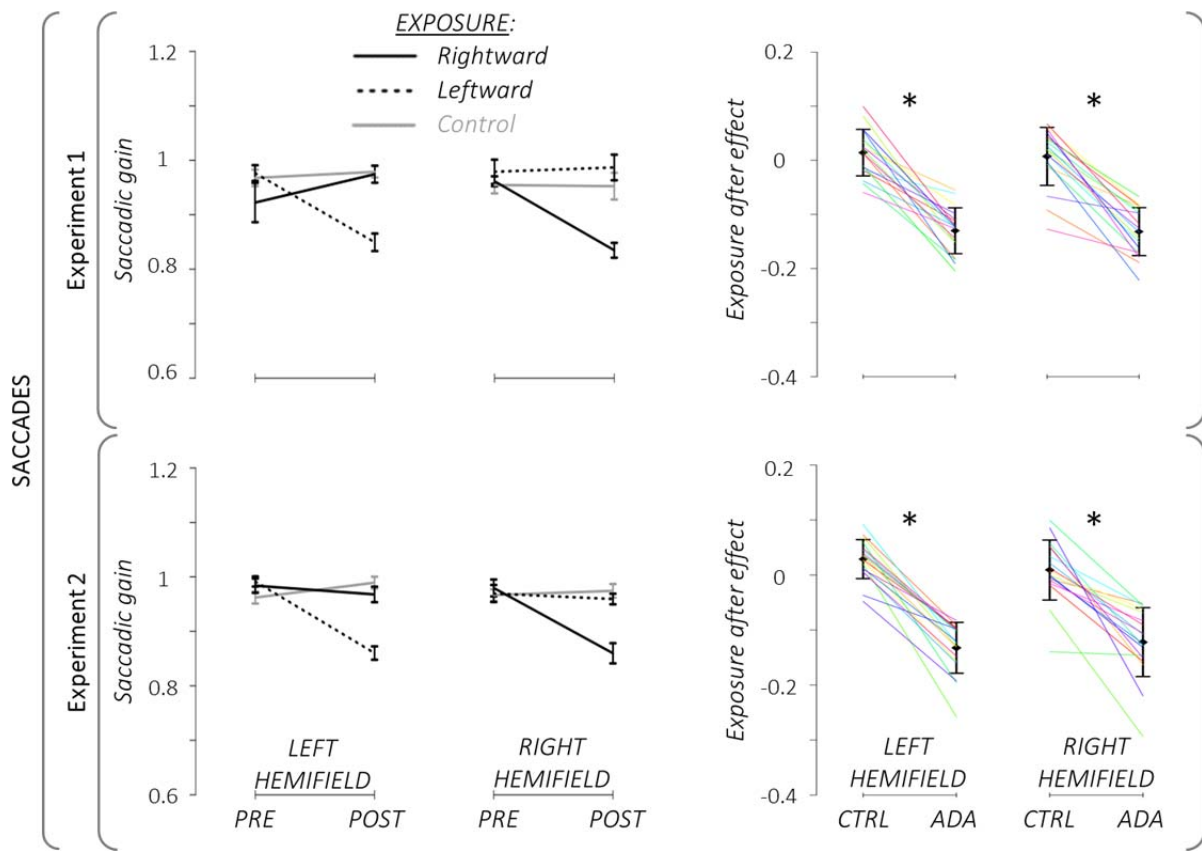
$$Relative\ Change_{Cue\ benefit} = \frac{Cue\ benefit_{post-exposure} - Cue\ benefit_{pre-exposure}}{Cue\ benefit_{pre-exposure}}$$

330

331 3. RESULTS

332 3.1. Pre- and post-exposure saccadic tasks

333 After rejection of subjects and trials following the above mentioned criteria (see paragraph
334 2.3.1.1 for details), the average number of analysed trials per condition was 13.6 +/- 1.3 SD in
335 Experiment 1 and 12.4 +/-1.7 SD in Experiment 2. The mean saccadic gain in pre- and post-
336 exposure, as well as the individual and mean adaptation after-effect, are illustrated in Figure 4.
337 As it was a pre-requisit (see paragraph 2.1), all subjects of each experiment showed in the
338 adaptation sessions a significant decrease of the saccadic gain for target presented in the adapted
339 hemifield, in the post-exposure as compared to the pre-exposure, thus having a significant after-
340 effect due to SA (Fig. 4, right panel). Moreover, as seen in Fig. 4 (left panel), this decrease was
341 not seen in the opposite, unadapted, hemifield, whether for the leftward or rightward exposure. In
342 addition, the amounts of adaptation in the adapted hemifields did not differ between the leftward
343 and rightward exposures, both for Experiment 1 (Cohen's $d = 0.04$; $t_{17} = 0.14$; $P = 0.89$) and
344 Experiment 2 (Student $t_{17} = 0.21$; $P = 0.84$, Cohen's $d = 0.05$). Finally, no gain change in either
345 hemifield took place in the control exposure.



346

347 **Figure 4: Pre- and Post-exposure saccadic task results.** *Left: Group mean (+/- SD) of saccadic gain for*
 348 *Experiment 1 (upper panel) and Experiment 2 (lower panel). **Black lines:** rightward adaptation exposure;*
 349 ***Black dotted:** leftward adaptation exposure; **Grey lines:** control exposure. **Right:** Individual percent gain*
 350 *changes between the pre- and the post-exposure tasks (after-effect) for Experiment 1 (upper panel) and for*
 351 *Experiment 2 (lower panel). Only data from the adapted hemifield are shown for adaptation exposures*
 352 *(ADA), i.e. left or right hemifield for adaptation exposure of leftward and rightward saccades,*
 353 *respectively; and values of the control exposure (CTRL) are plotted for each corresponding hemifield.*
 354 *Solid black lines represent group mean (+/- SD) and colored lines stand for individual values. *: p-*
 355 *value<0.05*

356

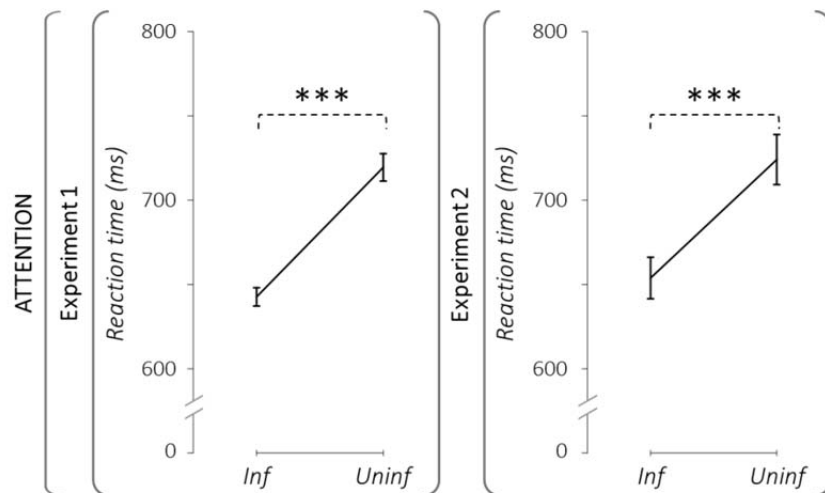
357 3.2. Attention task

358 3.2.1. Experiment 1 (target at 7.5°)

359 3.2.1.1. Outcome neutral criteria

360 After rejection of subjects and trials following the above mentioned criteria, the average number
361 of analysed trials per condition was 58.9+/- 5.6 SD (see 2.3.2.1 for details). The rmAnova of
362 outcome neutral criteria on the pre-exposure RT revealed a significant main effect of the cue type
363 (partial $\eta^2 = 0.81$; $F_{(1,17)} = 74.15$; $P = 1.32e^{-7}$; achieved power = 1; Fig. 5, left panel). The main
364 effect of the exposure was not significant (partial $\eta^2 = 0.01$; $F_{(2,34)} = 0.17$; $P = 0.85$), nor the
365 interaction between exposure and cue type (partial $\eta^2 = 0.01$; $F_{(2,34)} = 0.28$; $P = 0.76$). Therefore,
366 our attention task did engage the orienting of attention during the pre-exposure phase, and did so
367 similarly in the three sessions.

368



369
370 **Figure 5:** Effect of cue type on reaction time in the pre-exposure attention task. Group mean (+/- SD) of
371 median reaction times (ms) in Experiment 1 (left panel) and in Experiment 2 (right panel). A general

372 effect of cue type was disclosed by the decrease of RT for informative as compared to uninformative trials.

373 ***: p -value < 0.001

374

375 3.2.1.2. Statistical analysis

376 The performance in the attention task was evaluated by computing the cue benefit of subjects'

377 (Fig. 6). Submitting cue benefit to a rmANOVA with the factors exposure x phase x target

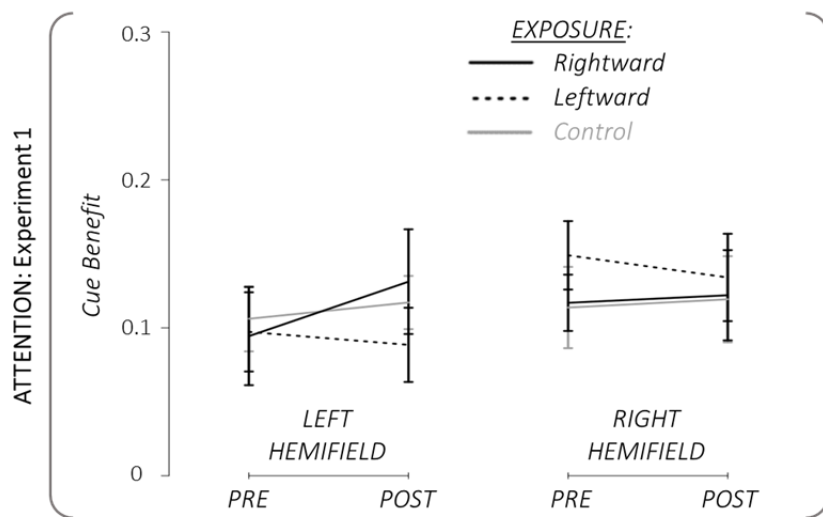
378 hemifield revealed no significant main effect and no significant double nor triple interaction (all

379 $P > 0.32$). Therefore, no further analysis was performed. In summary, no significant effect of

380 saccadic adaptation on attention performance could be revealed when the target was presented at

381 7.5°.

382



383

384 **Figure 6:** Pre- and Post-exposure attention results in Experiment 1. Group mean (+/- SD) of cue benefit

385 for the pre- and the post-exposure phases in the two hemifields of target presentation. **Black lines:**

386 rightward adaptation exposure; **Black dotted lines:** leftward adaptation exposure; **Grey lines:** control

387 exposure.

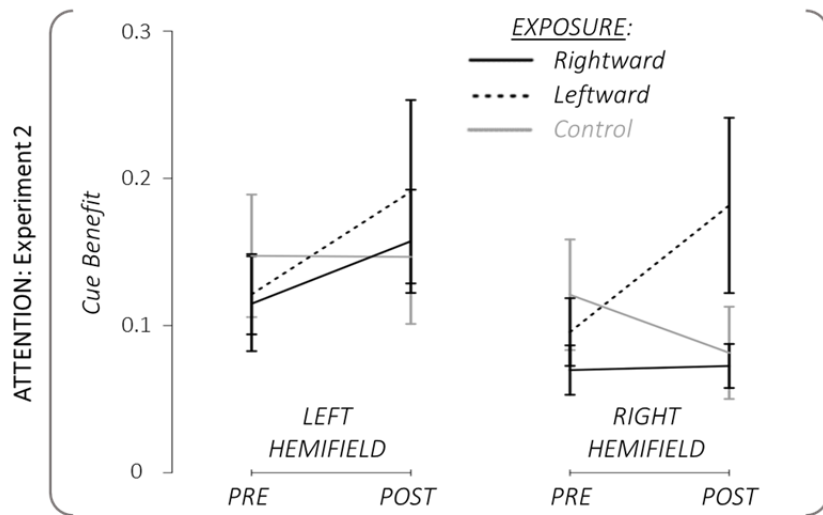
388

389 3.2.2. Experiment 2 (target at 3°)

390 3.2.2.1. Outcome neutral criteria

391 After rejection of subjects and trials following the above mentioned criteria, the average number
392 of analysed trials per condition was 57.0 +/- 7.4 SD (see 2.3.2.1 for details) . The rmAnova of
393 outcome neutral criteria on the pre-exposure RT revealed a significant main effect of the cue type
394 (partial $\eta^2 = 0.64$; $F_{(1,17)} = 30.81$; $P = 3.52e^{-5}$; achieved power = 1; Fig. 5, right panel). The main
395 effect of the exposure was not significant (partial $\eta^2 = 0.03$; $F_{(2,34)} = 0.44$; $P = 0.65$), nor the
396 interaction between exposure and cue type (partial $\eta^2 = 0.05$; $F_{(2,34)} = 0.81$; $P = 0.45$). Thus, as for
397 Experiment 1, the attention task in Experiment 2 engaged the orienting of attention during the
398 pre-exposure phase, and did so similarly in the three sessions.

399 3.2.2.2. Statistical analysis



400
401 **Figure 7: Pre- and Post-exposure attention results in Experiment 2.** Group mean (+/- SD) of cue benefit
402 for the pre- and the post-exposure phases in the two hemifields of target presentation. **Black lines:**

403 *rightward adaptation exposure; Black dotted: leftward adaptation exposure; Grey lines: control*
404 *exposure.*

405
406 As for Experiment 1, the performance in the attention task of Experiment 2 was evaluated by
407 computing the subjects' cue benefit (Fig. 7). The 3-factor rmANOVA (exposure x phase x target
408 hemifield) revealed no significant main effect (Phase: partial $\eta^2 = 0.10$; $F_{(1,17)} = 1.98$; $P = 0.18$;
409 Target hemifield: partial $\eta^2 = 0.11$; $F_{(1,17)} = 2.00$; $P = 0.18$; Exposure: partial $\eta^2 = 0.49$; $F_{(2,34)} =$
410 0.07 ; $P = 0.29$). The following interactions were not significant: double interactions (Exposure x
411 target hemifield: partial $\eta^2 = 0.09$; $F_{(2,34)} = 1.59$; $P = 0.22$; Phase x target hemifield: partial $\eta^2 = 0.05$
412 ; $F_{(2,34)} = 0.95$; $P = 0.34$), and the triple interaction (Exposure x phase x target hemifield: partial η^2
413 $= 0.05$; $F_{(2,34)} = 0.92$; $P = 0.41$). However, the double interaction exposure x phase was significant
414 (partial $\eta^2 = 0.18$; $F_{(2,34)} = 3.76$; $P = 0.03$; achieved power > 99%).

415 Post-hoc paired Student t-tests revealed that, irrespective of hemifield, the differences between
416 the pre- and the post-exposure phases for the control exposure and for the rightward adaptation
417 exposure did not reach significance ($t_{(35)} = 0.92$; $P = 0.36$; $t_{(35)} = -1.53$; $P = 0.13$; respectively). In
418 contrast, the exposure to leftward adaptation induced a significant difference between the pre-
419 and the post-exposure, yielding an increased cue benefit in both hemifields (from 0.11 to 0.19; 95
420 CI mean difference = [-0.14; -0.02]) after SA ($t_{(35)} = -2.56$; $P = 0.015$ (FDR- corrected $P = 0.045$);
421 Cohen's $d = 0.40$).

422 To identify the origin of the significant exposure x phase interaction, we further submitted RT to
423 2 one-way rmANOVAs, separately for informative or uninformative trials. The rmANOVA on
424 informative trials revealed a nearly significant effect of the phase (partial $\eta^2 = 0.20$; $F_{(1,17)} = 4.16$;
425 $P = 0.057$), whereas the rmANOVA applied to uninformative trials did not (partial $\eta^2 = 0.06$;

426 $F_{(1,17)} = 1.00$; $P = 0.33$). Therefore, the boosting effect of adaptation on discrimination speed is
427 mainly observed for informative trials, i.e. trials that most strongly elicited an endogenous
428 orienting of attention.

429 Concerning the link between the change in the left saccadic gain and the change in the cue benefit
430 (Figure in Supplementary), after leftward adaptation, we did not highlight a significant
431 correlation ($r_{(35)} = 1.30$; $P = 0.21$).

432 We then computed the post-saccadic target error during the exposure to leftward SA. Mean
433 values are plotted in Table 1 separately for each block of adaptation exposure and for the 4 blocks
434 pooled together.

Table 1: Mean and standard deviation (SD) across subjects of post-saccadic error in the exposure to backward adaptation of leftward saccades.

	Block 1	Block 2	Block 3	Block 4	All Blocks
Mean	0.42	0.99	1.64	1.35	1.09
SD	0.34	0.48	0.57	0.61	0.49

435

436

437 Table 2 summarizes the results of the correlation between this post-saccadic error parameter and
438 the cue benefit measured during the post-exposure discrimination task. None of the analyses
439 revealed any significant correlation, whether computed separately for the 4 blocks or for all
440 blocks together.

441 **Table 2** Rho coefficients (degree of freedom = 16) and p-values of the correlations between post-saccadic
442 error and cue benefit.

	Block 1	Block 2	Block 3	Block 4	All Blocks
$r_{(16)}$	-0.29	-0.34	-0.18	-0.10	-0.20
p-value	0.24	0.17	0.47	0.69	0.43

443

444 Finally we assessed as a potential attentional marker the preparation time of voluntary saccades

445 by measuring the fixation time between the first and second saccades: the overall median fixation

446 time is 271 ms with an interquartile range (IQR) of 122 ms (see details in Table 3). The

447 rmANOVA did not reveal any significant main effect (saccade direction: partial $\eta^2 = 0.02$; $F_{(1,17)}$

448 $= 0.30$; $P = 0.59$; phase: partial $\eta^2 = 0.02$; $F_{(1,17)} = 0.30$; $P = 0.59$) nor an interaction between the

449 two factors (partial $\eta^2 = 0.002$; $F_{(1,17)} = 0.03$; $P = 0.89$). Therefore, no significant effect of SA on

450 the fixation time preceding voluntary saccades could be demonstrated.

Table 3: Median and interquartile range (IQR) fixation time across subjects for the pre- and post-phase of the exposure to backward adaptation of leftward saccades and of rightward saccades.

	Leftward saccades		Rightward saccades	
	Pre-exposure	Post-exposure	Pre-exposure	Post-exposure
Median (ms)	269.75	272.75	275.75	266
IQR (ms)	117.25	138.5	97.125	117.75

451

452 In summary, the adaptation of leftward saccades resulted in significantly increased attention

453 performance when the target was presented at 3° in both the adapted and unadapted hemifield, but

454 without significant relationship with individual variations of the level of adaptation or of the

455 amount of post-saccadic target error.

456

457 4. DISCUSSION

458 The present study questioned the link between the oculomotor and visuospatial attention systems,
459 by testing the effect of sensorimotor plasticity of VS on covert endogenous orienting of attention.
460 Based on a within-subjects comparison between leftward adaptation, rightward adaptation, and
461 control exposure, we found the cueing effect on discrimination RT to increase specifically after
462 leftward adaptation for discrimination stimuli at 3° in either (adapted or unadapted) hemifields.
463 This boosting effect of SA was mainly related to a decreased RT for informative trials, i.e. those
464 that elicited an endogenous orienting of attention. No effect was observed on saccade preparation
465 time. These results demonstrate for the first time a boosting effect of oculomotor plasticity on
466 endogenous orienting of attention in healthy humans, deepening our knowledge of saccadic
467 adaptation mechanisms and providing evidence for shared neuronal representations for eye
468 movements and visuospatial attention.

469 As mentioned in the INTRODUCTION, a coupling between SA and covert shifts of attention has
470 been reported only once at the behavioral level in a previous study from our lab¹³. However,
471 contrary to the Posner-like paradigm used here, the detection task Habchi and colleagues¹³ used
472 could not entirely distinguish attention orienting from other potential cognitive or motor
473 components. In addition, they investigated exogenous attention orienting. Here we decided
474 instead to focus on the voluntary/endogenous modality, because it has been suggested to refute
475 the premotor theory of attention¹⁵. The present demonstration of a coupling in this latter modality
476 therefore provides a new piece of empirical argument in this debate.

477 Interestingly, despite these differences, in both Habchi and colleagues' study and ours, the
478 coupling was observed only after adaptation of leftward saccades. They interpreted this saccade
479 direction specificity as resulting from the known dominance of the right hemisphere in

480 controlling exogenous attention⁴, without making any assumption of hemispheric laterality for
481 saccadic adaptation, which is completely unknown. Indeed, in the relevant fMRI literature, only
482 cortical BOLD modulations after leftward SA have been investigated so far^{26,8,12}. A right
483 dominance interpretation of the saccade-direction specific coupling demonstrated here for the
484 voluntary/endogenous modality is not straightforward in this framework. However, TMS studies
485 have suggested that, although both left and right IPS play a role in voluntary orienting
486 visuospatial attention, the right hemisphere has a dominant contribution. Caposto and
487 colleagues^{27,28} reported that the disruption of the right IPS, and not the left IPS nor the right FEF,
488 led to a bilateral alpha band synchronization in the occipito-parietal cortex and therefore to a
489 decreased efficiency of target processing in both hemifields. Indeed, alpha synchronization and
490 desynchronization are known to index visual perception performance: the lower alpha power the
491 better the performances^{e.g. 29,30,31,32}. The impact of the right IPS disruption was also observed in
492 two studies^{33,34} using concurrent TMS/fMRI in which stimulation of right but not left posterior
493 parietal cortex caused changes of fMRI activity bilaterally in the occipital lobe. Thus, the
494 presently demonstrated effect of adaptation of leftward, but not rightward, VS fits in the
495 framework of a right hemispheric dominance in visuospatial attention. In addition, the benefit in
496 the two hemifields that we found in the attention task is consistent with the above mentioned
497 TMS studies. Indeed, it can be postulated that SA of leftward saccades, contrary to the disrupting
498 effect of TMS, increases brain excitability in the right IPS and therefore modulates neural
499 excitability in the occipital cortex bilaterally.

500 Other previous investigations of the link between SA and visuospatial attention have all focused
501 on the so-called pre-saccadic shift of attention, corresponding to an enhanced perception which
502 automatically occurs at the saccade target location just before saccade initiation³⁵. These studies

503 have shown that after saccadic adaptation, the spatial locus of highest perceptual performance
504 remains coupled with the saccade endpoint, not to the visual target^{36,37,38} (but see³⁹). In line with
505 the premotor theory of attention⁵, this observation reflects an adaptation-related change of a
506 prediction of saccadic commands, which is also consistent with the proposal that oculomotor
507 efference copy is modified after adaptation³⁸.

508 The present findings clearly point to a new oculomotor plasticity-visuospatial attention coupling
509 as compared to the studies mentioned above. First, the lack of significant correlation between the
510 adaptation rate and the cue benefit boost does not illustrate the metrical relationship found in
511 previous studies between saccade size and endpoint of pre-saccadic attention shift⁴¹. Together
512 with the specificity to a 3° eccentricity, this observation suggests an all-or-none effect restrained
513 to the peri-foveal part of the visual field. Second, we demonstrated an effect of SA on covert
514 shifts of attention, unrelated to any oculomotor preparation, as subjects always kept central
515 fixation throughout the attention tasks. Thus, possible changes of oculomotor efference copy are
516 unlikely to play any role in our experiments. Furthermore, the discrimination performance did not
517 change for a target at 7.5°, i.e. the eccentricity which matched best the adapted saccade endpoint.
518 Thus, the coupling we report is not related to the new metric of the adapted saccade, and not to
519 the adaptation field^{42,43}. Instead the boosting effect was actually found at the eccentricity of 3°
520 which corresponds to the size of the target intra-saccadic step (ISS) eliciting SA. This raises the
521 interesting possibility that it is the systematic exposure to the error signal driving SA, rather than
522 the oculomotor changes related to SA itself, which drives the changes in covert attention. Recall
523 however, that the same target jump and error signal were induced during the control exposure,
524 but 805 ms after the saccade, a delay which prevented SA to be elicited. Moreover, our analyses
525 failed to disclose any significant correlation between the post-saccadic error experienced during
526 the leftward adaptation exposure and the cue benefit measured during the post-exposure

527 discrimination task. Therefore the correspondence between the size of ISS and the eccentricity
528 for which the effect was found could be a mere coincidence. Further experiments would be
529 required to test this possibility. For example, one could induce adaptation of larger saccades with
530 larger target jumps and test whether the eccentricity where the boosting effect occurs changes
531 accordingly or remains in the peri-foveal part of the visual field.

532 Another possible explanation of this limitation to the peri-foveal part of the visual field is a SA-
533 induced compression of represented visual space (in case of backward adaptation) that would
534 shift the representation of visual stimuli toward the center of gaze. Indeed, Zimmermann and
535 Lappe^{39,45} showed that SA induces a shift of the subjectively-perceived location of objects
536 flashed before a saccade or during fixation, suggesting that spatial visual representations are
537 shaped by oculomotor planning^{46,47}. Consequently, when subjects have to localize (Zimmermann
538 and Lappe's) or discriminate (current study) such peri-foveal stimuli, they would both
539 underestimate the targets eccentricity and discriminate them with a faster reaction time. The
540 functional coupling between adaptation and attention, highlighted by the present results, strongly
541 suggests that the corresponding neural substrates overlapping at the macroscopic level (see
542 Introduction^{8,9,3}) actually host neuronal population co-activated for saccades and attention.
543 Although neuronal recordings in the monkey posterior parietal cortex have provided evidence for
544 distinct neuronal populations for orienting of attention and saccadic eye movements⁴⁸, other
545 studies have suggested that the monkey LIP hosts priority maps used both by attention and eye
546 movements to select targets⁴⁹. Therefore, we believe that SA acts on such 'common priority
547 maps', thereby transferring to covert attention mechanisms. Common priority maps for attention
548 and eye movements may have been implemented in the course of natural selection because
549 sharing neural substrates for cognitive functions is advantageous in terms of neural resource.
550 Accordingly, as mentioned above, we propose that the boosting effect would emerge from an SA-

551 induced increase of top-down signals from the right parietal cortex to the visual cortex of both
552 hemispheres. Indeed, there is increasing evidence that adaptation of leftward saccades relies on
553 metabolic activation in the right IPS^{50,8,12,9}. Second, during an endogenous attentional orienting
554 task, the right IPS send top-down signals to the visual cortices in both hemispheres to modulate
555 their excitability and therefore their readiness to process an upcoming stimulus ^{see 4 for review}. To
556 account for the observed boosting effect restricted to +/-3° eccentric targets, we further suggest
557 that the increased activity of the IPS is centered on the fovea, which is in accordance with the
558 oculocentric representation of visual space in the posterior parietal cortex⁵¹. The boosting of
559 attention we specifically observed after leftward SA for targets flanking the fovea bilaterally
560 seems to be related to the dominant role of the right IPS in the control of visual attention and to
561 its properties in representing the visual space. This specificity speaks for a functional link
562 between adaptation of voluntary saccades and endogenous visuospatial attention based on the
563 brain substrates common to these processes, rather than on a general increase in brain excitability
564 after SA.

565 The hypothesis of shared neural resource between adaptation and attention predicts the existence
566 of another functional coupling, opposite to that reported here, i.e. from attention to saccadic
567 adaptation. Indeed, some studies have suggested that attention shifts affect SA. Flashing in the
568 vicinity of a stationary saccade target a stimulus attracting exogenous attention, a perceptual
569 target⁵² or a salient visual distractor⁵³, is sufficient to induce SA. Further, McFadden et al.⁴⁶
570 showed that it is possible to adapt the exogenous shift of attention by ‘stepping the attentional
571 target’ during a covert attentional task, and that such ‘adapted attention’ transferred to saccades.
572 Finally, SA efficiency has been shown to increase with attentional load¹⁴. The hypothesis of
573 shared neural substrates between adaptation and attention also predicts that some neural changes

574 related to SA can be detected in the attentional task performed immediately after, akin to the
575 change of gamma band activity we could disclose recently, albeit in the exogenous/reactive
576 modality¹¹. A similar magnetoencephalographic study will be required to disclose whether the
577 coupling between adaptation of voluntary saccades and endogenous attention is subtended by an
578 increased brain activity, reflected in the gamma band, in the region of the right IPS of the dorsal
579 attention system.

580 Taken together, this study highlights a functional coupling between adaptation of voluntary
581 saccades and endogenous visuospatial attention. This finding provides deeper insight into the role
582 of the motor system in the updating of visual space representations, and leads toward promising
583 rehabilitation procedure for patients with visuospatial disorders.

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705 *AUTHOR CONTRIBUTIONS STATEMENTS*

706 DP, ABC and JN design the protocol, JN performed the experiment and the analysis. JN wrote
707 the main manuscript text and prepared figures. All authors reviewed the manuscript.

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