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2 **Perisaccadic and Attentional Remapping of Receptive Fields in**  
3 **Lateral Intraparietal Area and Frontal Eye Fields**

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37 coordinate transformation, compressive mislocalization.

38 **Summary**

39           The nature and function of perisaccadic receptive-field (RF) remapping have been  
40 controversial. We used a delayed saccade task to reduce previous confounds and  
41 examined the remapping time course in areas LIP and FEF. In the delay period, the RF  
42 shift direction turned from the initial fixation to the saccade target. In the perisaccadic  
43 period, RFs first shifted toward the target (convergent remapping) but around the time of  
44 saccade onset/offset, the shifts became predominantly toward the post-saccadic RF  
45 locations (forward remapping). Thus, unlike forward remapping that depends on the  
46 corollary discharge (CD) of the saccade command, convergent remapping appeared to  
47 follow attention from the initial fixation to the target. We modelled the data with  
48 attention-modulated and CD-gated connections, and showed that both sets of connections  
49 emerged automatically in neural networks trained to update stimulus retinal locations  
50 across saccades. Our work thus unifies previous findings into a mechanism for  
51 transsaccadic visual stability.

52

## 53 Introduction

54 Visual information enters the brain via the retina, which projects a point-to-point  
55 retinotopic map to the visual cortex via the lateral geniculate. The retinotopic map is  
56 maintained throughout a number of prestriate visual areas <sup>1,2</sup>. However, a retinotopic map  
57 is inadequate for spatially accurate perception, because eye movements change the retinal  
58 location of a given object in the world. Nonetheless the spatial perception of humans and  
59 monkeys is largely independent of gaze. A classic demonstration of the brain's ability to  
60 convert a retinotopic map into a spatially accurate map is the double-step task <sup>3,4</sup>.  
61 Subjects must make successive saccades to two flashed targets both of which disappear  
62 before the first saccade. The retinal and oculomotor vectors of the first saccade are  
63 identical. However, the first saccade creates a dissonance between the spatial and  
64 retinotopic locations of the second saccade target, and the brain must compensate for this  
65 dissonance. Helmholtz <sup>5</sup> theorized that the brain used the motor signal for the first  
66 saccade to update the visual representation and create a spatially accurate visual signal  
67 for perception and action. Duhamel et al <sup>6</sup> showed that Helmholtz's theory has a  
68 physiological correlate. When monkeys fixate, a given object in space occupies the RF of  
69 a neuron, the current RF (cRF). When monkeys make a saccade, the saccade will move  
70 the RF to a new spatial location, the future RF (fRF), even though the object's spatial  
71 location has not changed. Neurons in the lateral intraparietal area (LIP) respond to a  
72 stimulus in the fRF even before the saccade, effecting a forward shift of the RF in the  
73 direction of the saccade. Because the shift starts before the eye moves, the signal causing  
74 the shift must arise from a motor signal feeding back to the sensory system, a  
75 phenomenon now known as corollary discharge (CD). This forward shift is found in  
76 many brain areas, including the superior colliculus (SC) <sup>7</sup>, the frontal eye fields (FEF) <sup>8</sup>,  
77 V3 <sup>9</sup>, and the parietal reach area <sup>10</sup>. In LIP the perisaccadic visual responses are not

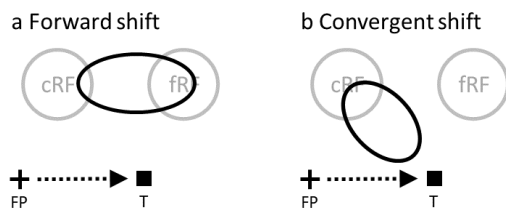


Fig. 1. Perisaccadic RF remapping found in LIP, FEF, and other brain areas, drawn in a screen reference frame. The cross, square and arrow represent the fixation point (FP), saccade target (T), and saccade vector, respectively. cRF and fRF refer to a cell's current (pre-saccadic) and future (post-saccadic) RFs, respectively. In each panel, the region(s) enclosed by black oval represent perisaccadic RF. (a) Forward shift in the saccade direction. (b) Convergent shift toward the target.

limited to the cRF and fRF. Instead, stimuli positioned in any of the spatial locations across which the saccade will sweep the retinal RF will evoke a response before the saccade (Fig. 1a). Stimuli closer to the fRF evoke larger responses with longer latencies than stimuli closer to the cRF <sup>11</sup>. Forward remapping has been postulated to provide a key mechanism for the transsaccadic maintenance of a spatially accurate signal <sup>12</sup>. Without forward remapping the brain's representation of the visual world would be inaccurate for at least 45 ms <sup>13</sup>, and behaviorally relevant visual signals inaccurate for 90 ms <sup>14</sup> after every saccade.

The original remapping studies only probed a cell's responses at a few positions. To measure perisaccadic RFs more completely, an influential study <sup>15</sup> sampled FEF cells' responses from an array of

98 spatial positions. The study found that around saccade onset, RFs shift toward the  
 99 saccade target (Fig. 1b), instead of toward the fRFs. The conclusion was that this  
 100 convergent shift is a substrate of attention to the saccade target, unrelated to the  
 101 maintenance of a spatially accurate signal across saccades, despite the  
 102 neuropsychological evidence. The study argued that the forward shift may be an artifact  
 103 of under-sampling the probe locations. However, the methods used caused a number of  
 104 confounds: 1) It is well known that both the abrupt onset of visual stimuli<sup>16</sup> and the  
 105 saccade target<sup>17</sup> evoke attention, and RFs shift toward the locus of attention<sup>18</sup>. Most  
 106 remapping studies, including that of Zirnsak et al, use the onset of the saccade target as  
 107 the saccade go signal, and thus could not separate the effect of the attention evoked by  
 108 the target onset from that of the saccade CD on the RF shifts. 2) The study integrated  
 109 neuronal activities from 50 to 350 ms after the probe onset. This large time window must  
 110 average attentional and CD effects together, and the strong attention from the target onset

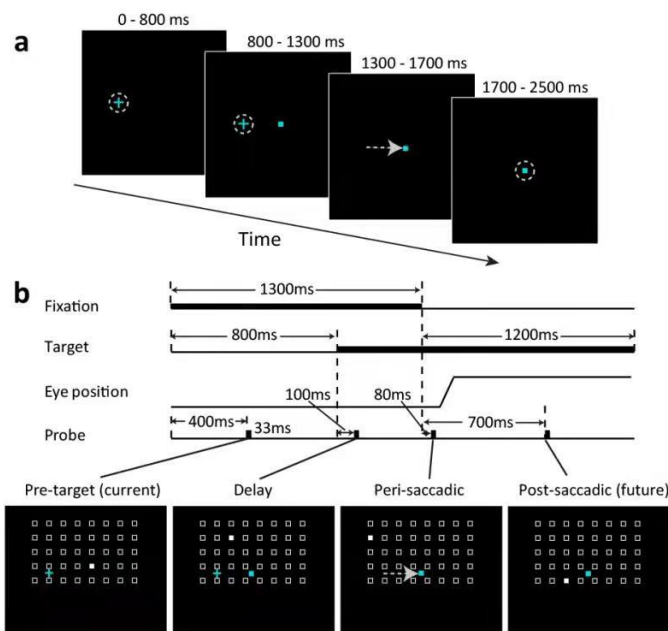


Fig. 2. The delayed saccade paradigm. The small cyan cross and square represent the fixation point and the saccade target, respectively. (a) The trial sequence. The flashed probes are not shown here but see panel b. (b) The time courses of a trial. Four probes were flashed, one for each of the four epochs: pre-target (**c**urrent), **d**elay, **p**eri-saccadic, and post-saccadic (**f**uture). A cell's RF mapped from these periods will be denoted **c**RF, **d**RF, **p**RF, and **f**RF, respectively. For each epoch, the probe stimulus (filled white square) appears randomly at one of the spatial array positions (open white squares, not shown in the experiment). The array size and location were tailored for each cell according pilot mapping.

could mask the CD effect. Additionally, around the saccade onset, neurons in LIP (one synapse away from the FEF) exhibit progressive RF shifts from cRF to fRF<sup>11</sup>, encompassing the entire portion of the space between them (Fig. 1a). The large time window could average response from the enlarged receptive field and underestimate the maximum forward-shift amplitude. 3) There might be differences between LIP and FEF.

Here we used a delayed saccade task to separate the attentional effect of the target onset and the effect of the saccade CD, and recorded LIP and FEF single-unit activities evoked by probes at different locations and times. The task allowed us to investigate the time course of the RF remapping in detail. We found that in the delay period, RFs shifted slightly in a direction close to the initial fixation 50 ms after the probe onset, but shifted toward the target 250 ms after the probe onset. In the

144 perisaccadic period, RFs shifted toward the target 50 ms after the probe onset, but around  
 145 the time of saccade onset/offset, the shifts became predominantly forward toward fRFs  
 146 and the amplitudes approached that of the saccades. When we integrated neuronal  
 147 activities from 50 to 350 ms after the probe onset, perisaccadic RFs were still closer to  
 148 the fRFs than to the target, indicating stronger forward than convergent remapping. Since  
 149 it first appeared in the delay period when the saccade was suppressed, convergent  
 150 remapping is not really perisaccadic but attentional.

151 To explain our data, we constructed a circuit model by integrating CD-gated  
 152 directional connections for forward remapping<sup>11</sup> and attention-modulated  
 153 center/surround connections<sup>19,20</sup> for convergent remapping. We further demonstrated that  
 154 both sets of connections emerged automatically in artificial neural networks trained to  
 155 update retinal positions of stimuli across saccades. Mechanistically, the center/surround  
 156 connections provide attractor dynamics to represent the retinal position of a stimulus as  
 157 an activity bump, and the CD-gated connections move the activity bump for  
 158 transsaccadic updating. The result has the surprising functional implication that the  
 159 center/surround connections might not only be modulated by attention to produce  
 160 convergent RF remapping but also work synergistically with the CD-gated connections to  
 161 enable accurate spatial perception across saccades.

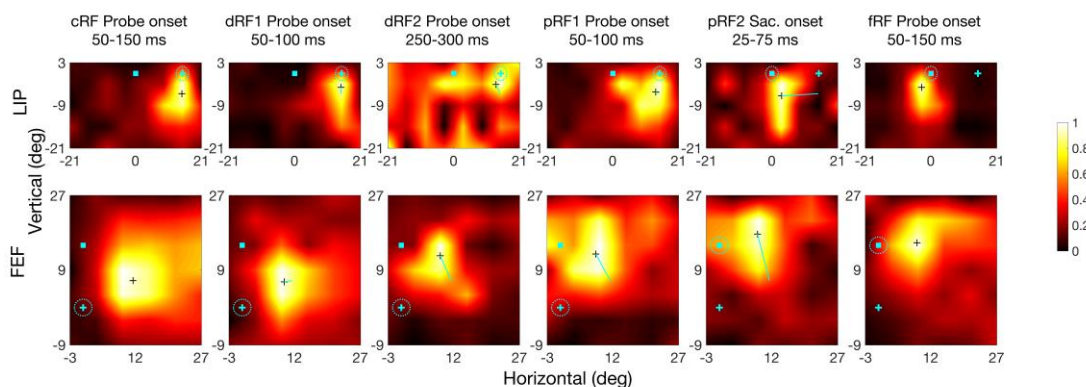


Fig. 3. RF heat maps for an example LIP cell (top row) and FEF cell (bottom row) from different time periods (columns). In each heat map, the cyan cross and square indicate the initial fixation point and saccade target, respectively (see Fig. 2 for their time course), and the dashed cyan circle indicates the eye position. The small black cross in a map marks the RF center. The cyan lines in dRF and pRF maps indicate the center shift relative to the cRFs. The scale of normalized responses is shown on the right. The fifth column is based on saccade onset alignment of the repeated trials whereas the other columns are based on the probe onset alignment.

## 162 Results

163 We used a delayed saccade task (Fig. 2) to sample a cell's responses from a grid  
 164 of spatial positions (tailored for each cell according to pilot RF mapping) and four time  
 165 epochs (after the monkeys achieved initial fixation, after the appearance of the target,

166 after the disappearance of the fixation (the go signal for the saccade), and well after the  
 167 saccade, respectively). In a given trial we flashed one probe stimulus in each epoch at a  
 168 random grid position, and across trials we sampled all grid positions and epochs. We  
 169 name the four epochs pre-target (current), **d**elay, **p**erisaccadic, and post-saccadic (**f**uture)  
 170 periods (Fig. 2b), and denote a cell's RFs mapped in these periods as its **c**RF, **d**RF, **p**RF,  
 171 and **f**RF, respectively. We recorded a total of 391 and 427 single units from LIP and FEF,  
 172 respectively, in 3 macaques. We then screened the data to select cells with significant  
 173 visual responses, well-sampled RFs, and significant RF shifts in the delay or perisaccadic  
 174 epoch (Methods).

175 **The direction and amplitude of RF remapping changed with time.**

176 We measured a cell's remapping in the delay and perisaccadic periods as the  
 177 shifts of its dRF and pRF centers relative to its cRF center, and defined the forward  
 178 direction as the direction from the cRF center to the fRF center. We present the RF heat

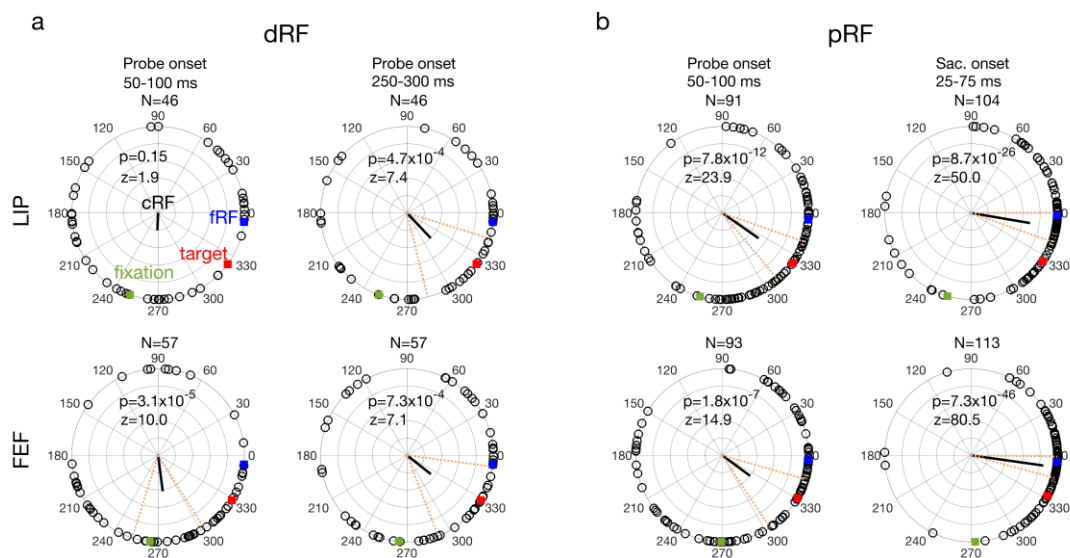


Fig. 4. The delay (a: dRF) and perisaccadic (b: pRF) shift directions of all LIP (top row) and FEF (bottom row) cells from different time periods (columns). In each polar plot, we align the cells' cRFs at the center and saccade directions along positive horizontal. The cells' mean fRF (forward), target, and initial-fixation directions are indicated by the blue, red, and green squares, respectively. Each open dot represents a cell's RF shift direction. The thick black line represents the circular mean of all the shift directions, and its significance is indicated by the p values from Rayleigh test for circular distributions. The dashed red lines mark the 95% confidence interval of the mean direction. The mean directions changed significantly across time in both LIP ( $p = 1.0 \times 10^{-5}$ ,  $F_{3,283} = 9.0$ ) and FEF ( $p = 2.1 \times 10^{-9}$ ,  $F_{3,316} = 15.5$ ), with Watson-Williams multi-sample test. The fourth column is based on saccade onset alignment of the repeated trials whereas the other columns are based on the probe onset alignment. Note that the numbers of cells (N) of the panels are different because the screening method was applied to each area, epoch, and alignment separately.

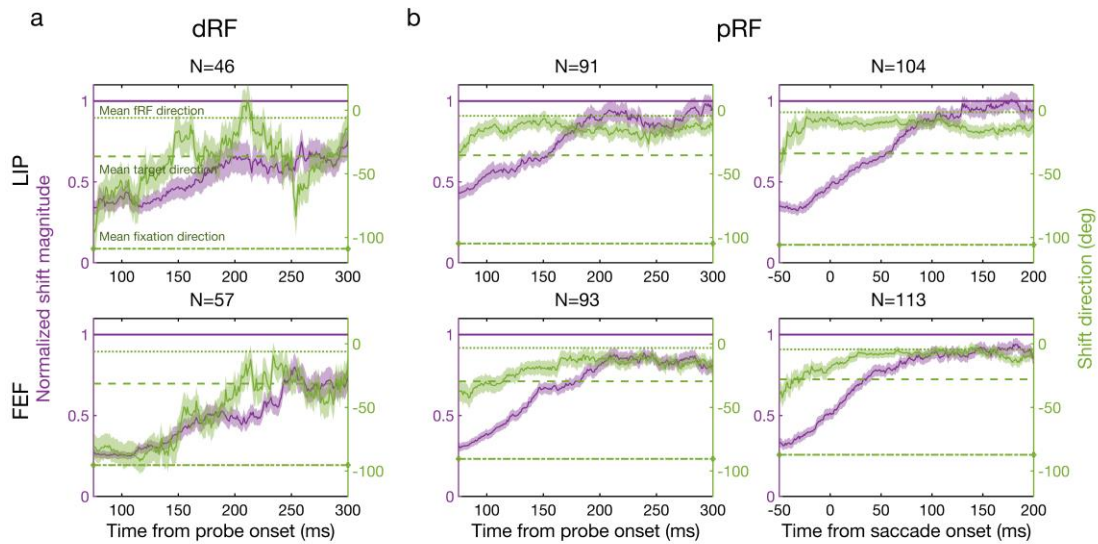


Fig. 5. Remapping time courses of the delay (a: dRF) and perisaccadic (b: pRF) periods for LIP (top row) and FEF (bottom row). Each panel shows the mean normalized shift magnitude (purple curve, left y-axis) and the mean shift direction (green curve, right y-axis) as a function of time. The amplitude of one (purple horizontal line) means a shift magnitude equals the saccade magnitude. The average fRF, target, and initial-fixation directions are indicated by the green dotted, dashed, and dot-dashed lines, respectively. Each data point is calculated from the responses of the 50-ms window centered around that point. The light purple and green regions indicate 1SEM. The third column is based on saccade onset alignment of the repeated trials whereas the other columns are based on the probe onset alignment.

179 maps of example cells in Fig. 3 and the population shift directions in Fig. 4. Only cells  
 180 with significant RF shifts in the delay or perisaccadic periods (according to  
 181 bootstrapping; see Methods) were included in the population analysis. For the delay  
 182 period, the dRFs obtained 50 to 100 ms after the delay probe onset shifted slightly in  
 183 directions between the initial fixation and the target (Fig. 3, second column; Fig. 4, first  
 184 column), but 250 to 300 ms after the delay probe onset, the shifts turned more towards  
 185 the target (Fig. 3, third column; Fig. 4, second column). For the perisaccadic period, the  
 186 pRFs obtained 50 to 100 ms after the perisaccadic probe onset shifted towards the target  
 187 (Fig. 3, fourth column; Fig. 4, third column). However, 25 to 75 ms after the saccade  
 188 onset, the pRFs shifted mostly towards the fRFs (Fig. 3, fifth column; Fig. 4, fourth  
 189 column). The mean shift directions were all significant except the early delay period of  
 190 LIP (see the p values in Fig. 4 plots). We marked the 95% confidence intervals of the  
 191 shift directions in Fig. 4. The mean directions changed significantly with time in both LIP  
 192 and FEF (Fig. 4 caption). In Supplementary Figs. S1 to S5, we show that these results  
 193 were robust against variations in analysis parameters.

194 To examine the time course of the delay and perisaccadic RF remapping in detail,  
 195 we used a moving window of 50 ms to analyze the dRF and pRF shifts as a function of



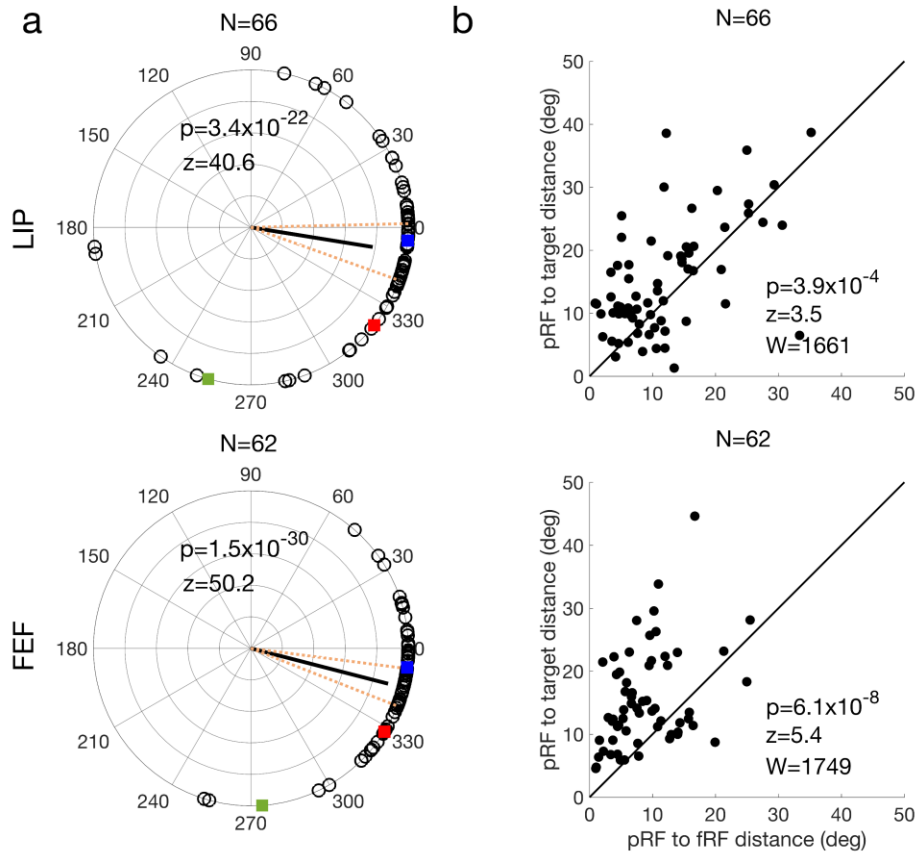


Fig. 6. Analysis of our perisaccadic data using the method of Zirnsak et al. The LIP and FEF results are shown in the top and bottom rows, respectively. The polar plot of remapping directions (a) have the same format as that of Fig. 4. In (b), each cell's pRF-to-target distance is plotted against its pRF-to-fRF distance. The p values in b indicate that for both LIP and FEF, the pRFs were significantly closer to the fRFs than to the targets on average (two-sided Wilcoxon signed-rank test).

196 time<sup>21</sup>. For the delay period, the shift direction changed gradually from a direction  
 197 between the initial fixation and target to the target (Fig. 5a, green). For the perisaccadic  
 198 period, the shift direction changed gradually from the target to the fRF (Fig. 5b, green).  
 199 When the shift directions pointed towards the initial fixation and subsequently toward the  
 200 target the shift magnitudes were at most only 55% and 67% of the distance between the  
 201 cRF and the target in LIP and FEF, respectively. When the shift directions pointed to the  
 202 fRF the shift magnitudes approached the distance between the cRF and fRF (Fig. 5,  
 203 purple). This shift would provide an accurate transsaccadic update of the retinal location  
 204 of the probe stimulus which was flashed before the saccade. When we used the larger  
 205 time interval used by Zirnsak et al. (50-350 ms after the onset of perisaccadic probe), we  
 206 did find the partial shifts toward the target that they described (Fig. 6a). This is not  
 207 surprising given that the time interval they used included both the time in which we  
 208 found convergent remapping and the time in which we found forward remapping. Note  
 209 that the cells' pRF centers were significantly closer to their fRF centers than to the targets



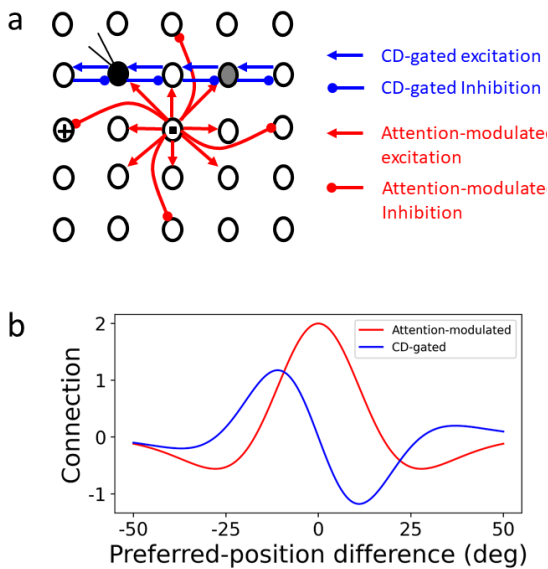


Fig. 7. The circuit model for explaining both forward and convergent RF shifts. (a) Schematic model structure. Black circles represent RF centers of a 2D array of topographically arranged LIP or FEF cells. The small cross and square indicate the initial fixation and target positions, respectively. The filled black circle is the cRF of the cell under recording, and the gray circle is its fRF. Only a small fraction of the connections is shown for clarity. (b) The attention-modulated symmetric connectivity (red) and CD-gated anti-symmetric connectivity (blue, for rightward saccades) as a function of the difference between two units' preferred locations (RF centers).

242 This center/surround connectivity in the spatial domain is similar to that in the orientation  
 243 domain<sup>22-25</sup>. Such connectivity simulated convergent shifts of orientation tuning curves  
 244 after perceptual learning or adaptation<sup>24-26</sup>; here we used it to account for convergent  
 245 shifts of spatial tuning (i.e., RFs) induced by attention.

246 The *second* mechanism is the CD-gated directional connections that can explain  
 247 perisaccadic forward expansion of RFs, as shown by Wang et al<sup>11</sup>. The cells have CD-  
 248 gated connections in all directions but for clarity, only a small subset of the connections  
 249 for the second-row cells gated by the CD for a rightward saccade are shown (Fig. 7, blue  
 250 lines). These connections are normally off. However, around the onset of the saccade,  
 251 they are tuned on by the CD signals, allowing stimulation at the cell's fRF (gray circle),  
 252 and at the region between the fRF and cRF, to propagate to the recorded cell, generating  
 253 perisaccadic forward RF shifts.

(see tests in Fig. 6b), indicating that on average, the forward remapping was stronger than the convergent remapping during the perisaccadic period in both LIP and FEF.

### A circuit model for the forward and convergent remapping

To account for the data, we modeled a 2D array of cells with their RF centers topographically arranged (Fig. 7, black circles). A rightward saccade is to be made from the cross to the square, and we record from the filled black cell. The above physiological results suggest that two mechanisms may be responsible for the convergent and forward RF shifts, respectively. The *first* mechanism is an attention-modulated circuit for convergent shift. Inspired by the physiological evidence of center-excitatory/surround-inhibitory modulation of visual responses around the saccade target in both LIP<sup>19</sup> and FEF<sup>20</sup>, we hypothesized center-excitatory/surround-inhibitory connections among all cells (Fig. 7, red lines; only the connections from the cell at the target location are shown). When there is attention at a location, we assume that connections from the cells tuned to that location are enhanced.

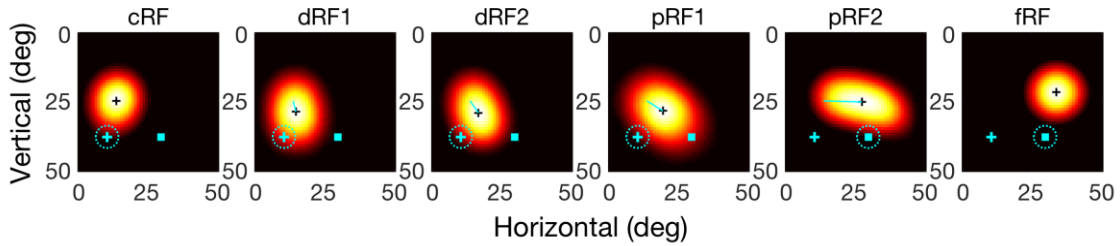


Fig. 8. Simulations of RF shifts in the delayed saccade task of Fig. 2. The first and last panels represent a model cell's cRF and fRF, respectively. The second and third panels represent the cell's early (dRF1) and late (dRF2) RFs in the delay period. The fourth and fifth panels represent the cell's early (pRF1) and late (pRF2) RFs in the perisaccadic period. In each panel, the cyan cross and square are the initial fixation point and target, respectively, and the dashed cyan circle indicates the eye position. The black cross marks the RF center. The thin cyan line in each dRF or pRF panel indicates the shift from the cRF center to the dRF or pRF center.

254 We considered a 2D array of 50x50 LIP/FEF units covering a space of 50°x50°,  
 255 each receiving feedforward visual inputs and recurrent inputs from other units via the  
 256 attention-modulated center/surround connections and CD-gated directional connections.  
 257 For the delay period, we divided the attention between the fixation point and the target,  
 258 and in the perisaccadic period, we introduced the CD signal (Methods). We probed the  
 259 model with flashes in the four epochs as in the experiment to measure cRFs, dRFs, pRFs,  
 260 and fRFs. By weighting the two mechanisms differently, it is straightforward to generate  
 261 cells with various degrees of forward and convergent shifts as we found in LIP and FEF  
 262 (an example shown in Fig. 8).

263 The model makes two predictions (Fig. 9, top row). 1) Since the CD-gated  
 264 connections propagate neuronal responses from a cell's fRF to its cRF, a distance equal  
 265 to saccade amplitude, the pRF forward shift amplitude should increase with the saccade  
 266 amplitude. 2) In contrast, the strengths of the attention-modulated center/surround  
 267 connections depend on the distance from the attentional locus, so the pRF convergent  
 268 shift amplitude should vary with the distance between a cell's cRF center and the target.  
 269 cRFs near the target have little room to shift toward the target and those far away are  
 270 barely affected by attention at the target. There is thus an intermediate, optimal distance  
 271 for maximal convergent shift. To test these predictions, we pooled the LIP and FEF cells  
 272 whose pRFs from 0 to 100 ms after the saccade onset shifted between the fRF and target  
 273 directions, and did a parallelogram decomposition of each shift vector into its forward  
 274 and convergent components. The results (Fig. 9, second row) are consistent with the  
 275 predictions. The scarcity of data at large cRF-to-target distance was due to technical  
 276 limitations: when this distance is large, it was difficult to keep a cell's entire cRF and fRF  
 277 within the screen boundaries.

278 To explain our data more quantitatively, we obtained distributions of the model  
 279 parameters by fitting the model to the perisaccadic LIP and FEF data, and interpolated  
 280 each parameter distribution as a mixture of Gaussians (Methods). We then randomly

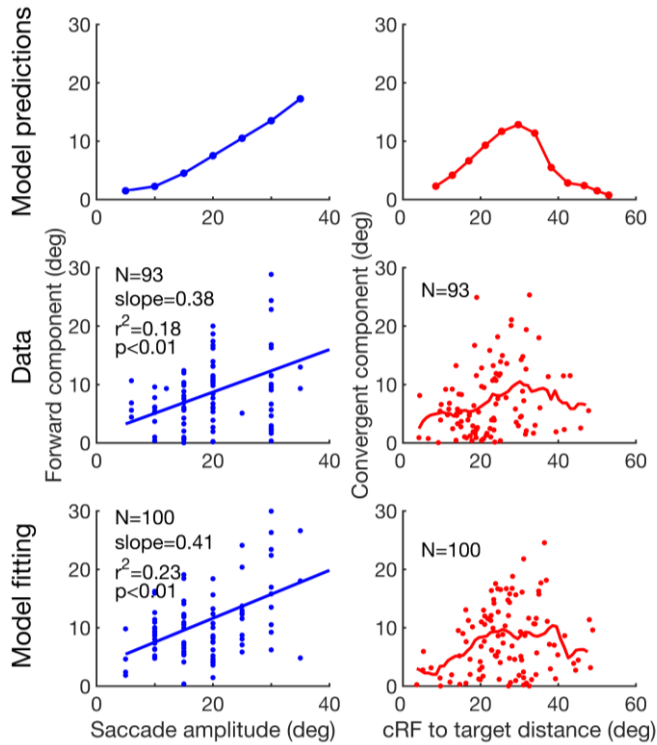


Fig. 9. Test the model predictions. First row: the predicted forward shift amplitude as a function of saccade amplitude, and the convergent shift amplitude as a function of the cRF-to-target distance. Second row: the pooled LIP/FEF pRF data confirming the predictions. Third row: Fitting the model to the data. For the second and third rows, the blue lines in the left column are linear fits, and the red curves in the right column are the moving average with a window size of 10 deg. For the forward component, the slopes of the data and the model are not significantly different from each other ( $p = 0.79$ ,  $t_{189} = 0.27$ , t-test). For the convergent component, the distributions of the data and the model are not significantly different from each other ( $p = 0.056$ , Peacock's 2D Kolmogorov-Smirnov test).

319 model are for such transsaccadic updating, with the center/surround connectivity for  
 320 storing the retinal position of a stimulus of interest<sup>27,28</sup>, and the CD-gated connectivity  
 321 for updating the memory across saccades<sup>6,11,29</sup>.

322 We tested this hypothesis by training neural networks on the predictive updating  
 323 task and examining whether randomly initialized connections converge to the required  
 324 patterns after training. For simplicity, we considered horizontal saccades only. The neural  
 325 networks consisted of two layers of units. The first layer provided visual inputs to the

resampled parameters from these distributions and run the model to obtain the pRF shifts. The results (Fig. 9, bottom row) matched the data well.

### The emergence of the required connectivity patterns in neural networks trained to update retinal positions across saccades

Both the attention-modulated center/surround connections and the CD-gated directional connections in our circuit model are motivated by physiological evidence in FEF and LIP. Nevertheless, one may argue that the model is ad hoc, designed specifically to explain the forward and convergent remapping. Is there a simple, functional consideration that leads to both connectivity patterns automatically? As we noted above, pRF remapping appears to update the retinotopic location of remembered (and disappeared) stimuli across saccades, a requirement for performing the double-step memory saccade task (Introduction). We therefore hypothesized that the two sets of connections in the circuit

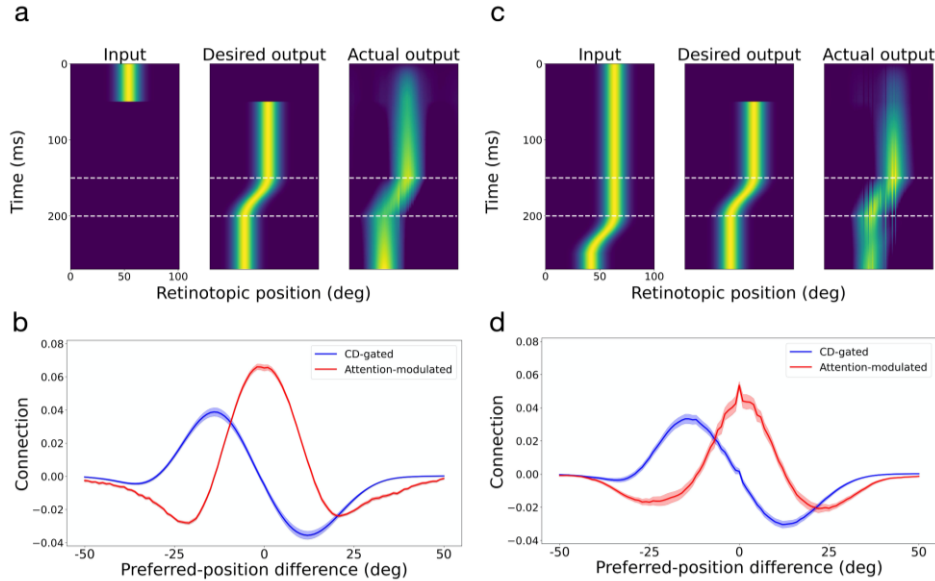


Fig. 10. Automatic generation of the required connectivity patterns in the circuit model by training neural networks to predictively update retinal positions of stimuli across saccades. Panels a and b are for the case of brief input stimuli and panels c and d are for the case of persistent input stimuli. Panels a and c show the test input (not included in the training), the desired output, and the actual output. Panels b and d show the average connection weights as a function of the difference between two units' preferred positions (RF centers). The red and blue curves are for the attention-modulated and CD-gated connections, respectively. Only the CD-gated connections for rightward saccades are shown. The shaded areas indicate 1SEM.

326 second layer which simulated LIP/FEF cells. The second-layer units were trained to  
 327 produce activity patterns representing the correct retinal locations of input stimuli across  
 328 saccades without reafference delay. The connections from the first- to second-layer units  
 329 were convolutional so that retinotopic inputs specified the feedforward component of  
 330 LIP/FEF RFs. The second-layer units were fully and recurrently connected to each other  
 331 with three sets of weights. The first two sets were gated by CD signals for saccades of  
 332 opposite directions whereas the third set was not gated by the CD signals but could be  
 333 modulated by attention (Methods). All connection weights in the network were randomly  
 334 initialized. The visual input in the first layer was a Gaussian bump centered at the initial  
 335 retinal location of a stimulus (Fig. 10a, left, for a 50 ms stimulus). Two additional input  
 336 units provided CD signals for opposite saccade directions. The desired output in the  
 337 second layer was the same Gaussian bump centered at the correct retinal position of the  
 338 (disappeared) stimulus across saccades (Fig. 10a, middle, for a rightward saccade started  
 339 at 150 ms and thus a leftward displacement of the representation for the stimulus' retinal  
 340 position). The weights were trained by minimizing the quadratic difference between the  
 341 actual and desired outputs. Many variations of the simulation produced similar results  
 342 (see Supplementary Information).

343 After the training converged and the actual output resembled the desired output

344 for test inputs not used during the training (Fig. 10a, right), we determined the units'  
345 mean connection weights to other units as a function of the distance between their  
346 preferred positions. Fig. 10b shows the results, with the attention-modulated and CD-  
347 gated connections in red and blue, respectively. The CD-gated connections shown are for  
348 rightward saccades and the mirror pattern for leftward saccades was also learned (not  
349 shown). Remarkably, these connectivity patterns closely resemble those we chose for the  
350 circuit model in Fig. 7b. Note that the connections in Figs. 7b and 10b are comparable  
351 only in their shapes, but not in their scales. This is because the circuit model and the  
352 artificial neural networks used different scales to represent the units' activities.

353 We then repeated the above neural network training but with persistent visual  
354 stimuli (Fig. 10c). Interestingly, we obtained very similar results, with not only the CD-  
355 gated directional connections but also the center/surround connections (Fig. 10d). The  
356 reason is that during saccades, the desired output position is different from the delayed  
357 refference input position (Fig. 10c, middle and left). The symmetric center/surround  
358 connectivity is needed to create an attractor activity pattern which was updated by the  
359 asymmetric CD-gated directional connectivity, independent of the input activity pattern  
360 <sup>30</sup>. We also considered the case where both brief and persistent input stimuli were trained  
361 together and where the attentional modulation was turned off, and again obtained similar  
362 results (see Supplementary Figs. S7 and S8).

363 We conclude that the connectivity patterns required by the circuit model emerge  
364 automatically and robustly in neural networks trained to update the representation of  
365 stimuli's retinal positions across saccades. This result suggests that although the  
366 center/surround connections and the CD-gated connections in the circuit model are for  
367 explaining convergent and forward RF shifts, respectively, they work synergistically to  
368 enable transaccadic stability.

## 369 **Discussions**

370 In the 19th century, Herman von Helmholtz, not only a great physicist but also a  
371 pioneering ophthalmologist, examined a patient who was blind in one eye from diabetes  
372 and sustained a paralysis of the lateral rectus muscle <sup>5</sup>. When the patient tried to look in  
373 the direction of the paralyzed muscle he perceived that the visual world moved in the  
374 opposite direction and then drifted back. Helmholtz postulated that under normal  
375 conditions, the brain uses the oculomotor signal to feed back to the visual system and  
376 adjust for the saccade. The discovery of perisaccadic forward remapping <sup>6-10</sup> provided a  
377 physiological mechanism for Helmholtz's theory. Neuropsychological evidence also  
378 supports this theory: patients with parietal lesions <sup>31,32</sup> cannot perform the double-step  
379 task when the subject makes the first saccade in the direction contralateral to the lesioned  
380 cortex. Furthermore, monkeys with inactivation of the medial dorsal nucleus of the  
381 thalamus, which relays CDs of saccadic commands from the SC to the FEF, cannot  
382 perform the double-step task, and their FEF neurons do not exhibit perisaccadic forward  
383 remapping <sup>29,33</sup>. However, an influential study questioned the existence of forward  
384 remapping <sup>15</sup> and instead showed that when they analyzed the activity of FEF neurons in  
385 the interval from 50 to 350 ms after the appearance of probe, which appeared around the  
386 time of the saccade target, many neurons seemed to remap their receptive fields toward

387 the target.

388 In humans<sup>34</sup> and monkeys<sup>17</sup> the abrupt onset of a visual stimulus evokes  
389 attention as measured by a change in perceptual threshold, as does the planning of a  
390 saccade<sup>17,35</sup>. Because of the large time interval (50-350 ms after probe onset) used by  
391 Zirnsak et al., we wondered if their results might be confounded by the presence both of  
392 an attentional event (the appearance of the saccade target) and the generation of a  
393 saccade, which would result in the combination of convergent remapping evoked by the  
394 saccade target and forward remapping evoked by the CD of the saccade command. Here,  
395 we addressed this controversy by using a delayed saccade task to separate the appearance  
396 of the target and the generation of the saccade. We recorded from LIP and FEF with  
397 matched procedures, and found that LIP and FEF showed similar patterns of remapping  
398 which varied with time: during the perisaccadic period, RFs converged toward the target  
399 shortly after the probe onset, but around the time of saccade and onward, the shift  
400 directions became predominantly forward toward fRF. When we used a large time  
401 window to integrate perisaccadic activities<sup>15</sup>, the pRFs were closer to the fRFs than to  
402 the targets, indicating stronger forward remapping than convergent remapping. We  
403 further found that the convergent shift started in the delay period when the saccade  
404 command, and its CD, must be suppressed; and the shift direction turned from between  
405 the initial fixation and the target to the target. Thus, unlike the forward shift that depends  
406 on the saccade CD<sup>29</sup>, the convergent shift appeared to follow attention from the initial  
407 fixation to the target. We conclude that both types of remapping are present in FEF and  
408 LIP and that forward remapping is not an artifact of undersampling convergent  
409 remapping. The convergent and forward RF shifts may be viewed as attentional  
410 remapping and perisaccadic remapping, respectively. These two types of remapping have  
411 also been found in V4<sup>21</sup> but with a much slower time course than that in LIP/FEF, raising  
412 the possibility that V4 might inherit the remapping from LIP/FEF.

413 Because our delayed-saccade paradigm helps distinguish between the forward-  
414 and convergent-remapping mechanisms, we were able to construct a circuit model for  
415 both types of remapping. Specifically, we integrated attention-modulated center/surround  
416 connections and CD-gated directional connections to explain the convergent and forward  
417 RF shifts, respectively. The model's predictions on the forward shift amplitude as a  
418 function of the saccade amplitude and the convergent shift amplitude as a function of the  
419 cRF-to-target distance agreed with the data. We then showed that both sets of  
420 connections emerged automatically and robustly in neural networks trained to update  
421 representations of retinal positions across saccades. Since this updating is needed for the  
422 double-step memory saccade task, it can be viewed as an operational definition of  
423 transaccadic stability. We suggest that the CD-gated connections and the center/surround  
424 connections together specify a mechanism for transaccadic stability. The mechanism  
425 follows a classic prescription<sup>30</sup>: symmetric center/surround connections produce attractor  
426 dynamics to represent a stimulus as an activity bump whereas the asymmetric CD-gated  
427 connections move the activity bump for updating across saccades. Although we initially  
428 used the center/surround connections to explain convergent remapping, they might be an  
429 integral part of transaccadic stability mechanism.

430 Zirnsak et al<sup>15</sup> suggested that convergent RF remapping explains compressive

431 perceptual mislocalization: stimuli flashed briefly before a saccade are perceived as  
432 occurring at the saccade target when postsaccadic visual references are present<sup>36,37</sup>.  
433 However, whether convergent remapping produces compressive mislocalization is  
434 unclear, and depends on, among other things, whether the positional decoder is aware of  
435 the remapping<sup>38</sup>. Additionally, when saccadic adaptation is used to dissociate the  
436 postsaccadic eye position and the target position, the perceived compression is toward the  
437 eye position, not the target position<sup>39</sup> whereas LIP and SC neurons represent the target  
438 position, not the eye position<sup>40,41</sup>. Therefore, one would not expect convergent  
439 remapping (in LIP and SC at least) to explain compressive mislocalization.

440 In addition to perisaccadic RF remapping, a prominent physiological finding  
441 relevant to transaccadic perceptual stability is gain fields, the modulation of visual  
442 response by eye position<sup>42</sup>. Whereas perisaccadic remapping may realize the stability by  
443 predictively updating retinal representations across saccades, gain fields may do so by  
444 combining eye position and retinal representations to form head-centered representations  
445<sup>43</sup>. Recent studies suggest that gain fields and perisaccadic remapping may be responsible  
446 for transaccadic stability at long and short time scales, respectively<sup>44,45</sup>, consistent with  
447 their respective dependence on slow proprioceptive eye-position signals and fast saccade  
448 CD signals<sup>29,46</sup>. On the other hand, it is theoretically possible to integrate fast CD signals  
449 to provide fast, predictive eye-position signals. The existence of such CD integrators is an  
450 open question for future research.

451 Our study may also have functional implications on potential relationships  
452 between working memory and attention. According to our models, although the  
453 center/surround connections may store working memories of visual stimuli including  
454 saccade targets, the same connections can be modulated by attention to generate  
455 convergent RF shifts. The relationship between working memory and attention has been  
456 discussed in the literature<sup>47</sup>. Our work, however, suggests a specific mechanism:  
457 attention to a stimulus modulates the connections that are responsible for storing the  
458 stimulus in working memory. Therefore, LIP and FEF circuits might integrate  
459 mechanisms for working memory, attention, saccade planning, and transaccadic stability  
460 all together.

461

462

## 463 **Methods**

464 **Animal preparation:** Three male adult rhesus monkeys (*Macaca mulatta*) weighing  
465 from 9 to 11 kilograms participated in this study. All procedures were approved by the  
466 ethics committee at Beijing Normal University. We have complied with all relevant  
467 ethical regulations. We surgically planted two search coils (one for each eye; Crist  
468 Instrument Sclera, sample rate at 2.7KHz), a head restraint post, and two recording  
469 chambers (for LIP and FEF, respectively; PEEK), for each monkey. We positioned the  
470 recording chambers according to our experience and/or the MRI scans. We centered the



471 LIP chambers for the three monkeys, respectively, at 3, 10, and 3.2 mm posterior to the  
472 interaural plane, and 13, 15, and 15 mm lateral from the middle line. We centered the  
473 FEF chambers for the three monkeys at 28, 18, and 23.5 mm anterior to the interaural  
474 plane, and 13, 15, and 18 mm lateral from the middle line. The two recording areas were  
475 verified later (see below).

476 **Recording procedures:** We used insulated tungsten microelectrodes (0.3~1.0 M $\Omega$ , FHC)  
477 to record single-unit activity. We inserted the electrodes through dura via stainless steel  
478 guide tubes, and controlled their advancement in the cortices with micromanipulators  
479 (NAN Instruments). Neuronal activities collected by the electrodes were amplified  
480 (Alpha Omega) and filtered (268-8036Hz) before online sorting with AlphaLab SnR  
481 (Alpha Omega). We identified LIP based on persistent activities in the delay period of a  
482 memory saccade task<sup>48</sup>, and FEF according to micro-stimulation (100ms, 0.05mA,  
483 biphasic pulses) evoked saccades of fixed vectors<sup>49,50</sup>. After several recording sessions,  
484 we did MRI scan of the first two monkeys' LIP chambers and the third monkey's LIP and  
485 FEF chambers, confirming that the LIP recording sites were within the lateral bank of the  
486 intraparietal sulcus, and the FEF recording sites were in the anterior bank of arcuate  
487 sulcus. The same recording and analysis procedures were applied to LIP and FEF.

488 After isolating a single unit with a template-matching method, we first did a pilot  
489 mapping of its visual RF: while the monkey maintained central fixation in each trial, we  
490 flashed a sequence of 6 probe stimuli (1°x1°) at random locations sampled from an 8x8  
491 array with adjacent locations separated by 6° in both horizontal and vertical directions. A  
492 probe lasted 33 ms and successive probes were separated by 400 ms. Each location had  
493 about five responses. If visual inspection determined that the unit showed clear responses  
494 for at least one probe location, we moved on to the main, delayed saccade task (Fig. 2).  
495 We tailored the array of probe positions to cover the unit's cRF-fRF region and the target  
496 region, according to the pilot RF mapping and the planned saccade target for the unit.  
497 Across cells, the array varied from 4x5 to 10x12 positions, with 5x8, 5x9, and 6x8 the  
498 most common. The spacing between adjacent positions (along both horizontal and  
499 vertical axes) varied from 2° to 6°, with 6° the most common. The saccade amplitude  
500 varied from 5° to 30°, with 15° and 20° the most common. Despite our effort, the RFs of  
501 some cells were not measured sufficiently complete because of the limited screen size  
502 and large RFs and/or large saccades; these cells were excluded (see below). As shown in  
503 Fig. 2 and described in the text, the delayed saccade task allowed us to measure a cell's  
504 cRF, dRF, pRF, and fRF from the initial-fixation (**c**urrent), **d**elay, **p**erisaccadic, and  
505 postsaccadic (**f**uture) epochs of a trial.

506 In the actual experiments, the initial fixation point and the target were both red  
507 squares of 0.3° width, but for the ease of illustration, we represented them as cyan squares  
508 and crosses, respectively, in the figures.

509 **Data screening and analysis:** We screened and processed the data as follows. (1) We  
510 selected the cells with significant visual responses. For each epoch, we aligned the repeated  
511 trials to the probe onset. For the perisaccadic epoch, we additionally aligned repeated trials  
512 to the saccade onset. For each epoch and probe position of a cell, we calculated the response  
513 as the mean firing rate 50-150 ms after the probe onset or 0-100 ms after the saccade onset  
514 (these windows were chosen because they contained most of the activities), and the  
515 baseline as the mean firing rate 0 to 50 ms before the probe onset. We found the probe  
516 position that had maximal response, and then performed a single Wilcoxon rank-sum test  
517 (two-sided) against the corresponding baseline at the 0.05 level. This procedure avoided  
518 multiple comparisons. Cells were selected separately for each epoch and alignment. For a  
519 selected cell in an epoch, we followed Zirnsak et al.<sup>15</sup> to normalize its spatial responses  
520 according to  $(r_k - r_{\min}) / (r_{\max} - r_{\min})$  for all  $k$ , where  $r_k$  is the response at position  $k$ , and  $r_{\max}$  and  
521  $r_{\min}$  are the maximum and minimum responses across all positions. An advantage of this  
522 normalization is that because of the subtraction of  $r_{\min}$ , any non-visual (such as saccade  
523 related) responses are discounted. Also note that because we always place the target outside  
524 a cell's RF, saccade contribution to the measured responses must be minimal. (2) We  
525 selected cells with well-measured RFs. For each epoch and alignment of a cell, we linearly  
526 interpolated the normalized responses across positions to obtain the RF heat map. We  
527 traced the response contour at 85% of the maximum (contour criterion) and required that  
528 80% of contour were within the sampled position grid (completeness criterion). We then  
529 estimated the RF center as the center-of-mass of the responses within the region set by the  
530 contour criterion. We used the 85% contour criterion instead of Zirnsak et al.'s<sup>15</sup> 75%  
531 because the higher value determined the RF center more reliably. (In Supplementary  
532 Information Figs. S1 to S5, we demonstrate that changing the two criteria do not change  
533 our conclusions.) (3) We selected cells with significant RF shifts. For each cell, we  
534 calculated the shifts of its dRF and pRF centers relative to its cRF center in visual angles,  
535 and determined the significance of a shift via the following bootstrapping<sup>51</sup>. For each  
536 epoch and probe position of a cell, we assumed that the spike count of a trial followed a  
537 Poisson distribution with the mean equal to the measured mean spike count. We then  
538 simulated the recording and analysis of the cell 1000 times by sampling spike counts from  
539 the distributions with the trial numbers equal to those of the actual experiment. To  
540 determine, for example, whether the 1000 dRF centers shifted significantly from the 1000  
541 cRF centers in the simulation, we calculated their overlaps along the axis linking the mean  
542 dRF center and cRF center, and required the overlap to be less than 5%. After the screening,  
543 we investigated how remapping changed with time by choosing various 50 ms windows to  
544 determine dRFs and pRFs as detailed in the main text.

545 We considered each recorded cell as a distinct sample. After the screening (see the  
546 paragraph above), the numbers of cells for different time periods were different, and for  
547 this reason, results from different time periods could not be treated as repeated measures.  
548 We thus used Watson-Williams test to determine whether remapping directions changed  
549 significantly over time in Figs. 4 and S1-S5.

550 We also applied Zirnsak et al.'s method to analyze perisaccadic remapping in our  
551 LIP and FEF data. In addition to screening for cells with sufficient trials, well-sampled RFs,  
552 and significant RF shifts in perisaccadic epoch, we selected the trials in which perisaccadic  
553 probes occurred within 150 ms before saccade onset, for all epochs used the responses from  
554 50 to 350 ms after the probe onset, and set the contour criterion to 75%<sup>15</sup>.

555 We used Matlab to perform the data screening and analysis.

556

557 **Circuit model:** We simulated a 2D array of 50x50 LIP/FEF units covering a space of  
558  $50^\circ \times 50^\circ$ , each unit governed by the standard equations:

559 
$$\tau du(x, y) / dt = -u(x, y) + W(x, y) * r(x, y) + I,$$

560 
$$r(x, y) = \alpha \max[u(x, y), 0],$$

561 
$$I(x, y, t) = \frac{1}{b^a \Gamma(a)} t^{a-1} e^{-\frac{t}{b}} \exp[-(x^2 + y^2) / 2\sigma_s^2],$$

562 where  $u(x, y)$  and  $r(x, y)$  are, respectively, the membrane potential (relative to spike  
563 threshold) and firing rate (relative to background rate) of the unit at location  $(x, y)$ ,  $\tau = 20$   
564 ms is the membrane time constant,  $\alpha$  is a constant relating  $u(x, y)$  to  $r(x, y)$  (which affects  
565 the model only through its product with  $W$ , specified below),  $*$  denotes spatial  
566 convolution,  $W$  specifies connections between the units, and  $I$  represents the feedforward  
567 visual inputs with  $a = 2$ ,  $b = 18$  ms. For each unit, its connection matrix  $W(x, y)$  to other  
568 units at relative coordinates  $(x, y)$  is a sum of two parts: (1) center-surround connections  
569 modeled as a difference between two circularly symmetric 2D Gaussians:

570 
$$W_1 = w_{exc} \exp[-(x^2 + y^2) / (2\sigma_{exc}^2)] - w_{inh} \exp[-(x^2 + y^2) / (2\sigma_{inh}^2)]$$

571 (with  $\alpha w_{exc} = 4$ ,  $\alpha w_{inh} = 2$ ,  $\sigma_{exc} = 12^\circ$ ,  $\sigma_{inh} = 18^\circ$ ), and (2) asymmetric connections with  
572 excitation in the opposite direction of the pending saccade<sup>11</sup>; for horizontal saccades, we  
573 used the antisymmetric form:  $W_2 = \beta \partial W_1 / \partial x$  along the horizontal axis, where  $\beta = 12$   
574 determines the relative strength between  $W_1$  and  $W_2$ . (For other saccade axis,  $W_2$  should be  
575 rotated to the saccade axis.) Many expressions for  $W_2$  would work; we choose the  
576 derivative of  $W_1$  for its known property in shifting activity profile in the direction where  
577  $W_2$  is excitatory<sup>30</sup> and for reducing the number of free parameters. Note that Wang et al  
578<sup>11</sup> used the CD-gated excitatory connections against the saccade direction and a global  
579 inhibition. We used an equivalent connectivity with inhibitory and excitatory connections  
580 along and against the saccade direction, respectively. Fig. 7b shows the shapes of  $W_1$  and  
581  $W_2$  along  $x$  for rightward saccade. For the delayed saccade task,  $W_1$  is multiplied by an  
582 attentional modulation factor:  $1 + w_{att} \exp[-(x^2 + y^2) / (2\sigma_{att}^2)]$  centered at the attentional  
583 locus (initial fixation or target position), where  $\sigma_{att} = 15^\circ$ . Similarly,  $W_2$  is multiplied by a  
584 CD gating factor:  $w_{CD} \exp[-(t / \sigma_{CD})^6 / 2]$  where  $w_{CD}$  is not independent but determines the  
585 CD strength through its product with  $\beta$ ,  $t$  is measured relative to the saccade onset time,  
586 and  $\sigma_{CD} = 65$  ms. For the six time periods in Fig. 8 (cRF, dRF1, dRF2, pRF1, pRF2, and  
587 fRF), we set  $w_{att}$  at the initial fixation to 0.4, 0.8, 0.6, 0, 0, and 0,  $w_{att}$  at the target to 0,  
588 0.2, 0.3, 0.45, 0.2, and 0, and  $w_{CD}$  to 0, 0, 0, 0.1, 0.6, and 0, respectively.  $w_{att}$  at the initial  
589 fixation was larger for dRF1 and dRF2 than for cRF because after the target onset, the  
590 monkeys had to suppress any saccades to the target and maintain the initial fixation.

591 Visual inputs from a stimulus to the LIP/FEF units were modeled as a circular Gaussian  
 592 centered at the stimulus with  $\sigma_s = 7^\circ$ . We probed the model with flashes in the four  
 593 epochs as in the experiment to measure cRFs, dRFs, pRFs, and fRFs. Many variations of  
 594 the model and/or the parameters produced similar results. For example, the attentional  
 595 modulation and CD-gating functions can be replaced by simple step functions, and the  
 596 parameters can be optimized to fit individual cell's RF shifts (see below). We  
 597 implemented the circuit model with COSIVINA, an open source toolbox for Matlab.

598 The model predicts that the forward-shift amplitude grows with the saccade  
 599 amplitude and that the convergent-shift amplitude depends on the cRF-to-target distance,  
 600 with a maximal shift at an intermediate distance (see text). To make these predictions  
 601 more quantitative, we obtained distributions of the model parameter set  $\Theta = (w_{exc}, \sigma_{exc},$   
 602  $w_{inh}, \sigma_{inh}, w_{att}, \sigma_{att}, w_{CD})$  by fitting the model to the perisaccadic LIP and FEF data. We  
 603 focused on these parameters as they are most relevant to the RF shifts. We first initialized  
 604 the parameters to random values, each drawn from the uniform distribution  $U(0, 20)$ . For  
 605 each recorded cell with a shift vector  $\vec{s}$ , we used PyTorch's differentiation engine and  
 606 Adam optimizer (both learning rate and weight decay set to 0.01) to perform gradient  
 607 descent on the parameters by minimizing the cost function:

$$608 \quad L(\Theta) = \frac{1}{2} \|\vec{s}' - \vec{s}\|^2 - \lambda \vec{s}' \cdot \vec{s}$$

609 where  $\vec{s}'$  is the shift vector produced by the model. The first term minimizes the  
 610 difference between  $\vec{s}$  and  $\vec{s}'$ . We included the second, dot-product term because when  $\vec{s}$   
 611 and  $\vec{s}'$  have small amplitudes, the first term can be small even when  $\vec{s}$  and  $\vec{s}'$  point at  
 612 different directions. The second term ensures that the two vectors point in similar  
 613 directions. We let  $\lambda = 0.01$  as it produced good fits for all cells. Because of the limited  
 614 number of recorded cells, we pooled together the optimized parameters obtained from  
 615 fitting both the LIP and FEF cells, and for each saccade amplitude, we fit each parameter  
 616 distribution through the Gaussian kernel density estimation. We treated the parameters  
 617 independently as we did not have nearly enough data to determine their joint distribution.  
 618 We then sampled parameters from these distributions and run the model to predict the  
 619 forward-shift amplitude as the function of the saccade amplitude, and the convergent-  
 620 shift amplitude as a function of the cRF-to-target distance (Fig. 9). The cRF centers were  
 621 resampled from the distribution of the measured cRF centers by fitting a mixture of  
 622 Gaussians.

623 **Artificial neural networks:** We trained artificial neural networks to predictively update  
 624 retinal locations of stimuli across saccades and demonstrated automatic and robust  
 625 emergence of both the center/surround connections and the CD-gated directional  
 626 connections needed in the circuit model for explaining the convergent and forward RF

627 shifts, respectively. A network consisted of two layers of units: the first layer provided  
 628 visual inputs, originated from retina, to the second, LIP/FEF layer. For simplicity, we  
 629 only considered the horizontal dimension and horizontal saccades. Each layer had 100  
 630 units representing  $100^\circ$  of space. The connections from the first to second layer was  
 631 translationally invariant<sup>52</sup> (convolution kernel size of  $5^\circ$ ) so that the second layer  
 632 preserved the retinotopic representation of the first layer. There were two additional input  
 633 units with a one-hot representation of the CD signals for the two opposite directions of  
 634 saccades. For a given saccade, the relevant CD unit was turned on for the duration of the  
 635 saccade. The second-layer units are fully and recurrently connected with three sets of  
 636 weights. The first two sets were multiplicatively gated by the two CD input units for  
 637 opposite saccade directions, respectively, whereas the third set was optionally modulated  
 638 by attention when the stimulus was considered task relevant (e.g., when it was the  
 639 saccade target). The simulations in Figs. 10 and S7 included attentional modulation with  
 640  $\sigma_{att} = 15^\circ$  and  $w_{att} = 0.4$ . In Fig. S8, we showed an example where we obtained similar  
 641 connectivity patterns in the absence of attentional modulation. The dynamics of the units  
 642 was governed by equations identical to those for the circuit model above, and we used the  
 643 ReLU activation function.  
 644

645 The networks were trained on the task of predictively updating the retinal position  
 646 of visual stimuli across saccades. Specifically, the output units should have the activity  
 647 pattern representing the correct retinal position of an input stimulus across saccades  
 648 without reafference delay. Both the input and desired output are Gaussian activity  
 649 patterns with  $\sigma = 6^\circ$ . We considered both brief and persistent input stimuli, with one  
 650 stimulus per training trial. The brief stimuli appeared for 50 ms before saccades and then  
 651 disappeared whereas the persistent stimuli stayed for the duration of the simulations.  
 652 Importantly, the input units provided inputs to LIP/FEF units and we assumed a 50 ms  
 653 delay from retina to LIP/FEF (Fig. 10c, left). The output activity pattern was trained to  
 654 compensate for this delay by using the CD signals (Fig. 10c, middle). Therefore,  
 655 regardless of whether the input stimuli were brief or persistent, the desired output was the  
 656 same: an activity pattern representing the correct retinal position of the stimuli without  
 657 delay. This is what we mean by “predictive updating.”  
 658

659 The model was trained to minimize the mean squared error between desired  
 660 output and actual output as follows:

$$L(\theta) = \frac{1}{2} \sum_i \sum_t \|h_{t,i} - y_{t,i}\|^2,$$

661 where  $h_{t,i}$  and  $y_{t,i}$  are the desired and actual outputs for unit  $i$  at time step  $t$ . All weights  
 662 were randomly initialized by a uniform distribution  $\mathcal{U}[-\frac{1}{\sqrt{n}}, \frac{1}{\sqrt{n}}]$ , where  $n$  is the total  
 663 number of weights of a given type (feedforward or recurrent) a unit receives. For the  
 664 feedforward weights,  $n = 7$  (the convolutional kernel size), and for the recurrent weights  
 665  $n = 100$  (the number of recurrent units). We updated the weights with the Adam  
 666 optimization algorithm (learning rate was 0.001, weight decay was 0.01). The model was  
 667 implemented in PyTorch.  
 668  
 669

670 **Supplementary Information**

671 Our physiological conclusions are robust against variations in the data analysis.  
672 There were two key criteria in our analysis. (1) Contour criterion: For a given RF heat  
673 map we measured, we set a percentage of the peak response to mark a contour around the  
674 peak for calculating the RF center of mass. The contour criterion was set to 85% in the  
675 main text. (2) Completeness criterion: We required that the measured RF heat map  
676 included a minimum percentage of the contour defined by the contour criterion. This  
677 completeness criterion was set to 80% in the main text. We did extensive additional data  
678 analysis to demonstrate that our physiological conclusions are robust against variations in  
679 these criteria. We focused on Fig. 4 of the main text as it contained the main results on  
680 the RF shift directions in the delay and perisaccadic periods for both LIP and FEF. In  
681 Figs. S1 and S2, we kept the contour criterion at 85%, but set the completeness criterion  
682 to 90% and 70%, respectively (instead of 80% in Fig. 4). In Figs. S3 to S5, we changed  
683 the contour criterion to 75%, a value used by Zirnsak et al.'s <sup>15</sup>, and set the completeness  
684 criterion to 90%, 80%, and 70%, respectively. These figures all show results similar to  
685 those in Fig. 4 of the main text.

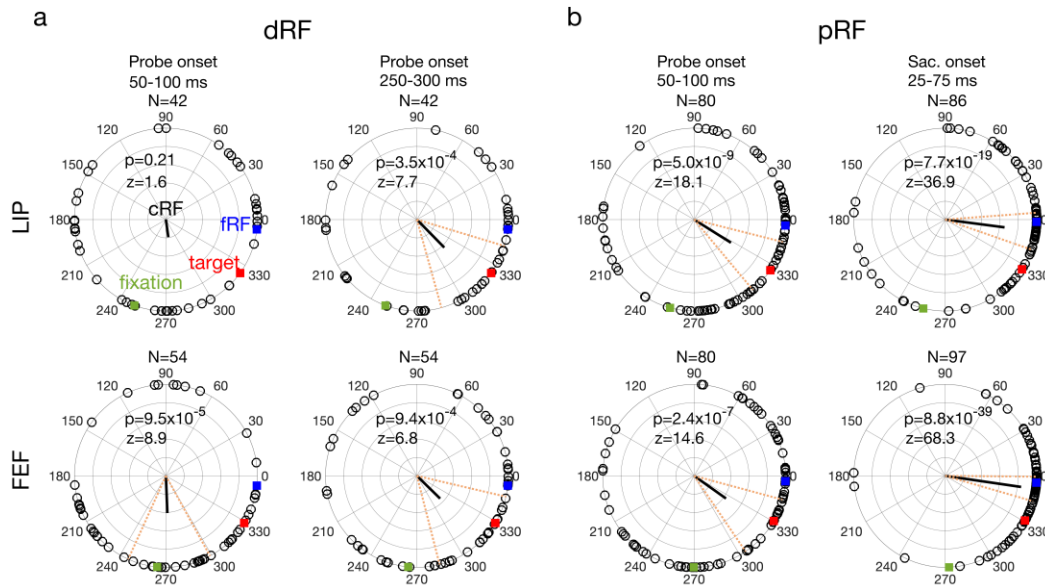


Fig. S1. The delay (a: dRF) and perrisaccadic (b: pRF) shift directions of all LIP (top row) and FEF (bottom row) cells from different time periods (columns). The contour criterion was 85% and the completeness criterion was 90%. The format was identical to that of Fig. 4 of the main text. The mean directions changed significantly across time in both LIP ( $p = 2.5 \times 10^{-4}$ ,  $F_{3,246}=6.6$ ) and FEF ( $p = 1.9 \times 10^{-9}$ ,  $F_{3,281}=15.7$ ), with Watson-Williams multi-sample test.

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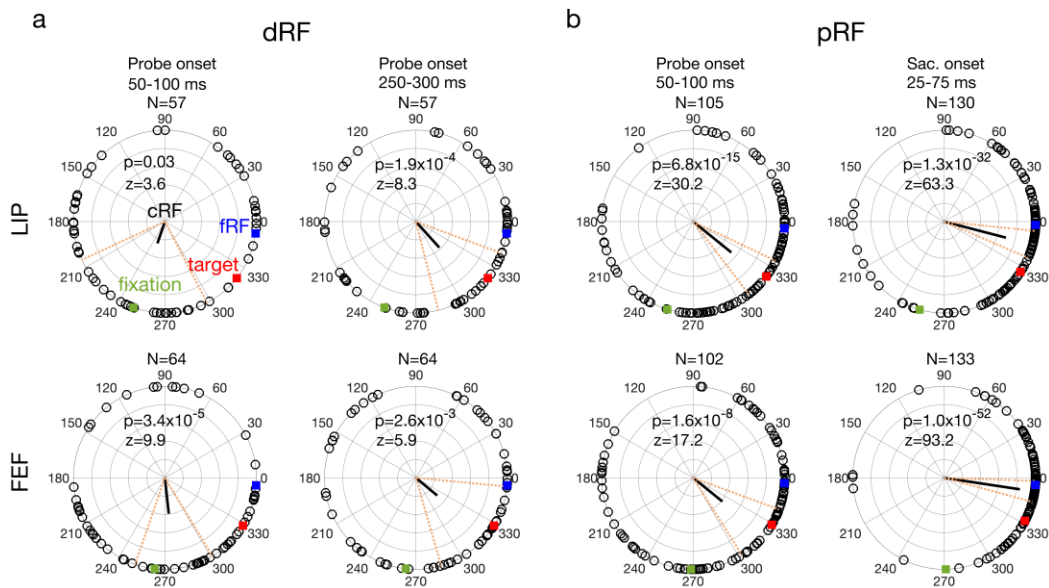


Fig. S2. The delay (a: dRF) and perrisaccadic (b: pRF) shift directions of all LIP (top row) and FEF (bottom row) cells from different time periods (columns). The contour criterion was 85% and the completeness criterion was 70%. The format was identical to that of Fig. 4 in the main text. The mean directions changed significantly across time in both LIP ( $p = 3.6 \times 10^{-9}$ ,  $F_{3,345} = 14.9$ ) and FEF ( $p = 1.7 \times 10^{-10}$ ,  $F_{3,359} = 17.3$ ), with Watson-Williams multi-sample test.

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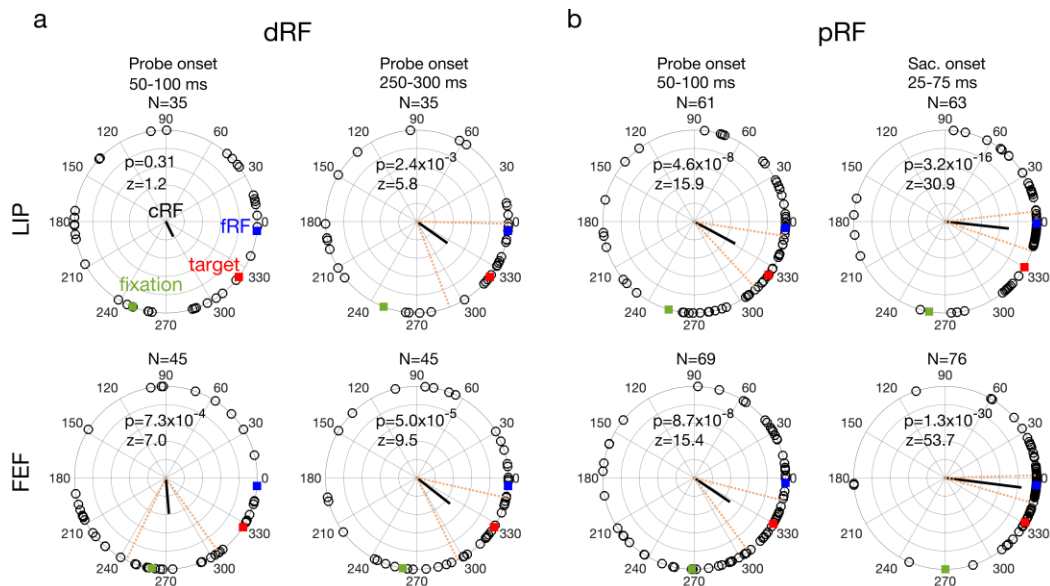


Fig. S3. The delay (a: dRF) and perrisaccadic (b: pRF) shift directions of all LIP (top row) and FEF (bottom row) cells from different time periods (columns). The contour criterion was 75% and the completeness criterion was 90%. The format was identical to that of Fig. 4 in the main text. The mean directions changed significantly across time in both LIP ( $p = 0.021$ ,  $F_{3,190}=3.3$ ) and FEF ( $p = 1.4 \times 10^{-7}$ ,  $F_{3,231}=12.5$ ), with Watson-Williams multi-sample test.

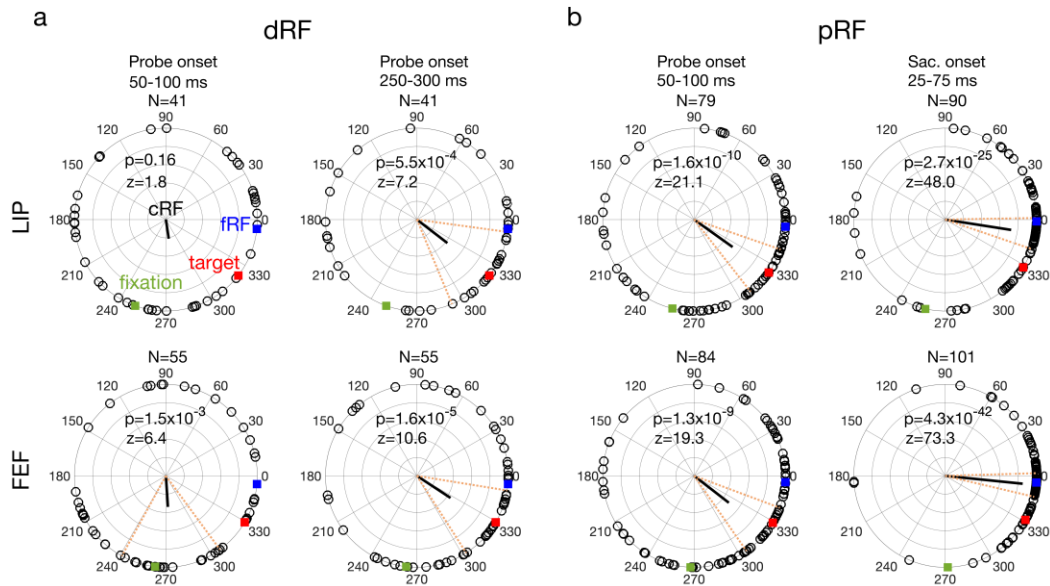


Fig. S4. The delay (a: dRF) and perrisaccadic (b: pRF) shift directions of all LIP (top row) and FEF (bottom row) cells from different time periods (columns). The contour criterion was 75% and the completeness criterion was 80%. The format was identical to that of Fig. 4 in the main text. The mean directions changed significantly across time in both LIP ( $p = 1.9 \times 10^{-4}$ ,  $F_{3,247}=6.8$ ) and FEF ( $p = 2.5 \times 10^{-9}$ ,  $F_{3,291}=15.4$ ), with Watson-Williams multi-sample test.

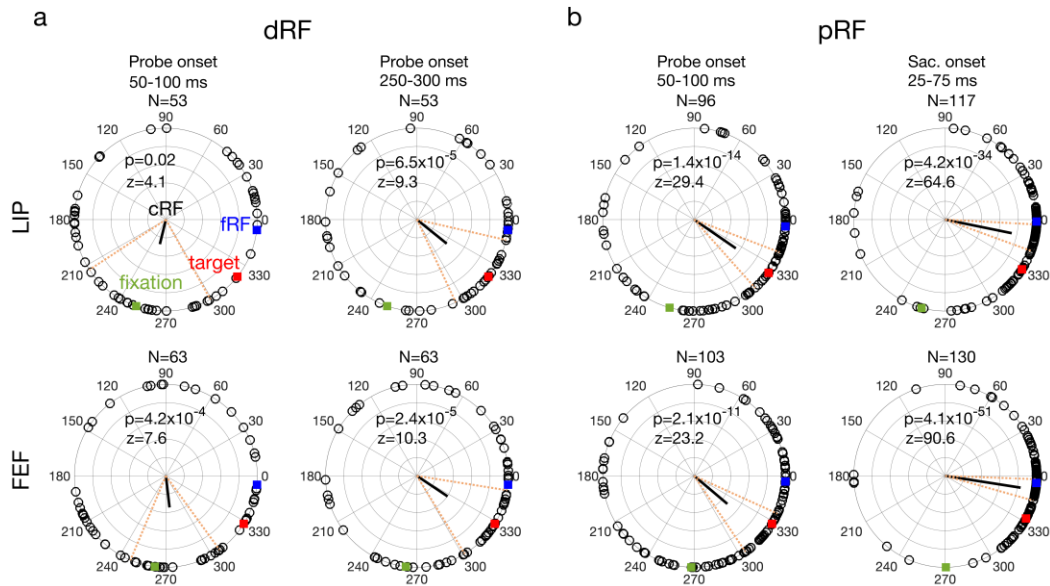


Fig. S5. The delay (a: dRF) and perrisaccadic (b: pRF) shift directions of all LIP (top row) and FEF (bottom row) cells from different time periods (columns). The contour criterion was 75% and the completeness criterion was 70%. The format was identical to that of Fig. 4 in the main text. The mean directions changed significantly across time in both LIP ( $p = 6.7 \times 10^{-9}$ ,  $F_{3,315} = 14.5$ ) and FEF ( $p = 7.0 \times 10^{-10}$ ,  $F_{3,355} = 16.2$ ), with Watson-Williams multi-sample test.

698 In the main text, we showed the distributions of the RF shift directions at four  
699 time points (Fig. 4). For completeness, we show in Fig. S6 the distributions of the shift  
700 vectors (both the directions and amplitudes)<sup>21</sup>.

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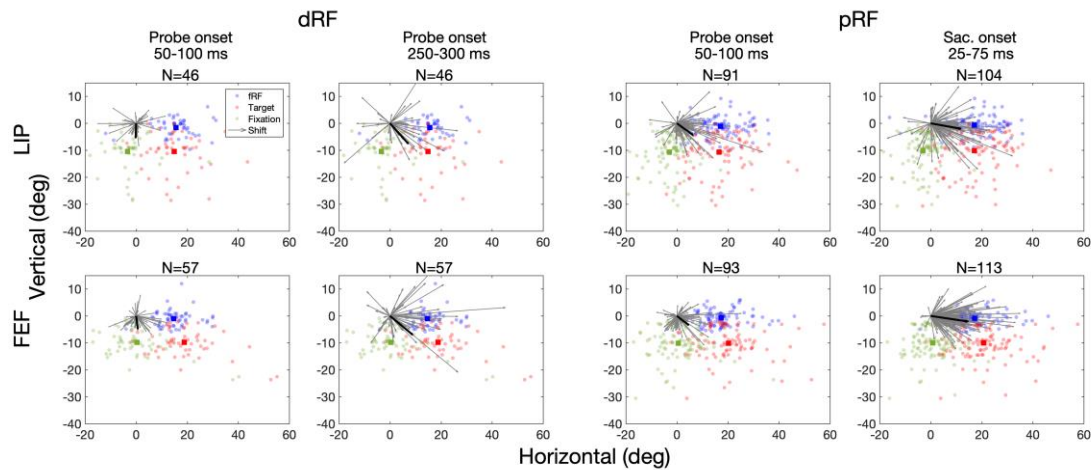


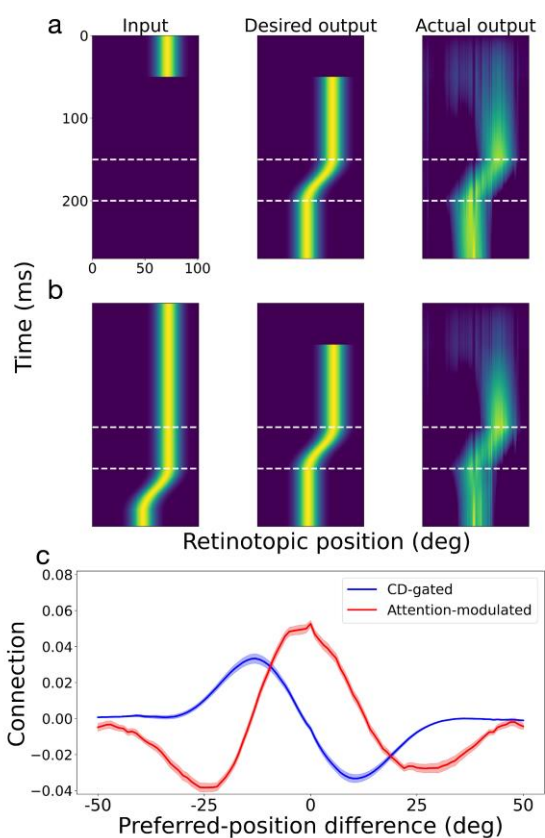
Fig. S6. The delay (a: dRF) and perrisaccadic (b: pRF) shift vectors of all LIP (top row) and FEF (bottom row) cells from different time periods (columns). This figure corresponds to Fig. 4 of the main text but shows both the shift direction and amplitude of each cell. In each panel, we align the cells' cRF centers at (0, 0) and saccade directions along positive horizontal. The cells' fRF centers, the targets, and the initial-fixation points are shown as blue, red, and green dots, respectively, and their mean positions as the blue, red, and green squares, respectively. Gray arrows indicate the cells' RF shift vectors and the black line is the vector determined by calculating the mean direction and mean amplitude of the individual vectors.

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705 In Fig. 10 of the main text, we showed the automatic emergence of both the  
 706 attention-modulated center/surround connections and the CD-gated directional  
 707 connections in artificial neural networks trained to predictively update, across saccades,  
 708 the representation of retinal locations of briefly flashed stimuli. We ran additional  
 709 simulations to show that the same was true under many other conditions, with two  
 710 examples in Figs. S7 and S8. In Fig. S7, we trained a neural network on both brief input  
 711 stimuli and persistent input stimuli. In Fig. S8, we repeated the simulation in Fig. 10 of  
 712 the main text but without the attentional modulation at the stimuli. In both cases, we  
 713 found similar connectivity patterns to those in Fig. 10. It is not surprising that attention at



the stimuli is not important for learning the connectivity patterns. To perform the task of updating the stimulus retinal positions, a network had to develop the center/surround connectivity to maintain the attractor activity pattern and the CD-gated directional connectivity to move the attractor pattern appropriately<sup>30</sup>. These requirements do not depend on attentional modulation. Once the connections are learned, attention can modulate the center/surround connectivity to enhance processing at the attended location and cause convergent RF shifts.

Fig. S7. Automatic generation of the required connectivity patterns in the circuit model by training neural networks to predictively update retinal positions of both brief (a) and persistent (b) input stimuli during saccades. The format of the figure was identical to that for Fig. 10 of the main text except that both an example of brief input (a) and an example of the persistent input (b) are shown.

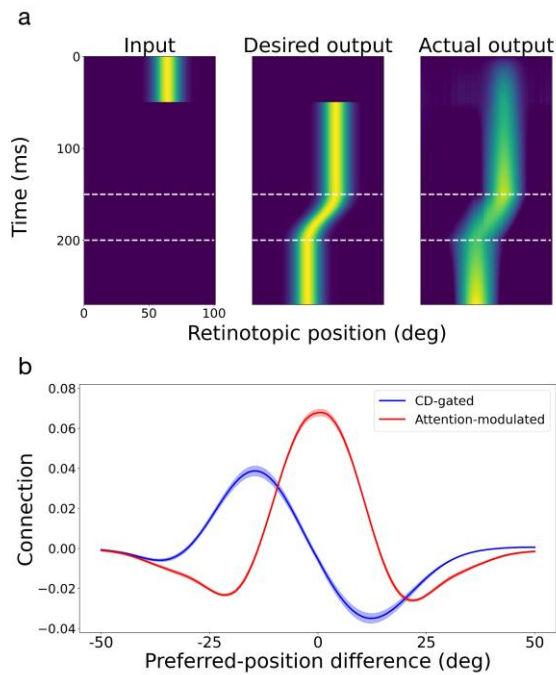


Fig. S8. Automatic generation of the required connectivity patterns in the circuit model by training neural networks to predictively update retinal positions of brief input stimuli during saccades without attentional modulation. We still labeled the symmetric connections (red) as attention-modulated for easy comparison with Figs. 10 and S7. The format of the figure was identical to that for Fig. 10 of the main text.



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736 **Author contributions**

737 MZ designed the experiments. NQ designed the models. MZ, NQ, and MEG  
738 supervised the project. CZ, LY, and MJ collected the data; XW, CZ, LY, and MJ  
739 analyzed the data. XW implemented and simulated the models. XW, LY, and MJ  
740 produced the figures. NQ, MEG, and MZ wrote the manuscript. All authors interpreted  
741 the data and edited the manuscript.

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