

1 **Synchronous spiking of cerebellar Purkinje cells during control of movements**

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13 **Running Head:** encoding of movements via synchrony

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16

17 **Abstract**

18 The information that the brain transmits from one region to another is often viewed through the lens of
19 firing rates. However, if the output neurons could vary the timing of their spikes with respect to each
20 other, then through synchronization they could highlight information that may be critical for control of
21 behavior. In the cerebellum, the computations that are performed by the cerebellar cortex are conveyed
22 to the nuclei via inhibition. Yet, synchronous activity entrains nucleus neurons, making them fire. Does
23 the cerebellar cortex rely on spike synchrony within populations of Purkinje cells (P-cells) to convey
24 information to the nucleus? We recorded from multiple P-cells while marmosets performed saccadic
25 eye movements and organized them into populations that shared a complex spike response to error.
26 Before movement onset, P-cells transmitted information via a rate code: the simple spike firing rates
27 predicted the direction and velocity of the impending saccade. However, during the saccade, the spikes
28 became temporally aligned within the population, signaling when to stop the movement. Thus, the
29 cerebellar cortex relies on spike synchronization within a population of P-cells, not individual firing rates,
30 to convey to the nucleus when to stop a movement.

31

32 **Introduction**

33 To understand how neurons in a region of the brain respond to sensory information or participate in
34 control of movements, we typically search for correlates of the sensory and motor variables in the
35 patterns of spikes. These patterns are usually quantified via the average firing rates of neurons.
36 However, there can be additional information in the timing of each spike, as exemplified by the
37 independent rate and temporal codes in the hippocampus¹, the thalamus², and the somatosensory
38 cortex³. A central question is whether neurons use spike timing to transmit functionally relevant
39 information from one region of the brain to another.

40 A special form of temporal coding is synchronization of spikes among a group of neurons. For example,
41 synchronization among glutamatergic thalamic neurons increases the efficiency of driving post-synaptic
42 neurons in the somatosensory cortex⁴. However, unlike the thalamus, the sole output from the
43 cerebellar cortex is via GABAergic Purkinje cells (P-cells). As a result, asynchronous activity of P-cells
44 inhibits the cerebellar nucleus neurons. Indeed, previous analysis of spike timing in single P-cells did not
45 find evidence that timing of spikes affected ongoing movements⁵. Yet, there are specialized
46 mechanisms in the cerebellar cortex that promote synchronization of nearby P-cells⁶, raising the
47 question of whether the cerebellum relies on synchronization to transfer information from its cortex to
48 its nuclei.

49 In principle, when a population of P-cells synchronizes their spikes, they can drive cerebellar output in a
50 way that is not possible via asynchronous spiking⁷. For example, when P-cells are synchronously
51 stimulated (in slice, and anesthetized preparations), they entrain the nucleus cells, transforming their
52 inhibitory inputs to the nucleus into production of spikes^{8,9}. This raises the possibility that analogous to
53 the thalamic input to the cerebral cortex, P-cells may rely on synchronization to convey information to
54 the nucleus, possibly affecting a specific part of the ongoing movement¹⁰.

55 Here, we focused on saccadic eye movements because they are so brief as to preclude the possibility of
56 sensory feedback, requiring the brain to rely entirely on its internal predictions¹¹⁻¹³. These predictions

57 depend critically on the cerebellum^{14,15}. For example, firing rates of populations of P-cells, but not
58 individual cells, predict the direction and velocity of the ongoing saccade^{16,17}. However, to check for
59 synchrony we needed to simultaneously record from multiple P-cells during saccades, something that to
60 our knowledge had not been accomplished in any primate species.

61 **Results**

62 We focused on marmosets, a primate that like macaques and humans relies on saccadic eye movements
63 to explore its visual scene, but is a fraction of the size of macaques, thus making it possible to record
64 from the cerebellum using short, multi-channel probes. We used MRI and CT-aligned maps of each
65 animal's cerebellum¹⁸ to guide electrodes and record from P-cells in lobule VI and VII of the vermis (Fig.
66 1C). Because there were no previous electrophysiological data from the marmoset cerebellum, we
67 searched for saccade related activity and found that P-cells in the posterior lobule VI and anterior lobule
68 VII produced simple spikes that were modulated during saccades. Thus, we focused on these regions
69 and recorded from n=149 well-isolated P-cells (Supplementary Fig. S1 provides characteristics of the
70 entire data set). Crucially, our data included n=42 pairs of simultaneously isolated P-cells that were
71 recorded from separate channels.

72 We trained the animals to fixate a central target and make a saccade to a primary target that appeared
73 at random in one of 8 directions (Fig. 1A & 1B). At the onset of the primary saccade the target was
74 erased and replaced with a secondary target, also at a random location. Following a random period of
75 fixation and delivery of reward, the secondary target was erased, the central target was displayed, and
76 the trial re-started. Whereas production of saccades accompanied modulation of simple spikes (SS), the
77 random nature of the visual stimuli produced sensory prediction errors, promoting modulation of
78 complex spikes (CS)^{16,17,19,20}.

79 Data from a pair of simultaneously recorded P-cells are shown in Figs. 1D & 1E. Despite their proximity
80 (50 μ m), one cell tended to pause its SS activity with saccades, while the neighboring cell tended to
81 pause then burst. Indeed, in both cells the SSs remained modulated long after the saccade ended.
82 However, when the target appeared to the left of the fovea, both P-cells responded with an increased
83 probability of CS (center subplot of Fig. 1D & 1E), and when the target appeared to the right, both
84 decreased their CS probability. Production of a CS in one P-cell was followed by 10-20 ms suppression of
85 SS in that cell but not the neighboring cell (Supplementary Fig. 2B and 2D). Yet, the SSs shared a degree
86 of temporal coordination: the probability of observing a SS in P-cell 2 in a 1 ms window of time increased
87 by 39.3% if P-cell 1 happened to generate a SS during the same period (Supplementary Fig. 2C).

88 To quantify SS coordination during saccades, we measured the probability of synchronization with
89 respect to chance^{6,21}. The synchronization index quantified the probability that both cells fired a spike
90 during a 1 ms interval of time, corrected for the independent probabilities of spiking in each cell (all
91 probabilities were conditioned on a saccade to a specific direction at time zero). Thus, the index
92 determined whether there was greater synchrony than expected, where chance was quantified from the
93 saccade related changes in the average firing rates of each neuron.

94 Remarkably, while both cells reduced their firing rates during saccades, their spikes became more
95 synchronized (Fig. 1F). Moreover, the probability of synchronization depended on the direction of the
96 saccade: it was greatest when the saccade was toward the direction for which complex spikes were least

97 likely (CS+180). In these two P-cells, the probability of SS synchronization reached a maximum around
98 the time when the saccade decelerated and came to a stop.

99 To analyze the data in our population, we began by measuring the CS response of each P-cell to the
100 various visual events (primary, corrective, or central target). For each event, we estimated the target
101 direction that produced the largest CS probability (CS-on, Fig. 2A). We found that the direction of CS-on
102 remained consistent across the various targets (Fig. 2C, within cell comparison of direction of CS-on in
103 response to visual event type, primary vs. secondary target $0.1^\circ \pm 5.4^\circ$ (SEM), $t(148)=0.03$, $p=0.98$;
104 primary vs. central target $-3.7^\circ \pm 5.2^\circ$, $t(148)=-0.71$, $p=0.48$, secondary vs. central target $-3.8^\circ \pm 5.3^\circ$,
105 $t(148)=-0.72$, $p=0.47$). Thus, we combined the CS response for all three visual events and used the
106 results to define the CS-on direction of each P-cell (Fig. 1A, all targets).

107 The distribution of CS-on directions across the P-cells varied widely (Fig. 2D). However, the CS-on
108 direction was not random. Rather, it varied with the location of the cell in the vermis: P-cells in the left
109 vermis tended to have right-ward CS-on, while P-cells on the right had left-ward CS-on (Fig. 2F). When
110 target directions were represented with respect to CS-on, the result was a unimodal tuning function that
111 described the CS response of P-cells following presentation of a visual target (Fig. 2E). The probability of
112 CS increased by $43.5 \pm 2.6\%$ (mean \pm SEM) above baseline when the stimulus was presented in direction
113 CS-on but decreased by $34.6 \pm 1.7\%$ below baseline when it was presented in direction CS+180.

114 Because each target instructed a saccade, we wondered whether the CS response was due to the
115 sudden onset of the stimulus or associated with the movement that followed. Although our experiment
116 was not designed to specifically answer this question, we made an interesting observation. Saccades
117 that were made in response to visual targets were preceded with large changes in CS firing rates (Fig.
118 2B, saccades to targets). However, saccades that were not instructed by a target, but were in the same
119 direction and amplitude, were preceded by significantly smaller modulation of CS firing rates (Fig. 2B,
120 “other saccades”, average CS firing rate 50ms before saccade onset in direction CS-on, paired t-test,
121 $t(296)=6.4$, $p=5 \times 10^{-10}$, direction CS+180, $t(296)=-2.8$, $p=0.005$). Thus, the complex spikes were modulated
122 primarily in response to sensory events that instructed movements, but not when similar movements
123 were made spontaneously.

124 The simple spikes exhibited a variety of patterns during saccades: some P-cells increased their activity,
125 some decreased their activity, while others produced more complicated patterns (Fig. 3A). The activity
126 patterns did not separate the cells into clusters, but rather formed a continuum (Fig. 3B). For the sake of
127 labeling, we divided the P-cells into two groups: pausers and bursters. 48% of our cells were bursters,
128 while 52% were pausers (Supplementary Fig. 3C). To quantify how well their activities were modulated
129 during saccades, for each P-cell we measured the change in SS rates aligned to saccade onset and
130 computed a z-score (Supplementary Fig. 3A). Indeed, the P-cell SS rates were modulated strongly during
131 the movements (Supplementary Fig. 3B, z-score 7.5 ± 0.3 , mean \pm SEM).

132 We next organized the P-cells based on a computational model that incorporated an important
133 anatomical feature of the cerebellum: P-cells that have similar CS tuning not only receive similar olivary
134 inputs, but they also are likely to be part of a single olivo-cerebellar module²²⁻²⁷. This anatomical
135 organization implied that to estimate activity of a population of P-cells that belonged to an olivo-
136 cerebellar module, we needed to compute saccade direction with respect to the CS-on of each P-cell. By
137 using this coordinate transformation, we estimated the population SS response in a hypothetical olivo-
138 cerebellar module when saccades were in direction CS-on, CS+90, etc. (Fig. 3C).

139 Unsurprisingly, activities of the bursters and pausers were modulated long after the saccade ended (Fig.
140 3C, top row). However, when the activities across all cells were organized into a population and
141 summed, the response exhibited a clear pattern: there was a burst that preceded saccades in all
142 directions. Notably, for direction CS+180 the burst was followed by a pause that ended near saccade
143 termination (Fig. 3C, bottom row). This burst-pause pattern was somewhat weaker for saccades in
144 direction CS±90, and the pause was missing entirely for saccades in direction CS-on.

145 The burst increased with the velocity of the impending saccade (Figs. 3D). However, the rate of increase
146 as a function of velocity was direction dependent, showing the greatest gain for saccades in direction
147 CS+180 (Fig. 3F, dir CS+180, $F(1,5)=88.3$, $p=0.0002$). This pattern is called a gain field, confirming earlier
148 findings in the macaque cerebellum¹⁶. Thus, before saccade onset the P-cells appeared to inhibit the
149 nucleus with a magnitude that depended on the velocity and direction of the forthcoming saccade.

150 In direction CS+180, the magnitude of the burst increased with saccade velocity, but its timing shifted
151 forward: the period from the peak of the burst to the onset of the saccade (Fig. 3E) became smaller as
152 saccade velocity increased (Fig. 3G, $r^2=0.65$, $F(1,5)=9.4$, $p=0.027$). As the saccade started toward
153 direction CS+180, the activity changed from a burst to a pause (Fig. 3D). However, unlike the burst that
154 preceded the saccade, the pause magnitude and timing remained invariant with respect to saccade
155 velocity (Fig. 3G, time of deceleration onset to pause peak as a function of velocity, $F(1,5)=2.4$, $p=0.18$,
156 Fig. 3D, rate of pause as a function of velocity, $F(1,5)=1.1$, $p=0.34$). Critically, despite a 7-fold change in
157 velocity, the timing of the maximum pause was unchanged with respect to saccade deceleration onset
158 (Fig. 3G). Thus, with increased saccade velocity the burst magnitude increased, and its timing shifted
159 forward. However, regardless of saccade velocity, the pause that followed the burst was time-locked to
160 the onset of saccade deceleration.

161 This invariant relationship between the timing of the pause in firing rates and the onset of saccade
162 deceleration (in direction CS+180) raised the possibility that the P-cells were signaling when the nucleus
163 cells should fire, presumably stopping the saccade. However, entraining the nucleus neurons would be
164 more efficient if the P-cells not only reduced their firing rates (thus disinhibiting the nucleus), but also
165 synchronized their spikes^{7,8}. To test this hypothesis, we computed the probability of synchronized firing
166 in our population of simultaneously recorded P-cells.

167 Production of a SS in a P-cell was associated with 31% increase in the probability (with respect to
168 chance) that there would be a simultaneous (1 ms window) SS in another P-cell (Fig. 4A, top row).
169 Similarly, production of a CS in a P-cell increased the probability of observing a CS in another P-cell at ±5
170 ms latency by 227% with respect to chance (Fig. 4A, bottom row). Finally, production of a CS in one P-
171 cell reduced the probability of SS in another P-cell at 1 ms latency by 27% (Fig. 4A, middle row). All of
172 these observations are consistent with earlier findings in mice, demonstrating that nearby P-cells not
173 only share a degree of spike synchrony⁶, but that a CS in one cell can briefly suppresses SS in another
174 cell²⁸. Furthermore, the simultaneously recorded P-cells tended to have very similar CS-on directions
175 (Fig. 4B, between cell difference $-4.4^\circ \pm 6.3^\circ$). However, did the P-cells synchronize their activities during
176 saccades?

177 Before saccade onset there was a burst in the P-cell population response. Surprisingly, probability of
178 synchronization remained at baseline (Fig. 4C, note that before saccade onset, synchronization is greater
179 than chance because at baseline the neighboring P-cells are more synchronous than chance). As the
180 saccade started and then began to decelerate (in direction CS+180) the firing rates fell, but the

181 synchronization index increased, reaching its peak probability after deceleration onset but before
182 saccade end (2.3 ± 3.5 ms after deceleration onset and -5.2 ± 4.1 ms before saccade end). That is, during
183 saccade deceleration the few spikes that remained were significantly more likely than chance to be
184 synchronized. Indeed, synchronization varied with the direction of the saccade: the greatest
185 synchronization occurred in direction CS+180 (Fig. 4D, Fig. 4D, Repeated Measure ANOVA, significant
186 effect of direction, $F(2,82)=1269.0$, $p=0.021$). Thus, the synchronization pattern was strongest in the
187 direction associated with the smallest probability of complex spikes.

188 Like targeted saccades, during other saccades the SS firing rates exhibited a burst before saccade onset,
189 with a magnitude that was largest for direction CS+180. These saccades also had a synchronization that
190 peaked before saccade end (Fig. 4C), with a probability that was largest for direction CS+180 (Fig. 4D).
191 Thus, while complex spikes showed strong modulation before targeted saccades but not task-irrelevant
192 saccades, SS firing rates were modulated during all saccades. Indeed, regardless of whether saccades
193 were target-driven or not, simple spikes reached their greatest probability of synchrony as the
194 movement decelerated and came to a stop.

195 To check whether the increased synchronization during saccades was an artifact of the change in firing
196 rates, we performed a simulation of spiking neurons that burst and paused like cells in our population,
197 but had independent probabilities of spike timing. The changes in rates during simulated saccades
198 produced a synchronization index that remained at chance (Supplementary Fig. 4).

199 Finally, we found that pairs of P-cells that had greater CS synchrony, as measured during non-saccade
200 periods, tended to have greater SS synchrony during saccades (Fig. 4E, $F(1,40)=7.6$, $p=0.009$). Pairs of P-
201 cells that had greater SS synchrony during non-saccade periods also exhibited greater SS synchrony
202 during saccades (Fig. 4E, $F(1,40)=29.0$, $p=3 \times 10^{-6}$). However, the gain of the relationship between saccade
203 period synchrony and general synchrony was significantly greater than 1 ($F(1,40)=10.7$, $p=0.002$). Thus,
204 although the timing of simple spikes among nearby P-cells was generally coordinated, during saccades
205 this coordination was greatly enhanced, especially during deceleration.

206 Discussion

207 In describing symptoms of cerebellar damage, Holmes²⁹ noted that “the most obvious errors are seen
208 toward the end of the movement [during which] the speed of the affected limb is often unchecked until
209 the object is reached or even passed.” For example, during an outward reach, many interposed nucleus
210 neurons of the cerebellum produce their greatest discharge during deceleration, with spiking activity
211 that plays a causal role in stopping the movement³⁰. Similarly, following inactivation of the fastigial
212 nucleus, extraocular motoneurons that act as saccade agonists produce an abnormally large amount of
213 activity during the deceleration period of ipsilateral movements³¹, resulting in saccades that overshoot
214 the target^{32,33}. Thus, the computations that are performed by the cerebellar cortex are critical for
215 monitoring the ongoing motor commands and predicting when the movement should be stopped. Yet,
216 P-cell simple spikes are often modulated long after the movement ends^{34–38}.

217 Here, we found that if P-cells were organized based on their complex spike response to visual stimuli,
218 their population simple spikes produced a burst-pause pattern that started before saccade onset and
219 ended with saccade termination. Changes in saccade velocity affected the timing and magnitude of the
220 burst, but the pause remained time-locked to deceleration onset. Critically, in simultaneously recorded
221 P-cells, during the pause period the probability of spike synchronization reached a maximum value. The

222 resulting inhibition-disinhibition pattern of firing rates, coupled with spike synchronization, hints that
223 the P-cells attempted to entrain the nucleus neurons specifically at the onset of deceleration^{7,9,39}.

224 What might be the behavioral consequence of this synchronization? The synchronization probability was
225 greatest for saccades that were in the direction that coincided with the least probability of complex
226 spikes (CS+180). For both saccades and limb movements, the CS tuning of a P-cell is likely aligned with
227 the direction of action of the downstream nucleus neuron^{17,40}. For example, trial-to-trial analysis of the
228 effects of complex spikes on simple spikes and behavior suggests that P-cells that have CS-on tuning to
229 the left project to nucleus neurons that have a downstream direction of action that indirectly promotes
230 production of leftward forces¹⁷. This implies that during a saccade in direction CS+180, the increased
231 synchrony combines with disinhibition (peak pause) to entrain the nucleus neurons during deceleration
232⁷, producing downstream forces that are aligned with the CS-on of the parent P-cells. As a result, the
233 effect of synchronization of P-cells, coupled with disinhibition of the nucleus, is likely the production of
234 forces that oppose the direction of movement, bringing it to a stop.

235 To our knowledge, one earlier work had reported increased spike synchrony among P-cells during
236 movements. Using multiunit signals (i.e., not single unit isolation of spikes), Heck et al.¹⁰ found
237 increased covariance between P-cells during reaching movements (in rats). That work found that
238 synchrony was most prominent as the hand approached the target, i.e., during deceleration. Here, we
239 found that P-cell firing rates and spike synchrony were coordinated, especially among populations that
240 had a common CS tuning.

241 Our results were obtained in the marmoset, a New World primate that like macaques and humans relies
242 on saccades to explore its environment. Like macaques, individual P-cells in lobule VI and VII of
243 marmosets produced simple spikes that were bursting, pausing, or a combination of the two, with no
244 obvious relationship to the direction of the saccade or its velocity. However, following onset of a visual
245 stimulus, the P-cells received information from the olive regarding the location of the stimulus with
246 respect to the fovea, producing complex spikes that were tuned to the direction of the target. In both
247 species, this tuning was anatomically organized, with P-cells in the right vermis showing highest CS
248 probability for targets to the left. A similar anatomical representation of contralateral
249 stimuli/movements of the arm has recently been noted in the vermis of mice⁴¹.

250 In both marmosets and macaques, when we organized P-cells based on their complex spike tuning
251 properties, the simple spikes produced firing rates that varied strongly with direction and velocity of the
252 movement^{16,17}. Indeed, the gain of the response with respect to velocity was highest when saccades
253 were in direction CS+180, and in both species the response was a burst followed by a pause that ended
254 as the movement came to a halt. The consistency of these results across species suggests that viewing P-
255 cell activity through the lens of population coding²⁷, i.e., a lens in which the climbing fibers organize the
256 P-cells into olivo-cerebellar modules⁴², may provide a key for unlocking the language with which the
257 cerebellar cortex encodes information.

258 How does synchronization arise during a specific phase of a movement? P-cells that show elevated
259 synchrony in their complex spikes also tend to fire simple spikes more synchronously⁴³. Indeed, here we
260 found that P-cells with greater complex spike synchrony tended to have greater simple spike synchrony
261 during saccades. In addition, we found that following a CS in one P-cell, after a 1 ms delay there was a 1-
262 2 ms period of simple spike suppression in the neighboring P-cell, confirming recent findings in mice²⁸.
263 One possibility is that P-cells that receive a common input from the olive generate a synchronized CS,

264 leading to SS suppression, which may then be followed by a synchronized resumption of SS firing⁴⁴.
265 However, SS synchrony was greatest in direction CS+180, i.e., the direction for which there was the
266 smallest probability of CS. Furthermore, complex spikes showed modulation before the onset of
267 targeted saccades but not task-irrelevant saccades, whereas simple spikes showed synchronization for
268 both types of saccades. These observations make it seem unlikely that during saccades, presence of
269 complex spikes played a role in synchronization of simple spikes.

270 Synchrony is also present in P-cells that are likely to have common parallel fiber inputs (on-beam) but
271 different climbing fibers^{10,45}. Indeed, here we found that nearby P-cells not only had greater than
272 chance levels of synchrony, but that cells with greater SS synchrony in general had much greater than
273 expected SS synchrony during saccades. Thus, it is possible that SS synchrony arises from a shared input
274 from ascending granule cell axons that are positioned directly beneath the P-cells and receive inputs
275 from the same mossy fibers¹⁰. However, why this synchrony would be focused during the deceleration
276 phase, particularly for saccades in direction CS+180, is unclear. Because granule cells also recruit
277 molecular layer interneurons⁴⁶, synchronization of P-cells may engage a network wide organization to
278 overcome inhibition by the basket and stellate cells⁴⁷.

279 In a typical artificial neural network, information transfer from one layer to the next is via firing rates of
280 neurons, and learning modifies synaptic weights to change the activity of each neuron and minimize
281 error in the output layer. The cerebellum resembles a 3-layer network where learning is at least partially
282 guided by the climbing fibers^{27,48}. Our results demonstrate that the information that is transmitted from
283 the P-cells to the nucleus is encoded in an exquisite coordination of firing rates and synchronization. This
284 implies that when there is error in performance, cerebellar learning cannot simply focus on changing the
285 P-cell firing rates. Rather, learning must also alter network wide synchronization. This conjecture
286 predicts that complex spikes that arise following movement errors not only promote learning via
287 changes in the activity of individual P-cells^{17,49,50}, but may also alter the synchronization patterns of
288 populations of P-cells. Learning to transfer information via synchronization is an exciting new direction
289 with which to explore the function of the cerebellum.

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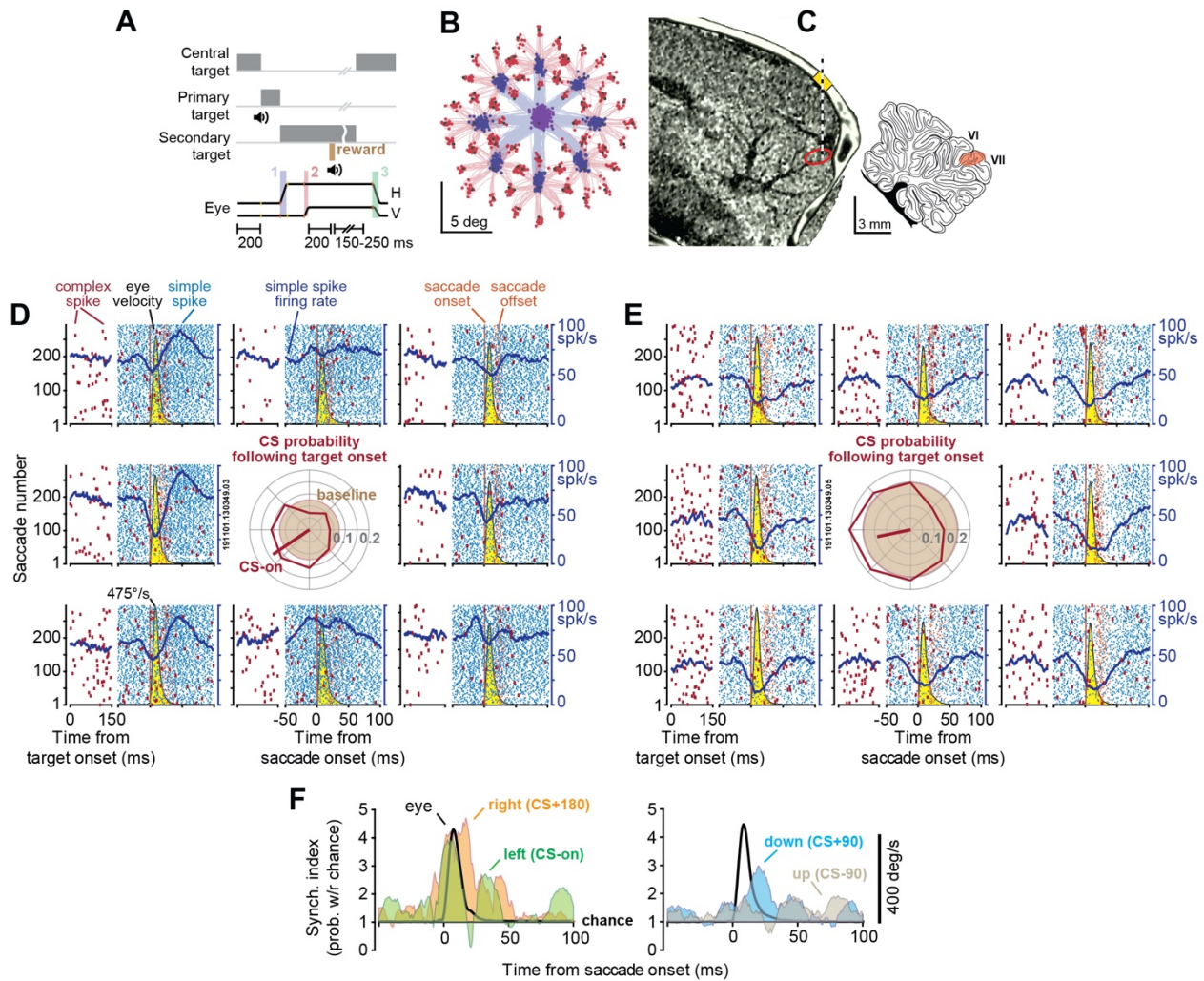
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294 **Author contributions:** E.S.N., J.S.P., P.H., and R.S. conceived and performed experiments. E.S.N., J.S.P.,
295 M.A.F., and R.S. analyzed data, E.S.N. made figures, performed statistical analysis, and performed
296 simulations, R.S. and E.S.N. wrote the manuscript.

297 **Competing interests:** None.

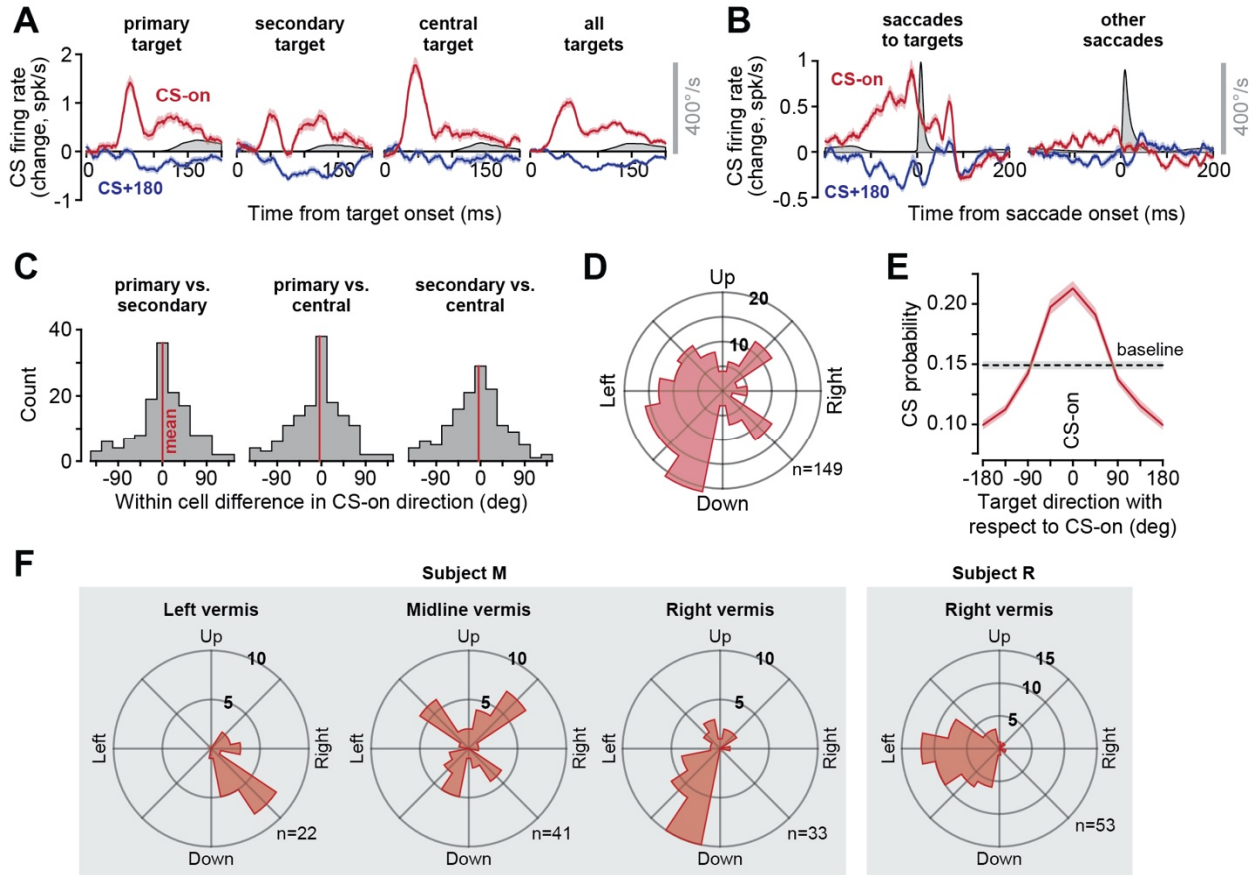
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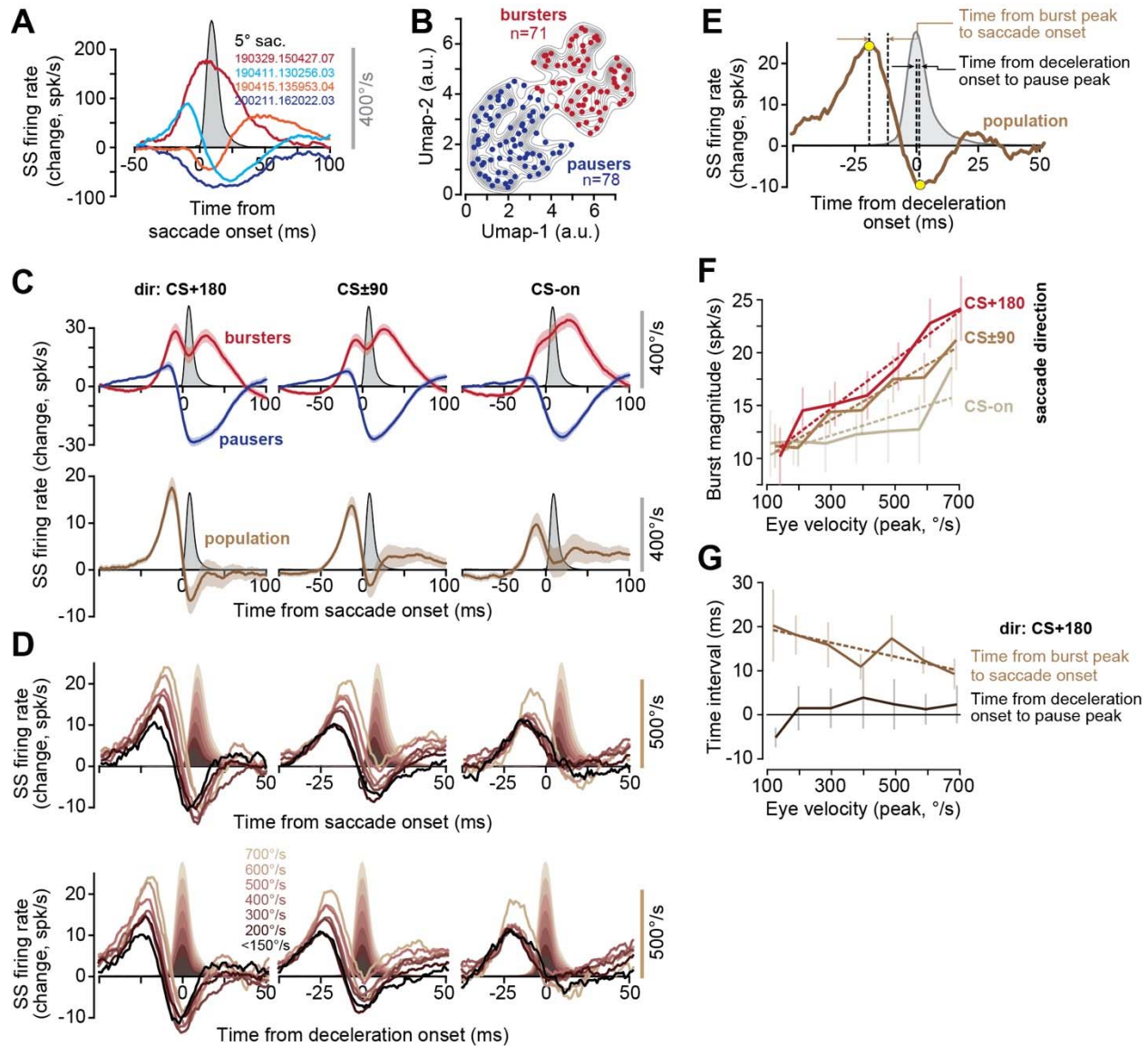
301 **Fig. 1.** P-cells synchronized their simple spikes during saccades. **A.** Experimental paradigm. Marmosets
 302 were trained to make saccades to visual targets that appeared randomly at one of 8 directions. Onset of
 303 the primary saccade (labeled 1 in the lowest trace) resulted in the replacement of the primary target
 304 with a secondary target, also at a random direction. Following the secondary saccade (labeled 2), and a
 305 fixation period, reward was delivered, and the center target was displayed, resulting in a centripetal
 306 saccade (labeled 3). **B.** Eye position for the primary (blue) and secondary (red) saccades in a typical
 307 experiment. **C.** We used the MRI and CT images of each animal to guide the electrodes to lobule VI or VII
 308 of the cerebellar vermis. **D & E.** Simple (blue) and complex spikes (red) in two simultaneously recorded
 309 P-cells during saccades to various directions. Eye velocity is shown via the yellow curve. The complex
 310 spikes are also aligned to the onset of the visual target. Both cells exhibited a reduction in simple spikes
 311 during saccades, with a modulation pattern that lasted much longer than the saccade. CS probability
 312 during the 200ms following target onset is quantified via the center plot. Baseline CS probability is
 313 shown by the brown circle at center. The target direction that produces the highest CS probability (CS-
 314 on) is estimated by the red line at center. **F.** Synchronization index during saccades to various directions.
 315 This index quantified the probability of synchronization with respect to chance at 1 ms time bins. Eye
 316 velocity is indicated by the black curve. Probability of synchronization is greatest for saccades in
 317 direction CS+180, reaching a peak at around saccade deceleration.



318

319 **Fig. 2.** Complex spikes exhibited tuning with respect to the direction of target, and this tuning was
 320 anatomically organized. **A.** CS response aligned to target onset. For each type of target, the direction of
 321 stimulus that produced the greatest probability of CS was labeled as CS-on. Eye velocity is shown in gray.
 322 **B.** CS response aligned to saccade onset. Modulation of CS response was present before saccades that
 323 were visually instructed. The response was muted before “other saccades”. **C.** Within cell difference
 324 between CS-on directions as computed following the onset of the primary target, the secondary target,
 325 and the central target. We found no systematic differences in the estimate of CS-on between various
 326 types of targets, and thus combined the response for all targets to compute the CS-on of each P-cell. **D.**
 327 Distribution of CS-on across the population of P-cells. **E.** CS tuning function. **F.** Distribution of directions
 328 of CS-on in various regions of the vermis in two animals. Error bars are SEM.

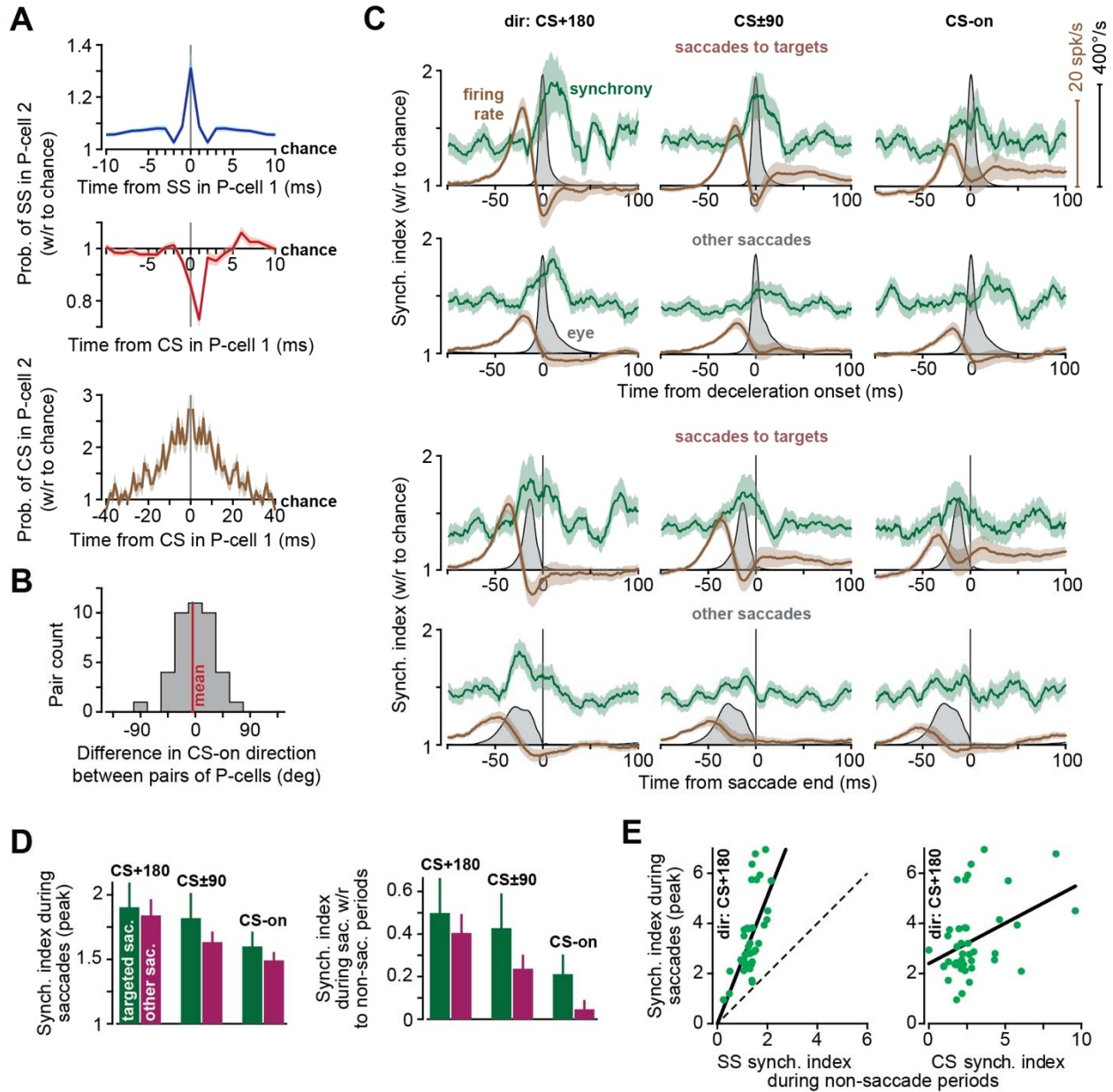
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331 **Fig. 3.** Population response of simple spikes encoded saccade direction, peak velocity, and the onset of
 332 deceleration. **A.** Average change in the firing rates of four representative P-cells with respect to baseline,
 333 during saccades (data collapsed across all directions). **B.** Clustering of saccade-aligned change in firing
 334 rates for all P-cells, using the algorithm UMAP⁵¹. Separating the data into two clusters produces
 335 bursters (red) and pausers (blue). **C.** Activities of the bursters and pausers during saccade in various
 336 directions. The population response is the sum of firing rates in all P-cells. **D.** Population response
 337 aligned to saccade onset and deceleration onset. The burst tends to grow with saccade velocity and
 338 shifts forward in time, but the pause remains invariant with respect to the onset of deceleration. **E.**
 339 Quantification of the population response with respect to saccade kinematics. **F.** Magnitude of the burst
 340 before saccade onset as a function of saccade peak velocity in various directions. **G.** Timing of the burst
 341 with respect to saccade onset decreased with increased velocity, while the timing of the pause with
 342 respect to deceleration onset remained invariant.

343

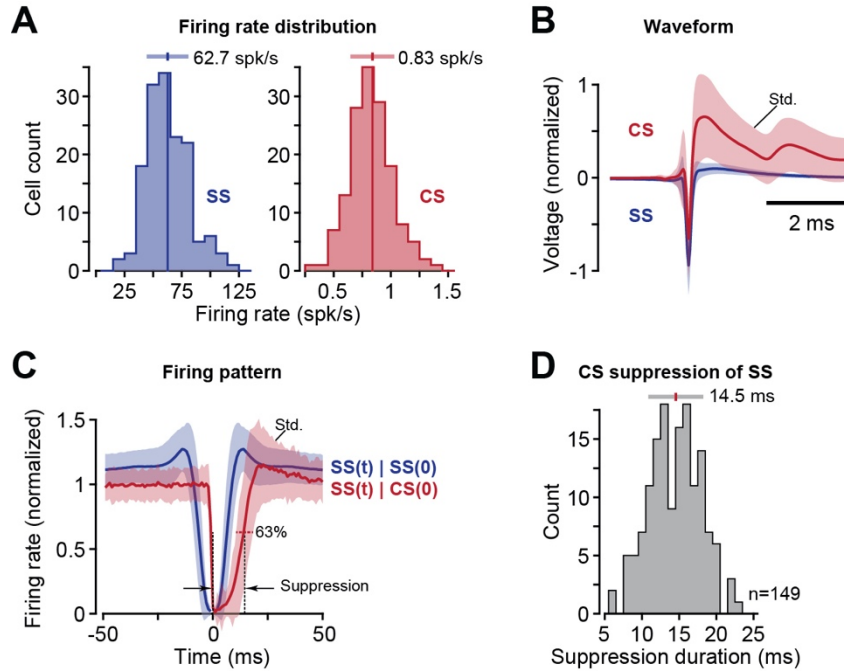


344

345 **Fig. 4.** P-cells synchronize their spikes during saccade deceleration. **A.** Probabilities of spike
 346 synchronization in pairs of P-cells during the entire recording session (41±2 minutes, mean±SEM). Top:
 347 probability of simple spike in P-cell 2 at time point t (with respect to chance), given that a simple spike
 348 occurred in P-cell 1 at time zero. Middle: probability of simple spike in P-cell 2, given that a complex
 349 spike was produced in P-cell 1 at time zero. Bottom: probability of complex spike in P-cell 2 given that a
 350 complex spike was produced in P-cell 1 at time zero. Bin size is 1 ms. **B.** Difference in CS-on directions
 351 among pairs of simultaneously recorded P-cells. **C.** Synchronization index (green) and firing rates
 352 (brown) for targeted saccades and other saccades. In the top two rows, data are aligned to deceleration
 353 onset. In the bottom two rows, data are aligned to saccade end. Firing rate is the population response.
 354 Bin size is 1 ms. **D.** The magnitude of the synchronization index during saccades (peak value) for
 355 saccades in various directions. **E.** Left plot shows the magnitude of the synchronization index during
 356 saccades (peak value) in direction CS+180, with respect to SS synchronization index as measured during

357 non-saccade periods. Dashed line is identity. Center plot shows the synchronization index during
358 saccades with respect to complex spike synchronization index as measured during non-saccade periods
359 (1ms bin for SS and 10ms bin for CS). Error bars are SEM.

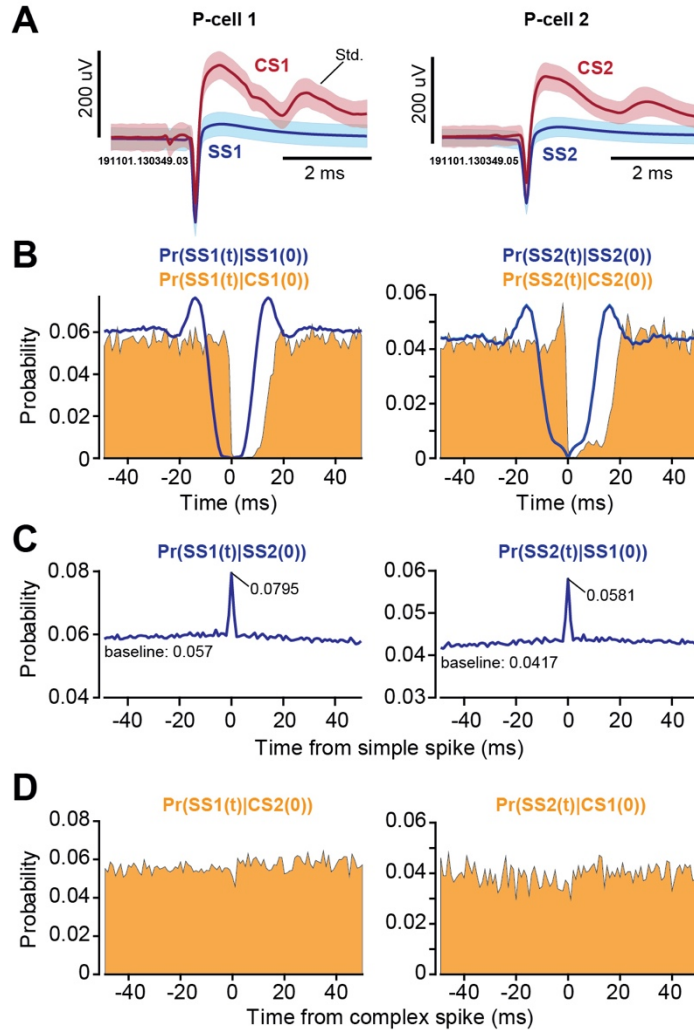
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361

362 **Supplementary Fig. S1.** Properties of saccade-related P-cells (n=149) in the marmoset vermis lobule VIa
363 and VIIa-c. **A.** Distribution of average firing rates for simple spikes (blue) and complex spikes (right). The
364 bar at the top indicates mean and standard deviation. **B.** Waveforms for the simple and complex spikes.
365 The waveforms were normalized by setting the cell's mean voltage to 0 and the maximum negative
366 going simple spike deflection to -1. Error bars are standard deviation. **C.** Within-cell interactions
367 between simple and complex spikes. The blue curve shows the firing rate of simple spikes at time t,
368 given that the cell produced a simple spike at time zero, labeled as SS(t) | SS(0). The red curve shows the
369 firing rate of simple spikes at time t, given that the cell produced a complex spike at time zero, labeled
370 as SS(t) | CS(0). Simple spike rates for each P-cell were normalized with respect to average simple spike
371 firing rate as computed over the entire recording session. Error bars are standard deviation. **D.**
372 Suppression period of simple spikes following production of a complex spike. Suppression period for
373 each P-cell was defined as the duration of time after a complex spike that was required before the
374 simple spike firing rate recovered 63% of its pre-complex spike value. The bar at the top indicates mean
375 and standard deviation.

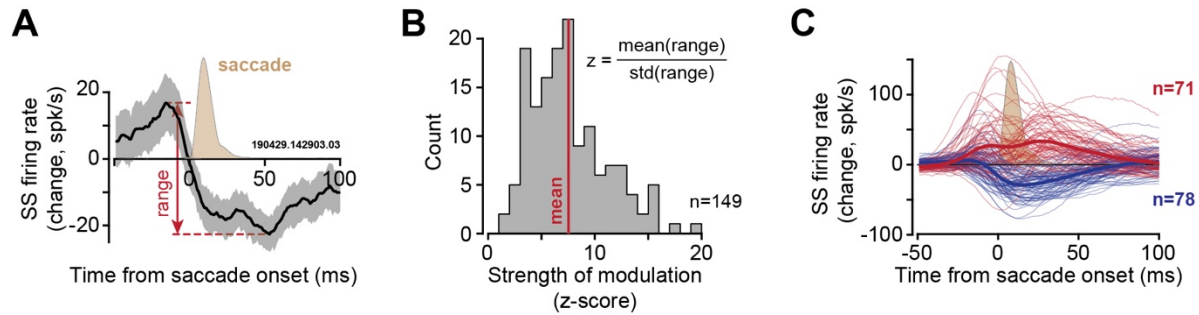
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378 **Supplementary Fig. S2.** Spike timing properties of a sample pair of simultaneously recorded P-cells.
 379 These are the same cells as in Fig. 1. **A.** Simple and complex spike waveforms. Error bars are standard
 380 deviation. **B.** The curve $\Pr(SS1(t)|SS1(0))$ quantifies the probability of a simple spike in P-cell 1 at time t ,
 381 given that P-cell 1 produced a simple spike at time zero. This quantifies the simple spike refractory
 382 period. The curve $\Pr(SS1(t)|CS1(0))$ quantifies the probability of production of a simple spike in P-cell 1
 383 at time t , given that P-cell 1 produced a complex spike at time zero. This indicates the complex spikes
 384 induced suppression of simple spikes. **C.** The curve $\Pr(SS1(t)|SS2(0))$ quantifies the probability of
 385 production of a simple spike in P-cell 1 at time t , given that a simple spike was produced by P-cells 2 at
 386 time zero. **D.** The curve $\Pr(SS1(t)|CS2(0))$ quantifies the probability of production of a simple spike in P-
 387 cell 1 at time t , given that a complex spike was produced by P-cells 2 at time zero. Bin size is 1 ms in the
 388 probability plots.

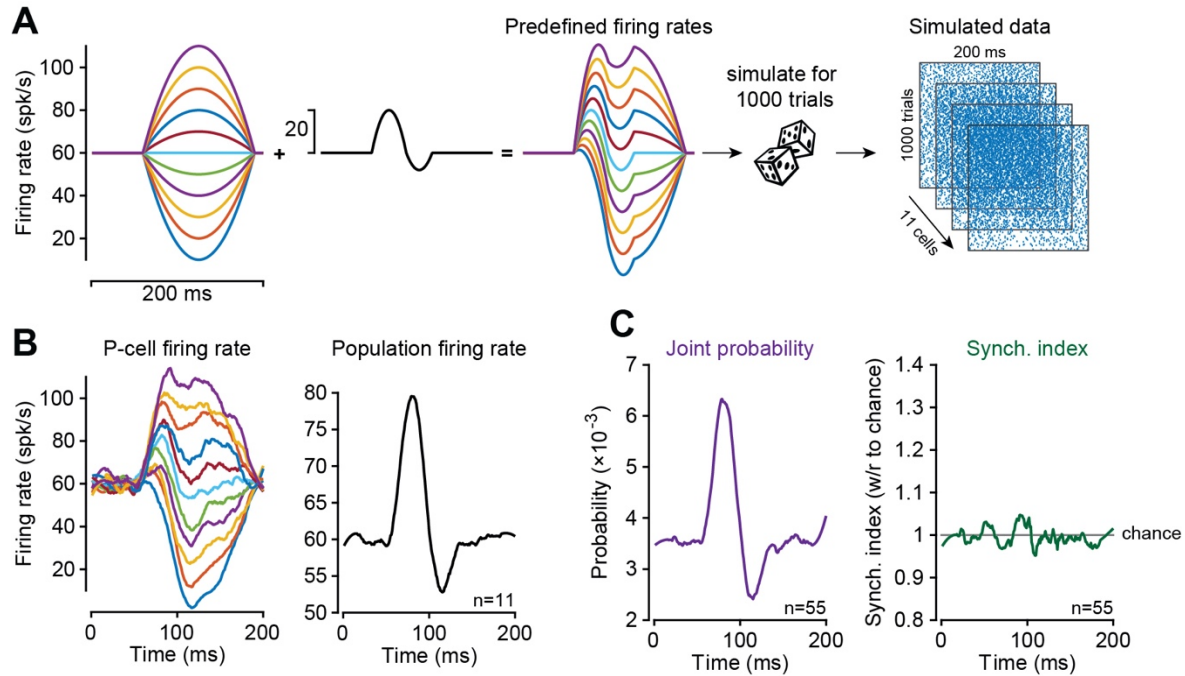
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391 **Supplementary Fig. S3.** Modulation of simple spikes in individual P-cells during saccades. **A.** Data from a
392 single P-cell, quantifying the change in simple spike rates, aligned to saccade onset. Range was defined
393 as the maximum change in firing rate in the pre- to post-saccade period. The light brown curve shows
394 the average saccade velocity (peak value is 475 deg/s). Error bars are standard deviation computed via
395 bootstrapping. **B.** Strength of saccade-related modulation of each P-cell was defined via a z-score. The z-
396 score computed the mean of the range of the firing rates divided by the standard deviation of the range
397 (7.5 ± 0.3 mean \pm SEM). Strongly modulated P-cells were those that had a z-score greater than 3,
398 composing 96% (142 out of 149) of the cells in the population. **C.** Change in simple spike rates with
399 respect to baseline for the bursters (red) and pausers (blue). Baseline firing rate is defined as the
400 average firing rate as measured during the entire recording session.

401



402

403 **Supplementary Fig. S4.** To check whether the increased synchronization index during saccades was an
404 artifact of the change in firing rates of P-cells, we performed a simulation of spiking neurons that burst
405 and paused like the cells in our population. However, the simulated cells had independent probabilities
406 of spiking. **A.** The simulated population consisted of 11 neurons ranging from bursting to pausing in their
407 activity. The plots show the construction of the average firing rates. We started with a unimodal
408 function of various amplitudes, added the response in the middle row to each neuron, producing the
409 data on the right, representing the instantaneous average firing rate $\bar{r}_i(t)$ of each simulated neuron. **B.**
410 Data from 1000 simulated trials. The plot shows the average firing rate of each simulated cell. **C.** The
411 population firing rate of the simulation. **C.** Joint probability, and synchrony index of 55 pairs of cells (11
412 cells taken 2 at a time). Joint probability reflected modulation of firing rates, but because spike timing in
413 each neuron was independent, the synchrony index remained at chance level.

414

415 **Methods**

416 Neurophysiological data were collected from two marmosets (*Callithrix Jacchus*, male and female, 350-
417 370 g, subjects M and R, 6 years old. The marmosets were born and raised in a colony that Prof. Xiaoqin
418 Wang has maintained at the Johns Hopkins School of Medicine since 1996. The procedures on the
419 marmosets were evaluated and approved by the Johns Hopkins University Animal Care and Use
420 Committee in compliance with the guidelines of the United States National Institutes of Health.

421 *Data acquisition*

422 Following recovery from head-post implantation surgery, the animals were trained to make saccades to
423 visual targets and rewarded with a mixture of applesauce and lab diet¹⁸. Visual targets were presented
424 on an LCD screen (Curved MSI 32" 144 Hz - model AG32CQ) while binocular eye movements were
425 tracked using an EyeLink-1000 eye tracking system (SR Research, USA). Timing of target presentations
426 on the video screen was measured using a photo diode.

427 We used the MRI and CT imaging data for each animal and designed an alignment system that defined
428 trajectories from the burr hole to various locations in the cerebellar vermis, including points in lobule VI
429 and VII. We used a piezoelectric, high precision microdrive (0.5 micron resolution) with an integrated
430 absolute encoder (M3-LA-3.4-15 Linear smart stage, New Scale Technologies) to advance the electrode.

431 We recorded from the cerebellum using four types of electrodes: quartz insulated 4 fiber (tetrode) or 7
432 fiber (heptode) metal core (platinum/tungsten 95/05) electrodes (Thomas Recording), and 64 channel
433 checkerboard or linear high density silicon probes (M1 and M2 probes, Cambridge Neurotech). We
434 connected each electrode to a 32 or 64 channel head stage amplifier and digitizer (RHD2132 and
435 RHD2164, Intan Technologies, USA), and then connected the head stage to a communication system
436 (RHD2000 Evaluation Board, Intan Technologies, USA). Data were sampled at 30 kHz and band-pass
437 filtered (2.5 - 7.6 kHz). We used OpenEphys⁵², an open-source extracellular electrophysiology data
438 acquisition software, for interfacing with the RHD2000 system and recording of signals.

439 *Behavioral protocol*

440 Each trial began with fixation of a center target for 200 ms, after which a primary target (0.5x0.5 deg
441 square) appeared at one of 8 randomly selected directions at a distance of 6.5 deg. Onset of the primary
442 target coincided with presentation of a distinct tone. As the animal made a saccade to this primary
443 target, that target was erased, and a secondary target was presented at a distance of 2-2.5 deg, also at
444 one of 8 randomly selected directions. The subject was rewarded if following the primary saccade it
445 made a corrective saccade to the secondary target, landed within 1.5 deg radius of the target center,
446 and maintained fixation for at least 200 ms. Onset of reward coincided with presentation of another
447 distinct tone. Following an additional 150-250 ms period (uniform random distribution), the secondary
448 target was erased, and the center target was displayed.

449 *Data analysis*

450 All saccades, regardless of whether they were instructed by presentation of a visual target or not, were
451 identified in the behavioral data using a velocity threshold. Saccades to primary, secondary, and central
452 targets were labeled as targeted saccades, while all other saccades were labeled as task irrelevant.

453 Electrophysiological data were sorted into spikes using P-sort⁵³, a newly developed open-source
454 software that we developed specifically for identification of simple and complex spikes. P-sort provides
455 tool to help confirm that the complex and simple spikes originate from the same P-cell. Briefly, we
456 compared the conditional probability $Pr(S(t)|C(0))$ with $Pr(S(t)|S(0))$. For example, $Pr(S(t)|C(0))$
457 is the probability that a simple spike occurred at time t , given that a complex spike was generated at
458 time zero. $Pr(S(t)|S(0))$ is the probability that a simple spike occurred at time t , given that a simple
459 spike was generated at time zero. Simple spikes that originate from a single P-cell produce a refractory
460 period. Thus, $Pr(S(t)|S(0))$ exhibits a low probability period of roughly 5 ms in duration after time
461 zero. On the other hand, a complex spike coincides with suppression of future simple spikes, but not
462 those that occurred before. As a result, $Pr(S(t)|C(0))$ is asymmetric, with a long period of low simple
463 spike probability (around 15 ms) following time point zero. Examples are provided in Supplementary Fig.
464 S2.

465 Simple and complex spike baseline firing rates were computed by dividing the total number of spikes by
466 the duration of the entire recording. Simple and complex spike instantaneous firing rate were calculated
467 from peri-event time histograms with 1 ms bin size. Events of interest included: visual events (target
468 onset), saccade onset, deceleration onset, and saccade offset. We used a Savitzky–Golay filter (2nd
469 order, 31 datapoints) to smooth the traces for visualization purposes.

470 Complex spike tuning was computed by measuring the CS probability following target onset. We
471 counted the number of complex spikes after target onset up to saccade onset or a fixed 200 ms window,
472 whichever happens first. This approach ensured that the complex spikes during saccades or after
473 saccade offset did not get included in the measurements. Dividing the spike count by the number of
474 events resulted in the CS probability in each direction.

475 Suppression duration of simple spikes following a complex spike (Supplementary Fig. S1C) was
476 computed by measuring the period during which the simple spikes recovered 63% of their pre-complex
477 spike firing rate.

478 To compute population response during saccades, we began by computing the change in simple spike
479 firing rate of each P-cell with respect to its baseline. Next, we labeled each saccade by measuring its
480 direction with respect to the CS-on of the recorded P-cell. Finally, we summed the activities in all P-cells
481 (i.e., changes with respect to baseline) for saccades in direction CS-on, CS+45, etc., using a bin size of
482 ± 25 deg.

483 *Analysis of the simultaneously recorded P-cells*

484 Multi-channel electrodes allowed for analysis of simultaneously recorded neurons. However, spiking
485 activity in one neuron can easily influence the data recorded by two nearby channels, thus giving an
486 illusion that the two channels are picking up two distinct neurons. To guard against this, after we sorted
487 the data in each channel, we waveform triggered the data recorded by channel A by the spikes recorded
488 on channel B. This identified the waveform of the neuron recorded by channel B on the spike recorded
489 on channel A. We compared this cross-channel triggered waveform with the within channel triggered
490 waveform generated by the spikes recorded by channel A. The cross-channel triggered waveform must
491 produce a different cluster of spikes in A than the main neuron isolated by A. If there were spikes that
492 occurred within 1 ms of each other on channels A and B, we used these coincident-spike events to
493 trigger the waveform in A. The spikes in A that were identified to be coincident with B should look

494 approximately the same as the non-coincident spikes in A. Examples of this approach are provided in
495 Sedaghat-Nejad et al.¹⁸.

496 To quantify coordination between activities of two P-cells, we computed joint probabilities, corrected
497 for chance^{6,21}. We computed $\Pr(S2(t), S1(0)) / (\Pr(S2) \Pr(S1))$, which is equal to $\Pr(S2(t)|S1(0)) /$
498 $(\Pr(S2))$. This quantified whether the occurrence of a simple spike on channel 1 at time zero altered the
499 probability of simple spikes on channel 2 at time t, corrected for probabilities expected from their
500 average firing rates. Because channel labels 1 or 2 are interchangeable, we considered the average of
501 the two cases as the corrected conditional probability for a pair of P-cells. We implemented a similar
502 analysis to quantify the coordination between complex spikes in two cells, or complex spikes in one cell
503 and simple spikes in another cell.

504 To compute the probability of synchronization of simple spikes during saccades, we began by computing
505 the joint probability of spiking at time t, given that a saccade took place at time zero in a particular
506 direction, $\Pr(S1(t), S2(t)|sac(0))$. To correct for the fact that firing rates changed during the saccade,
507 we divided the joint probability by the independent probabilities of spike production in each cell,
508 measured when a saccade took place in the given direction at time zero. Thus, the synchronization index
509 was defined for each saccade direction as:

$$SI = \frac{\Pr(S1(t), S2(t)|sac(0))}{\Pr(S1(t)|sac(0)) \Pr(S2(t)|sac(0))}$$

510 *Modeling*

511 To check whether the increased synchronization index during saccades was an artifact of the change in
512 firing rates of P-cells, we performed a simulation of spiking neurons that burst and paused like cells in
513 our population but had independent probabilities of spike timing. We pre-defined the firing rate pattern
514 for 11 hypothetical neurons all with a 60 spk/s baseline firing rate (Supplementary Fig. S4A) and then
515 add a 130 ms duration modulation. The result produced a population response firing pattern that
516 mimics the P-cells in our dataset (Supplementary Fig. S3C). Using a Bernoulli process and pre-defined
517 firing rates, we simulated the spiking activity for the 11 hypothetical neurons for 1000 trials. Next, we
518 used the methods described above to compute the estimated firing rate of each cell and the population
519 response (Supplementary Fig. S4B). Finally, we computed the joint probability as well as the
520 synchronization index between 55 (2 choose 11) pairs of cells (Supplementary Fig. S4C). Our results
521 confirmed that while the joint probability was modulated according to the change in firing rates in the
522 population of cells, the synchronization index stayed at chance level.

523

524

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