

1 **TITLE PAGE**

2 **Title:** Population temporal structure supplements the rate code during sensorimotor transformations

3 **Abbreviated title:** Population temporal dynamics in gaze control

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17 **Author Contributions:** U.K.J and N.J.G designed the study. U.K.J. performed the experiments, analyzed
18 the data, and performed model simulations. U.K.J and N.J.G wrote the manuscript.

19 **Number of pages:** 25

20 **Number of figures:** 4 + 6 extended figures

21 **Number of words:** Abstract - 345, Main text - 2078, Supplementary text - 669, Methods - 2895

22 **Conflict of interest:** The authors declare no competing financial interests.

23 **Acknowledgements:** We thank Drs. A. Batista, C. Olson, M. Smith, and B. Yu for scientific discussions and
24 critical feedback on previous versions of the manuscript and J. McFerron for programming assistance.
25 The study was funded by the following NIH R01 grants: EY022854 and EY024831 awarded to N.J.G.

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36 **Abstract**

37 In order to successfully interact with the environment the brain must funnel down the sensory inputs it
38 receives to specific movements at specific times. Such sensory-to-motor transformations are critically
39 mediated by neurons in premotor brain networks whose evolving activities represent sensory, cognitive,
40 and movement-related information¹⁻³. However, this multiplexing poses a conundrum – how does a
41 decoder know precisely when to initiate a movement if such neurons are also active at times other than
42 when a movement occurs (e.g., in response to sensory stimulation)? Extant models of movement
43 generation that rely on firing rate, including rise-to-threshold⁴, inhibitory gating⁵, and dynamical
44 switches at the population level^{6,7}, leave certain explanatory gaps unfilled. Here, we propose a novel
45 hypothesis: movement is triggered not only by an increase in firing rate, but critically by a reliable
46 temporal pattern in the population response. We show that in brain regions involved in orienting eye
47 movements - the superior colliculus (SC) and the frontal eye fields (FEF) - the temporal dynamics
48 between neurons during visually-driven activity is different from that during pre-movement activation.
49 Specifically, using a measure that captures the fidelity of the population code - here called temporal
50 stability - we show that the temporal structure fluctuates in the visual response but remains stable
51 during the pre-movement response, thus distinguishing incoming sensory input from motor output. This
52 is an important attribute because SC and FEF “visuomovement” neurons project directly to the
53 brainstem^{8,9} which houses the controller for gaze shifts, and any increase in the incoming drive is poised
54 to trigger a (potentially undesirable) gaze shift. We also demonstrate that a simple firing rate-based
55 network with synaptic facilitation can distinguish between stable and fluctuating population codes,
56 suggesting a putative mechanism for interpreting temporal structure. These findings offer an alternative
57 perspective on the relationship between spatial attention and motor preparation^{10,11} by situating the
58 correlates of movement initiation in the temporal features of activity in shared neural substrates. They
59 also suggest a need to look beyond the instantaneous rate code and consider the effects of short-term
60 population history on neuronal communication and its effects on behaviour.

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73 Main text

74 Across brain regions involved in the control of gaze, including the SC in the midbrain and the FEF in the
75 neocortex, so-called visuomovement neurons burst a volley of spikes both in response to a visual
76 stimulus and for generating a gaze shift or saccade to the spatial location of the stimulus¹. The dual
77 nature of visuomovement neurons is best illustrated by examining their activity in the delayed response
78 paradigm (Figure 1A). In this task, the subject is required to withhold a saccade in response to the
79 presentation of a stimulus in the visual periphery until after the disappearance of a central fixation cue.
80 Figure 1C shows the average activity aligned on target (left) and saccade (right) onsets for 57 SC and 22
81 FEF neurons in two monkeys (*Macaca mulatta*). The visual response exhibits a high frequency burst,
82 reaching levels observed 30-40 ms prior to saccade onset, yet the former rise does not trigger a
83 movement while the latter does. In fact, it is not uncommon to find neurons whose peak visual response
84 is greater than peak pre-movement activation¹², an observation that casts doubt on thresholding⁴ as a
85 singular mechanism. Given that such neurons project directly to the brainstem saccade burst generator
86 that initiates and guides saccadic gaze shifts^{8,9} (Figure 1B), we asked how downstream structures are
87 able to differentiate between the two bursts.

88 Previous studies have examined the across-trial reliability of neural responses as a correlate of
89 behavioural states¹³⁻¹⁵, including movement preparation¹⁶. We computed the Fano factor, a ratio of the
90 spike count variance to the mean across trials. In both SC and FEF, the Fano factor decreased prior to
91 saccade onset (Figure 1D), but showed a mixed pattern during the visual burst following stimulus onset
92 (decrease in FEF and a transient increase in SC). Thus, spike count reliability alone is insufficient to
93 explain the lack of a saccade during the high-frequency visual burst. More importantly, since reliability is
94 necessarily computed across trials, the brain cannot use this information to make movement decisions
95 on any given trial.

96 Zooming in on the activity profiles of individual neurons at the time of the visual and premotor bursts
97 (Figure 2A, insets) sheds light on a possible solution to this puzzle. Qualitatively, the neurons' firing
98 profiles seem to be unstructured in the visual burst, while they rise more coherently in the premotor
99 burst. We hypothesized that the degree to which the relative levels of activation across the population is
100 preserved over the course of a burst controls saccade initiation. To quantify this, we created a pseudo-
101 population of our single-unit recordings and constructed a trajectory of population activity over time.
102 We normalized the population vector at each time point, which constrains it to a unit hypersphere in
103 state space. This step factors out global changes in firing across the population and focuses on the
104 relative activity levels. We then computed the dot product between two of these unit vectors separated
105 in time (20 ms here) - we call this the temporal stability of the population code. This procedure is
106 schematized in Figure 2B (for details, see Online Methods and Extended Data Figure 1). Figure 2C shows
107 the evolution of temporal stability of the SC and FEF populations (solid and dotted traces, respectively,
108 inset shows individual animals). In both regions, the population code is relatively unstable during the
109 visual burst and remains stable during the premotor burst. This was true across a range of separation
110 times between the population vectors (EDF 2). Thus, the stability across the population of
111 visuomovement neurons seems to function as a switch that prevents movement initiation at an
112 undesirable time (visual epoch), allowing the animal to successfully perform the task at hand (while
113 presumably also allowing the activity to be routed to other brain areas for further processing). Once
114 cued to execute a gaze shift, the activity in the same population rises in a stable manner allowing
115 saccade initiation. Scrambling the population code by either shuffling the activations of individual

116 neurons at each time point or the temporal sequence of population activity drastically alters the stability
117 profile (EDF 3), further suggesting that the above result is due to the specific temporal structure in the
118 recorded population.

119 Next, we explored the robustness of the temporal stability hypothesis. Neurons in the rostral SC are
120 tonically active during fixation¹⁷ and burst for small saccades and microsaccades¹⁸, but they reduce their
121 activity during larger movements¹⁷ (Figure 3A,B). Importantly, they also project to the saccade burst
122 generator¹⁹. Assuming population stability sets part of the input drive to downstream structures, we
123 hypothesized that the temporal structure in rostral SC neurons must decrease during large saccades but
124 remain elevated during fixation, even when a visual burst occurs in other parts of SC and FEF. In other
125 words, we expected the evolution of temporal stability across rostral SC neurons to be the inverse of
126 what occurs in caudal SC - Figure 3C confirms the prediction. Since rostral SC is also known to play a
127 causal role in the generation of microsaccades, we considered whether the population of rostral SC
128 neurons that burst for microsaccades had a stable temporal structure. This was indeed the case, as seen
129 in EDF 4. Thus, the population activity of neurons in rostral SC, including its temporal structure,
130 supplements the pattern in other parts of SC in suppressing and initiating movements. We also
131 examined the stability of the population in the ipsilateral SC with a response field opposite the location
132 of the saccade target (EDF 5). The results were similar to the profile observed for rostral SC, consistent
133 with the idea of reducing the drive from competing neuronal populations. Moreover, pooling neurons in
134 SC and FEF to form a combined population (since they all project to saccade-generating structures) did
135 not impact the main result (EDF 6).

136 Note that the delayed saccade task is designed to temporally separate the visual and premotor bursts.
137 In the gap task (Figure 3D), in contrast, the two bursts often overlap and saccade reaction times are
138 reduced (“gap effect”²⁰); for the subset of movements with the shortest reaction times, the visual and
139 motor bursts merge^{20,21} (Figure 3E). As another critical test, we hypothesized that if temporal stability is
140 crucial to movement initiation, the visual burst during the shortest latency saccade trials should have a
141 stable temporal structure. This was indeed the case (Figure 3F), providing additional evidence in support
142 of the stability hypothesis.

143 Having established that the temporal structures of visual and premotor bursts differ, we sought to
144 identify a mechanism by which downstream neurons could discriminate between stable and unstable
145 population codes. The mere presence of a differential signal does not imply that the brain utilizes that
146 signal in a meaningful manner. In order to provide a framework within which the signal could be used to
147 control behavior, we constructed a rate-based accumulator model that receives input from
148 visuomovement neurons. The accumulator is an abstraction of a neuron receiving population inputs
149 through its network of dendrites (Figure 4A). For simplicity, we assumed that the output firing rate of
150 the neuron is a monotonically increasing function of the net post-synaptic current²². How might the
151 population pattern be converted into a signal that initiates a movement only when the pattern being
152 decoded is stable? Heuristically speaking, the decoder should keep track of the short-term history of
153 population activity, use this “memory” to evaluate stability, and respond selectively when the activity
154 pattern is deemed stable over some time scale. We incorporated these requirements in our model by
155 introducing short-term synaptic plasticity in the form of facilitatory connections^{23,24} from the input
156 population to the output unit (accumulator), with a Hebbian-like learning rule and leak current. Since
157 the network only contained excitatory connections, we also included weight normalization to prevent
158 unbounded accumulation in the output (Online Methods and Supplementary Info).

159 We then tested whether the leaky accumulator with facilitation (LAF) model could exploit temporal
160 structure by feeding it population activity from visuomovement neurons (only inputs from SC shown
161 here, Figure 4B1). We focused on two snippets of activity - the 180 ms period ending at the peak of the
162 visual response, and the 180 ms period terminating 35 ms before saccade onset (when the mean activity
163 in the premotor burst reached a level similar to the peak visual response). To discount effects of the
164 mean firing rate, we matched the population means at each time point by scaling the population vector
165 of one set of inputs by the running ratio of their respective means (Figure 4B2). Note that the temporal
166 structure is preserved in each signal (Figure 4B3). The accumulator increased its activity at a faster rate
167 in response to the stable pattern compared to the unstable pattern (Figure 4B4), suggesting that the
168 network was able to discriminate between two types of population patterns even though the net input
169 drive was identical.

170 How critical is facilitation to this function? Like the inputs themselves, the weights also showed greater
171 fluctuation during the visual burst (around $t=140$ ms in red traces, Figure 4B5). Consistent with our
172 heuristic, the weights tracked the input rates when the input was stable, but this correlation dropped
173 away when the input was unstable (Figure 4B6). Note that the shape of the correlation profile is not
174 unlike the stability profile in Figure 4B3. Therefore, facilitation allows the weights to retain the memory
175 of a pattern over the time scale of tens of milliseconds, but the memory can be scrambled by a
176 fluctuating input pattern. We quantified the network's ability to discriminate between the two patterns
177 with a pattern sensitivity index (Figure 4B7). The results here suggest that the accumulator is driven
178 more strongly by the stable premotor burst, even when other features of population activity remain the
179 same.

180 Neurons in premotor structures are constantly bombarded with information from thousands of
181 presynaptic neurons that are active during sensorimotor processing. It is unclear how activity relevant
182 for movement initiation is discriminated from activity related to other processing. A canonical model of
183 movement initiation, especially in the oculomotor system, is threshold-based gating⁴. Current
184 knowledge points to a role by the omnipause neurons (OPNs) in defining the threshold and controlling
185 saccade initiation^{4,25}. However, thresholds vary across behavioural paradigms¹², raising the question of
186 how the threshold is set in a particular condition. Furthermore, evidence that the threshold changes
187 during the course of a trial purely based on OPN activity is limited²⁵. A related explanation is that a
188 movement is initiated only when movement-related neurons are active, but most neurons in
189 sensorimotor structures likely span a continuum between having visuomovement activity to pure
190 movement activity¹. Other models posit that a movement is initiated when neural activity traverses
191 certain optimal regions of the population state space⁶ and is inhibited otherwise⁷. While the optimal
192 subspace and nullspace hypotheses are certainly appealing, a mechanistic understanding of why certain
193 patterns of activity translate to movement commands is lacking. We reason that for a decoder
194 downstream, it is important to ensure the stability - or consistency over time - of the input code before
195 processing it to influence output. Population activity that creates a high firing rate drive but is
196 inconsistent should be prevented from triggering a movement. This requirement is critical especially in
197 the case of ballistic movements such as saccades, where the ability to reverse the decision once the
198 movement has been initiated is limited.

199 Our findings are closely related to the premotor theory of attention¹¹ and offer a way to reconcile the
200 attention-or-intention debate. They could also account for the mirror-like activity recorded during both
201 action observation and execution in neurons known to project directly to motoneurons in the spinal

202 cord²⁶. In both cases, it is unclear how the same neuronal population represents two distinct signals that
203 serve different functional roles. Our results suggest that this multiplexing ability may be provided by the
204 distinct temporal structures of population activity patterns. Previous studies have looked at the role of
205 precise synchronization in the timing of incoming spikes in the transmission of information and the
206 efficacy of driving the recipient neuron^{27,28}. However, input firing rates may vary greatly across the
207 population, limiting the ability to compare to spike times. Our study proposes a mean-field equivalent to
208 the spike-based temporal or correlation code^{29,30} by looking at the temporal structure of population
209 firing rates, thus tying together the notion of rate, temporal, and population codes. We suggest that
210 temporal structure of population activity is a critical to understanding movement generation as well as,
211 more broadly, all neuronal communication and its relationship to behaviour.

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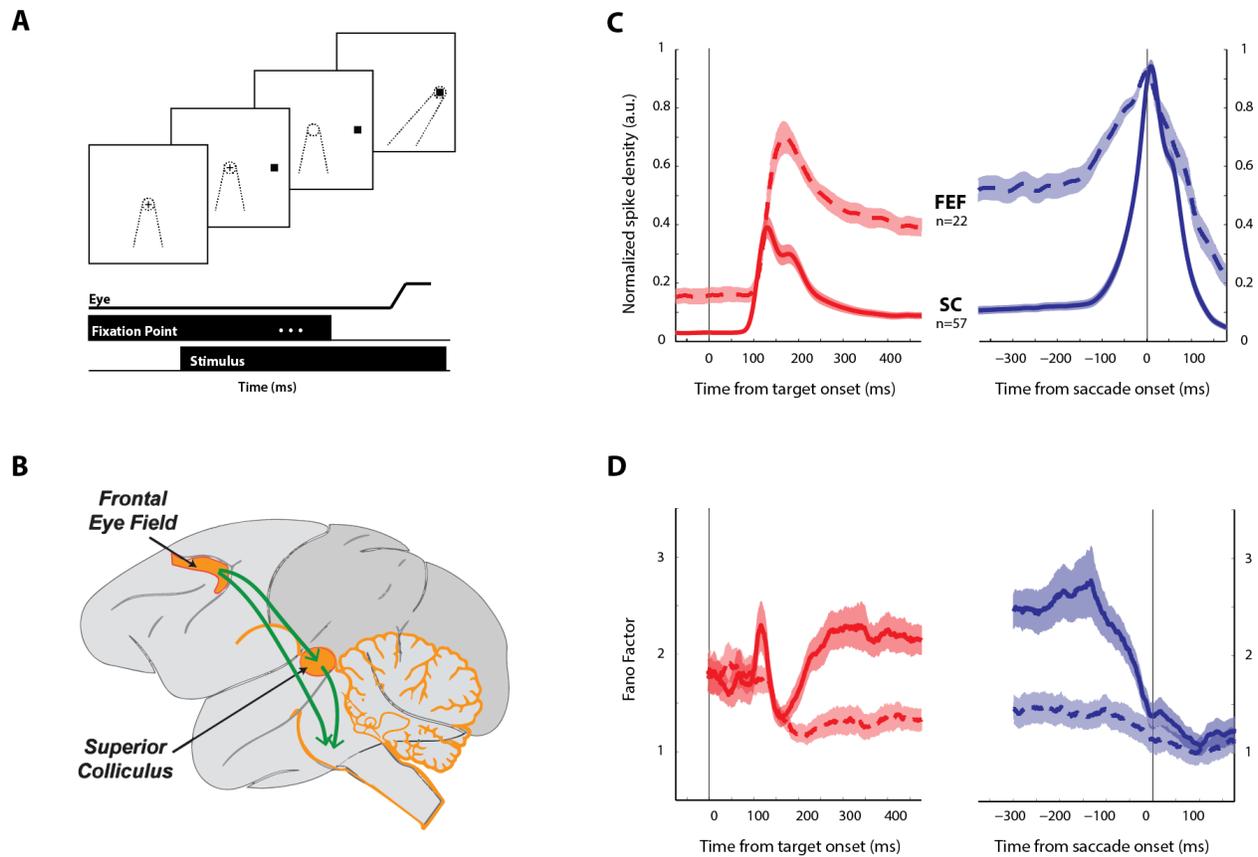
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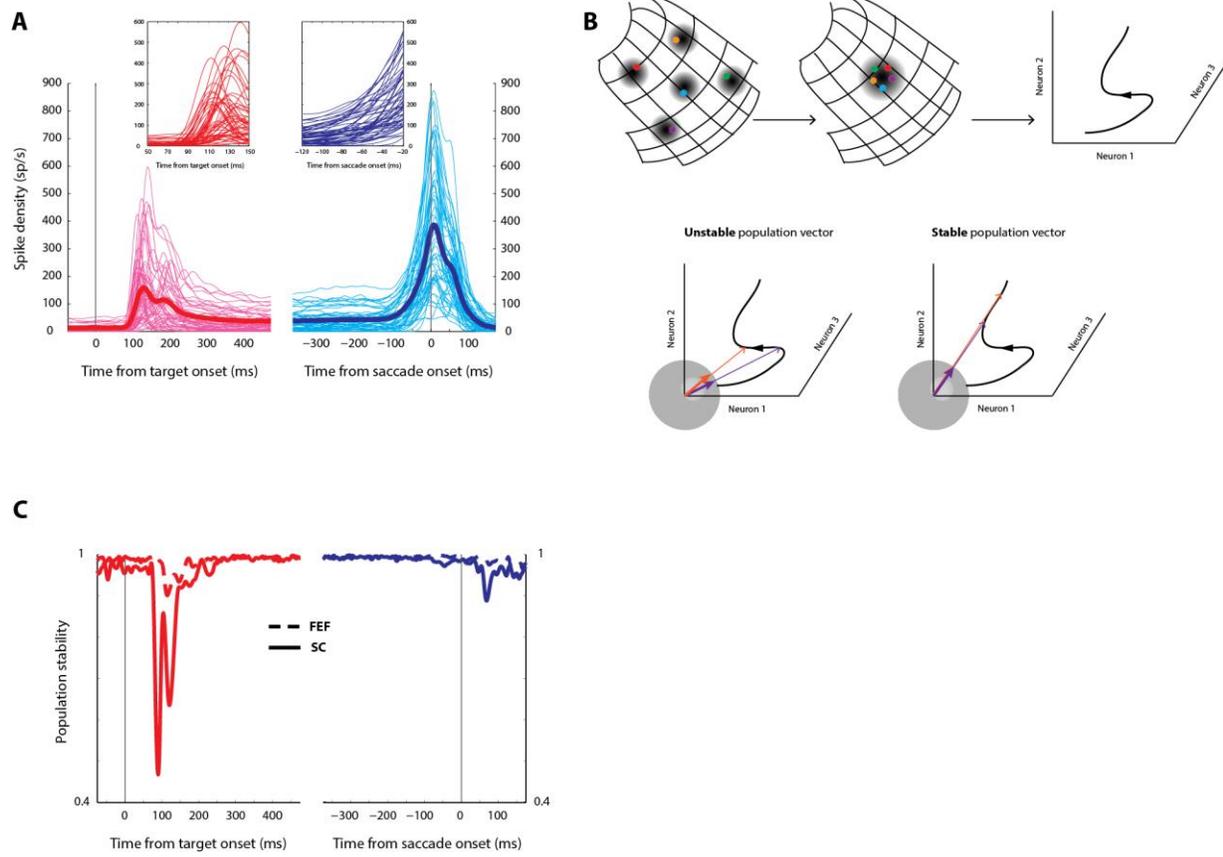
228 **Figure 1. Sensorimotor transformations are mediated by neurons that multiplex sensory and motor**
229 **information.** A. Sequence of events in the delayed saccade task. The top panel shows a typical display
230 sequence and the bottom panel shows the timeline. The fixation target is shown as a plus symbol for
231 illustration purposes. Dotted lines depict line of sight of the animal. B. Schematic of the monkey gaze
232 control network showing projections from the FEF and SC to the saccade burst generators in the pons. C.
233 Normalized mean population activity of visuomotor neurons recorded in the SC (solid) and FEF
234 (dashed) during the delayed saccade task. Since SC neurons have firing rates ranging from less than 100
235 spikes/s to 800 spikes/s, we normalized the spike density of each neuron by its peak magnitude to
236 enable meaningful averaging across the population, mainly for display purposes. The left panel shows
237 data aligned to the onset of the target. The right panel shows data aligned to saccade onset. This colour
238 scheme (red and blue for the two alignments respectively) will be followed throughout the article.
239 Shaded regions indicate s.e.m. D. Fano factor averaged across the population of SC and FEF neurons.
240 Line types/colour scheme same as in C.

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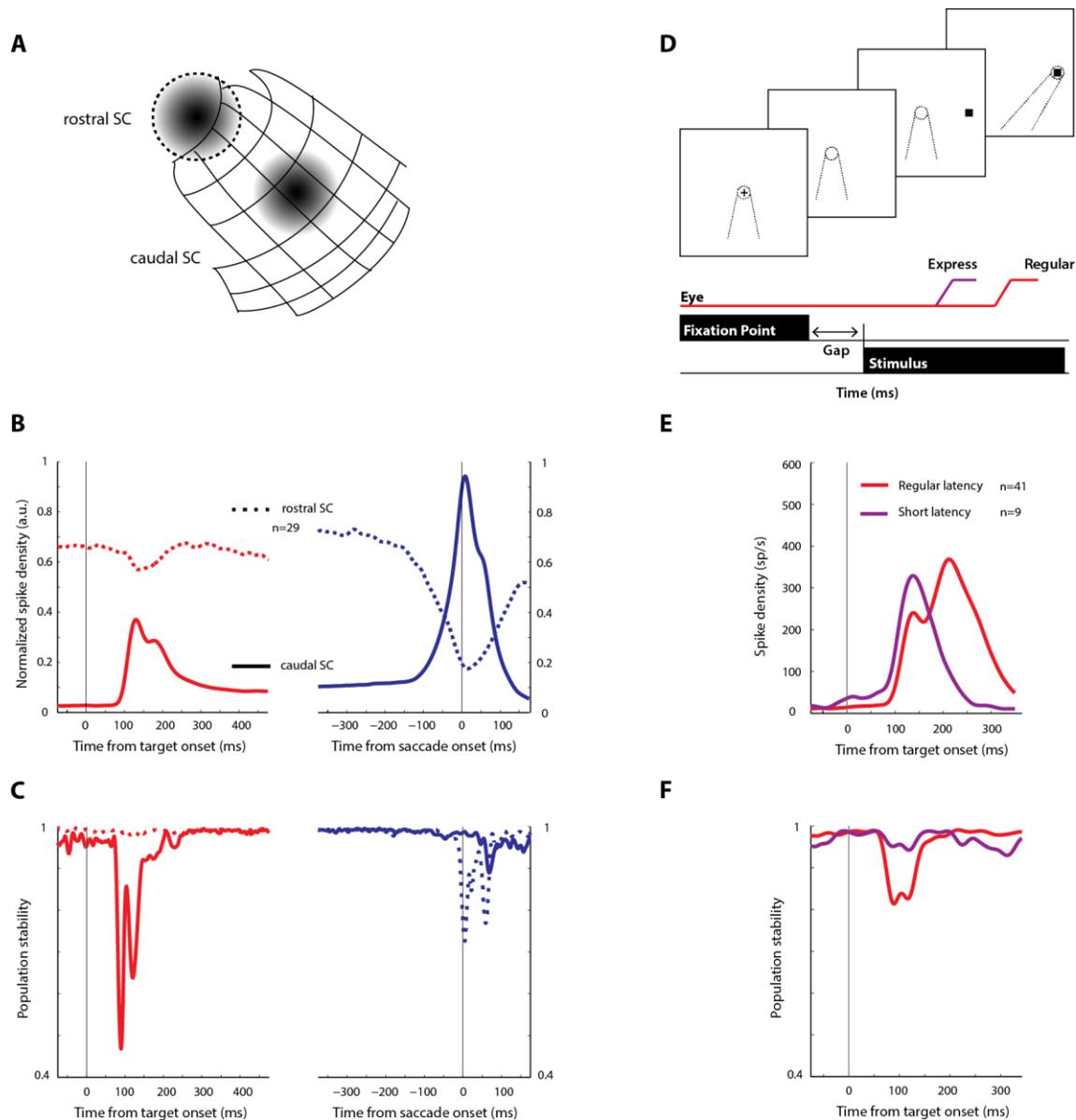
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247 **Figure 2. Population activity is temporally unstable in the visual burst and stable in the motor burst.** A.
 248 Activity profiles of individual SC neurons (thin lines) with the population mean (thick line) overlaid. The
 249 zoomed in insets highlight the temporal structure of the two bursts. B. Inferring population dynamics
 250 from single-unit recordings. The top row depicts the procedure of combining recordings made in
 251 different active populations on the SC map (left) into one pseudo-population (middle) that is active for
 252 any saccade, enabling the construction of an expected population trajectory (right) in neural state space.
 253 The bottom row shows examples of stable and unstable parts of the trajectory. Note that in both cases,
 254 the two population vectors (thin arrows in orange and purple) are separated by a similar length of time.
 255 In the unstable case, the normalized vectors (thick arrows) move around on the surface of the
 256 hypersphere, whereas in the stable case, they stay pointed roughly in the same direction. C. Temporal
 257 stability is computed as a dot product between two population vectors separated by $\tau=20$ ms for SC
 258 and FEF. Line types as in Figure 1. Population stability shows a drastic reduction at the time of the visual
 259 burst, but is stable before and during the onset of the movement.

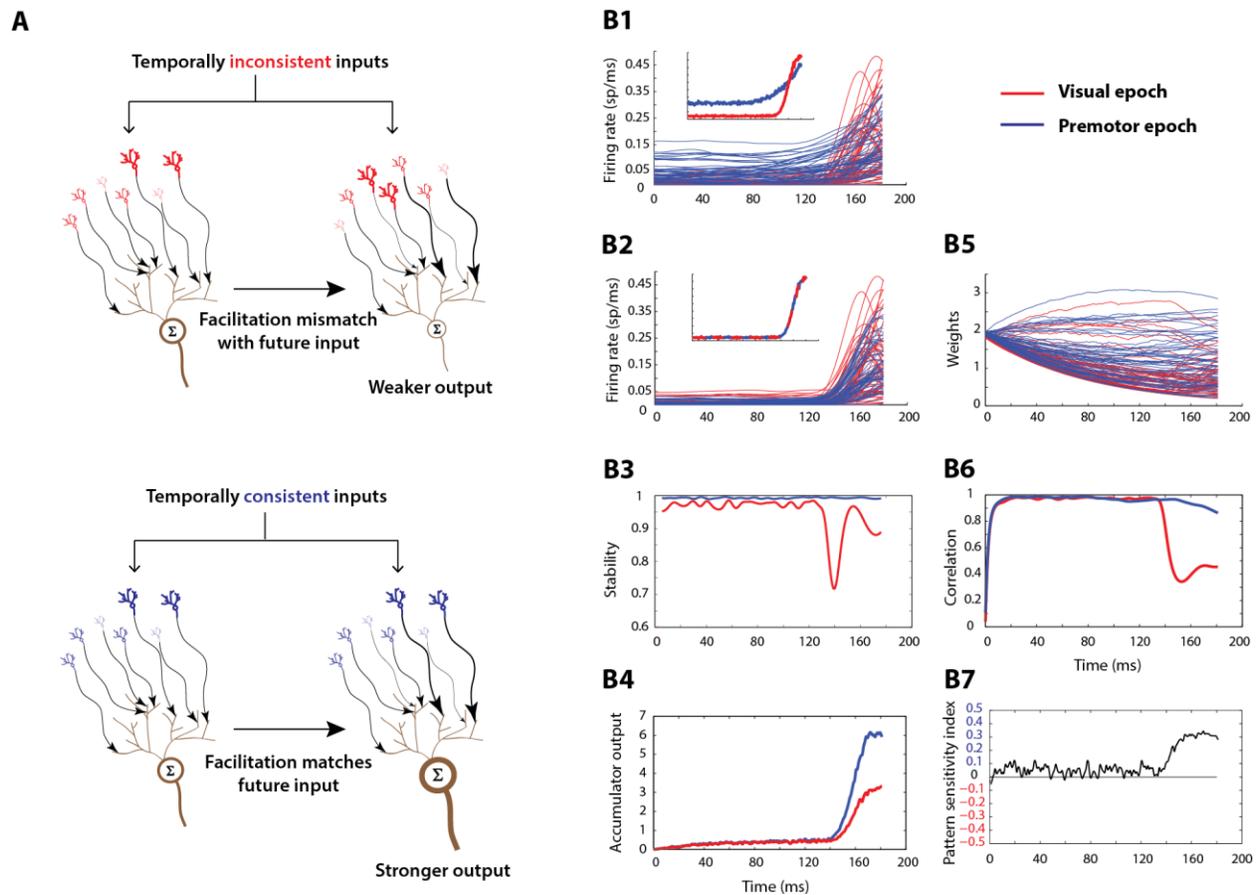


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262 **Figure 3. The stability hypothesis is validated for other neural populations and tasks.** A. Schematic of
 263 the SC depicting active populations in the rostral (dotted boundary) and caudal SC (plain). B and C.
 264 Normalized activity and temporal stability of the rostral SC population (dotted) with the caudal
 265 population shown for comparison. D. Timeline of the gap task with a schematic of regular (red) and
 266 express (purple) saccades. E. Mean population activity in caudal SC during the gap task for regular and
 267 express-like short latency saccades (colours as in D). Since this is a reactive task, saccade onset is
 268 separated from target onset only by 100-200 ms. Data are therefore only shown aligned on target onset
 269 for the sake of simplicity. Note the single peak in the burst for the short latency saccades. F. Temporal
 270 stability during regular and short latency saccades.

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274 **Figure 4. Leaky accumulator with facilitation model can discriminate population temporal structure. A.**

275 Schematic of the LAF model. The model incorporates a neuron that receives population inputs through
 276 its dendritic network and integrates them to produce an output. Each input neuron's activation level is
 277 represented by its size and thickness, and its connection strength to the accumulator is represented by
 278 the thickness of the arrow. The synaptic weights can grow over time due to short-term facilitation. Two
 279 time points are shown for illustration (left – arbitrary initial point, right – subsequent time point). Top –
 280 Unstable inputs lead to a scenario where the facilitated synapses that are no longer driven by the strong
 281 inputs that created them, whereas, bottom – stable inputs lead to matched strong input and synapses,
 282 resulting in stronger accumulation. B. Inputs to the model from SC visuomovement neurons. 1 - Raw
 283 unstable (red) and stable (blue) input profiles (inset – population means). The two sets of inputs are 180
 284 ms snippets taken from the visual and premotor bursts, respectively, in the spike density profiles shown
 285 in Figure 1. 2 - Mean-matched input profiles and population means. 3 - Temporal stability of the two
 286 mean-matched populations. 4 - Output of the LAF accumulator in response to the stable and unstable
 287 model inputs. 5 - Evolution of synaptic weights for the two conditions. 6 - Correlation between
 288 instantaneous weights and input rates for the two conditions. 7 – Pattern sensitivity index of the
 289 model's ability to discriminate between the two types of inputs. Values in the top half of the plot
 290 indicate higher sensitivity (faster accumulation) to stable population input.

291

292 **Methods**

293

294 *General and surgical procedures*

295 All experimental and surgical procedures were approved by the Institutional Animal Care and Use
296 Committee at the University of Pittsburgh and were in compliance with the US Public Health Service
297 policy on the humane care and use of laboratory animals. We used three adult rhesus monkeys (*Macaca*
298 *mulatta*, 2 male, ages 8 and 6, and 1 female, age 10) for our experiments. Both SC and FEF were
299 recorded in monkey BB whereas only one of either SC or FEF was recorded in monkeys WM and BL,
300 respectively. Under isoflourane anaesthesia, recording chambers were secured to the skull over
301 craniotomies that allowed access to the SC and FEF. In addition, posts for head restraint and scleral
302 search coils to track gaze shifts were implanted. Post-recovery, the animal was trained to perform
303 standard eye movement tasks for a liquid reward.

304

305 *Visual stimuli and behaviour*

306 Visual stimuli were displayed either by back-projection onto a hemispherical dome (monkeys BB and
307 WM), or on a LED-backlit flat screen television (monkey BL). Stimuli were white squares on a dark grey
308 background, 4x4 pixels in size and subtended approximately 0.5° of visual angle. Eye position was
309 recorded using the scleral search coil technique, sampled at 1 kHz. Stimulus presentation and the
310 animal's behaviour were under real-time control with a Labview-based controller interface³¹. All
311 monkeys were trained to perform standard oculomotor tasks. In the delayed saccade task, the monkey
312 was required to initiate the trial by acquiring fixation on a central fixation target. Next, a target
313 appeared in the periphery but the fixation point remained illuminated for a variable 500-1200 ms, and
314 the animal was required to delay saccade onset until the fixation point was extinguished (GO cue). In the
315 gap task, initial fixation on a central target was followed by a gap period (200 ms) during which the
316 fixation point disappeared, while the animal was required to maintain fixation at the now empty
317 location. This was followed by the appearance of a saccade target in the periphery which was also the
318 GO cue for the animal to make a saccade. All animals performed these tasks with >95% accuracy.
319 Incorrectly performed trials were removed from further analyses. The delayed and gap saccade tasks
320 were occasionally interleaved with visual search paradigms used for a different study. The gap task,
321 when performed in isolation with predictable spatial and temporal features, is often characterized by a
322 bimodal distribution of saccade reaction times (SRTs) with a proportion of saccades occurring at
323 significantly short latencies³². Since we interleaved several behavioral tasks, the animal was prevented
324 from constantly being in "gap task mode", and the bimodality of the SRT distribution was not as
325 noticeable. Hence, we divided the gap task trials into two sets based on SRT and labelled the set with
326 shorter SRTs "short latency" saccades (100-150 ms) and the other set as "regular" saccades (170-250
327 ms). The temporal features of the visual and motor bursts in SC neurons during the short latency trials
328 resembled the profiles observed during express saccades²¹.

329

330

331 *Electrophysiology*

332 During each recording session, a tungsten microelectrode was lowered into the FEF or SC chamber using
333 a hydraulic microdrive. Neural activity was amplified and band-pass filtered between 200 Hz and 5 kHz
334 and fed to a digital oscilloscope for visualization and spike discrimination. A window discriminator was
335 used to threshold and trigger spikes online, and the corresponding spike times were recorded. Both SC
336 and FEF were confirmed by the presence of visual and movement-related activity as well as the ability to
337 evoke fixed vector saccadic eye movements at low stimulation currents (20-40 μ A, 400 Hz, 100 msec).
338 Before beginning data collection for a given neuron, its response field was roughly estimated. In most
339 cases, during collection of data, the saccade target was placed either in the neuron's response field or at
340 the diametrically opposite location in a randomly interleaved manner. In addition, the simulation-
341 evoked saccades were recorded to identify the response field centers (or "hotspots") for the cells
342 recorded during that session. For recordings in rostral SC, stimuli were presented at one of four
343 locations at an eccentricity sufficient to induce a reduction in activity during the large amplitude
344 saccade.

345

346 *Data analysis and pre-processing*

347 Data were analyzed using a combination of in-house software and Matlab. Eye position signals were
348 smoothed with a phase-neutral filter and differentiated to obtain velocity traces. Saccades were
349 detected using standard velocity criteria. The animal was considered to be maintaining fixation if the
350 gaze remained within a 2-3° window around the fixation target. We also detected any microsaccades
351 that occurred during the delay period in each trial by using a velocity criterion based on the noise in the
352 velocity signal for that trial. Only one of the two monkeys (WM) in whom we recorded neural activity in
353 the rostral SC made sufficient number of microsaccades to permit further analysis.

354 Raw spike density waveforms were computed for each neuron and each trial by convolving the spike
355 trains with a Gaussian kernel (width = 4ms; in some instances, we used 10 ms for display purposes only).
356 The spike densities were averaged across condition-matched trials (same target location) following
357 alignment with target or saccade onset. We refer to this averaged spike density function for the i^{th}
358 neuron as $R_i(t)$ below. Since SC neurons have firing rates ranging from less than 100 spikes/s to 800
359 spikes/s (Figure 2A), we also normalized the average spike density of each neuron by its peak magnitude
360 to enable meaningful averaging across the population, mainly for display purposes (e.g., Figure 1C).
361 Neurons were classified as visuomovement neurons if the spike density was significantly elevated above
362 baseline during the visual epoch (50-200 ms following target onset) and during the premotor epoch (50
363 ms before and after saccade onset). This classification yielded our data set of 57 neurons in caudal SC
364 and 22 neurons in FEF. In addition, rostral SC neurons were defined as fixation-related if the activity
365 during the premotor epoch of large saccades was significantly reduced below baseline (29 neurons). A
366 subset of these neurons also elevated their discharge around the onset of microsaccades (7 neurons). To
367 minimize the effect of noise in the spike density waveforms due to insufficient number of trials in our
368 analysis, we used only neurons which had at least 10 trials for a given condition. This was not a factor in
369 most conditions (we typically had 50-100 trials) except in the case of the gap task. Applying this
370 minimum number of trials criterion resulted in 9 neurons for the short latency condition and 41 neurons
371 for the regular latency condition.

372

373 We also computed the Fano factor - the spike count variance across trials divided by the mean spike
374 count - for each neuron in the delayed saccade task. Spikes were counted in 100 ms windows sliding in
375 increments of 1 ms. The Fano factor trace was then shifted forward in time by half the window size (50
376 ms). The rationale behind this correction is that the beginning of any change in the spike count statistics
377 at a given time t is going to be captured in a window centered at $t-50$ ms. The Fano factor was then
378 averaged across neurons to obtain a population trace (Figure 1D).

379

380 *Inferring population dynamics from single-unit recordings*

381 Our analyses and results build on the ability to infer features of population dynamics from single-unit
382 activity profiles recorded sequentially over multiple sessions. In order to do this, we constructed a
383 simulacrum of the population activity in SC and FEF by combining all of our single-unit recordings. We
384 define $\mathbf{R}(t)$ as a population activity vector:

$$385 \quad \mathbf{R}(t) = \begin{bmatrix} R_1(t) \\ R_2(t) \\ \dots \\ R_n(t) \end{bmatrix}$$

386 where $\mathbf{R}(t)$ represents the instantaneous activity at time t as a point in an n -dimensional space, where n
387 is the number of neurons in the population. The curve connecting successive points over time is the
388 neural trajectory that describes the evolution of population activity. Note that each neuron's firing rate
389 waveform $R_i(t)$ is the average across many matched trials (identical stimulus/response conditions).
390 Thus the neural trajectory is the *expected trajectory* of population activity. Since these neurons have a
391 fairly broad RF, many neurons constitute the active population for any given visual stimulus or
392 saccade³³. Our neurons were sampled roughly (but not exactly) around the hotspot of the active
393 population for a given session. Therefore, the pooled data from individual sessions can be thought of as
394 an approximation of the population mound active for an arbitrary location/RF in the visual field on any
395 given trial. Many recent studies have reconstructed such "pseudo-populations" from sequentially
396 recorded neurons³⁴⁻³⁶ and found comparable properties from simultaneous and serial recordings⁷.
397 Indeed, this is also expected of our pseudo-population under the assumption of isotropy – that each
398 neuron's contribution to its respective local active population is similar regardless of the locus of the
399 population. To better demonstrate this, we estimated the location of a given neuron in the active
400 pseudo-population as follows (we use SC for illustrative purposes because of its convenient topography).
401 We used the point image of the target location on the SC map as a representation as the center of the
402 active population for that session, and used the stimulation-evoked saccade vector to identify the
403 location of that neuron on the SC. We referenced the point image of each target location to a single
404 location to create an active pseudo-population and translated the neuron locations relative to this
405 population center (EDF 1). All mathematical equations for transforming between visual and SC tissue
406 coordinates have been defined previously³⁷. Inclusion of stimuli/saccades in the anti-preferred RF of the
407 neurons allowed us to also estimate a pseudo-population of neurons in the ipsilateral SC. To complete
408 the representation of activity across the SC topography, we also recorded from and included in our
409 analyses neurons in the rostral portion of SC, which are active during fixation, burst during

410 microsaccades, and are suppressed during large saccades. Moreover, to account for correlations
411 between neurons on individual trials (noise correlation), we show that the temporal stability profile
412 derived from the across-trial mean population dynamics (see next section) is a theoretical upper bound
413 on the stability expected on single trials (Supplementary Material and EDF 1). The findings in this letter
414 are therefore independent of our methodological assumptions and are robust in the face of correlations
415 within the population. It is also important to emphasize that these results are also independent of any
416 across-trial jitter in the phase of the display system's refresh cycle since we use the trial-averaged neural
417 activity for all our analyses.

418

419 *Temporal stability analysis*

420 To assess temporal stability, we first normalized the population trajectory $\mathbf{R}(t)$ by its Euclidean norm
421 ($\|\mathbf{R}(t)\|$), equivalently its magnitude, at each time point to yield $\hat{\mathbf{R}}(t)$:

$$422 \quad \hat{\mathbf{R}}(t) = \frac{\mathbf{R}(t)}{\|\mathbf{R}(t)\|}$$

423 The normalized trajectory can be visualized as a unity length population vector that points in an n-
424 dimensional direction at each instant in time. That is, while $\mathbf{R}(t)$ is free to traverse the n-dimensional
425 activity space, $\hat{\mathbf{R}}(t)$ is constrained to the surface of an n-dimensional hypersphere (Figure 2B). Temporal
426 stability or consistency of the evolving population was then quantified by the dot product of two time-
427 shifted unity length vectors:

428

$$429 \quad S(t) = \hat{\mathbf{R}}(t - \tau) \cdot \hat{\mathbf{R}}(t + \tau)$$

430 The stability metric ($S(t)$) tracks the running similarity of the normalized trajectory separated in time by
431 2τ msec. Crucially, the normalization constrains $S(t)$ between 0 and 1. Thus, if $S(t) \rightarrow 1$, the
432 population activity is considered stable since the relative contribution of each neuron is consistent. If
433 $S(t) \ll 1$, the population activity is deemed unstable because the relative contribution is variable. If the
434 neural trajectory is not normalized, the dot product quantifies similarity across the vectors' magnitude
435 and direction. It roughly mimics the quadratic of the firing rate (exactly so for $\tau=0$). When the vector
436 direction remains constant, the dot product yields no additional information than that already present in
437 the firing rate. In contrast, the normalization scales the neural trajectory so it always has unity
438 magnitude. It neither alters the relative contributions of the neurons nor compromises the vector
439 direction. The dot product therefore performs an unbiased evaluation of stability based only on vector
440 direction, and is an estimate of the fidelity of the population code modulo a multiplicative gain factor.
441 Intuitively, the stability measure is analogous to the correlation between the neurons' activities at two
442 different time points. We chose the dot product, however, because of its interpretability as a measure
443 of pattern similarity in n-dimensional activity space.

444 Since temporal stability is defined over the whole population and gives rise to a singular measure at a
445 given time, it is not directly amenable to statistical significance analysis. Therefore, we took an
446 alternative approach in order to ensure that the observed stability profiles were contingent on the
447 recorded activity. We shuffled the population activity in two different ways and bootstrapped the
448 stability across multiple shuffles for each type of shuffle (EDF 3). First, we shuffled the activity by
449 randomly assigning each vector in the set of population vectors across time to different time points. This

450 temporal shuffle retains each neuron's identity in the population but scrambles the structure across
451 time. Next, we randomly shuffled the neuron's activities at each time point. This shuffle retains the
452 average firing rate at each instant but removes any temporal correlation in the firing rate across
453 neurons. We performed multiple shuffles of each kind and computed temporal stability for each
454 instance to obtain bounds for the stability profile in the original data.

455

456 *Leaky accumulator with facilitation (LAF) model*

457 We developed a computational model to demonstrate how the temporal structure of population activity
458 could be used by neural circuits to gate decisions such as movement initiation. The core idea behind the
459 model is the hypothesis that signal integration is stronger when the temporal structure in input activity
460 is stable, which allows the decoder neuron to reach threshold during the high frequency burst that
461 triggers the movement. We constructed an accumulator as an abstraction of a neuron receiving
462 population inputs through its network of dendrites. The total synaptic current ($I(t)$) and the firing rate
463 ($v(t)$) of the accumulator were defined as

$$464 \quad \tau_s \dot{I} = -I + \sum_1^n w_i u_i$$

465 and

$$466 \quad v = F(I)$$

467 where u_i and w_i are the instantaneous firing rate and synaptic gain of the i^{th} input neuron, and τ_s is the
468 time constant of the synaptic current. The output firing rate of the single decoder neuron ($v(t)$) can be
469 described by a standard monotonic function (e.g., linear or sigmoid) applied to the net current. The
470 family of stochastic, leaky accumulator models that integrate the firing rate of neurons toward a
471 threshold criterion has been commonly used to explain reaction time, decision making, and perception
472 (*refs*). We used the following heuristic in order to extend this framework to incorporate temporal
473 structure. In order to assess stability, the decoder neuron must keep track of the short term history of
474 the population activity, use this "memory" to evaluate stability, and respond selectively when the
475 activity pattern is deemed stable over some time scale. We implemented these requirements by
476 introducing short-term plasticity in the form of facilitatory connections from the input population onto
477 the output unit (accumulator). The change in connection strength or gain of each neuron on the
478 decoder neuron (w_i) can be defined by a differential equation that incorporates a Hebbian-like learning
479 rule and leak current:

$$480 \quad \tau_w \dot{w}_i = -w_i + \frac{u_i v}{w n f_i}$$

481 The Hebbian-learning component, $(u_i v)$ describes the change in weight coupled to the firing rates of the
482 i^{th} pre-synaptic neuron and the post-synaptic accumulator neuron. τ_w is the time constant of the weight
483 parameter. Since this version of the model contains only excitatory connections, the weight parameter
484 can exhibit unbounded accumulation, which can be controlled by incorporating normalization. We
485 normalized the Hebbian-learning component $(u_i v)$ with the contribution of that neuron to the total
486 resource pool. The resource pool available for facilitation at any given time was defined as the sum of
487 the Hebbian-learning component across all input units ($\sum_1^n u_i v$). The contribution of a neuron to the

488 output rate v then determines its contribution to the overall pool, giving rise to the weight
489 normalization factor:

$$490 \quad wnf_i = \sum_1^n u_i w_i u_i$$

491 For the model simulation, we used visuomovement neuron population activity in SC as inputs. We
492 created two sets of input snippets from the visual and premotor bursts, each 180 ms in length. For the
493 visual burst, we used the activity upto the peak in the population response. For the premotor burst, we
494 used activity upto 35 ms before saccade onset, when the response magnitude reached the same level as
495 the peak of the visual burst. In order to control for the effect of average firing rate on the accumulation,
496 we mean-matched the input profiles as follows. We divided the activations in the premotor input set at
497 each instant by the mean activation of the visual inputs at the corresponding instant. That is,

$$498 \quad u_i^{\text{pre}}(t)_{\text{mm}} = \frac{u_i^{\text{pre}}(t)}{\frac{1}{n} \sum_1^n u_i^{\text{vis}}(t)}$$

499 where $u_i^{\text{vis}}(t)$ and $u_i^{\text{pre}}(t)$ are the activity of neuron i at time t in the visual and premotor input sets,
500 respectively, and $u_i^{\text{pre}}(t)_{\text{mm}}$ is the premotor input instantaneously rescaled to match the mean of the
501 visual input. This ensured that we isolated the effect of temporal structure, independent of firing rate,
502 on the model's response. We also quantified the ability of the synaptic weights to track the inputs by
503 computing the Pearson's correlation between the weight and input vectors at each time point. To
504 quantify the accumulator's ability to discriminate temporal pattern in population input, we computed a
505 pattern sensitivity index (psi) as

$$506 \quad \text{psi}(t) = \frac{v^{\text{pre}}(t) - v^{\text{vis}}(t)}{v^{\text{pre}}(t) + v^{\text{vis}}(t)}$$

507 where $v(t)$ is the output of the accumulation for the corresponding inputs.

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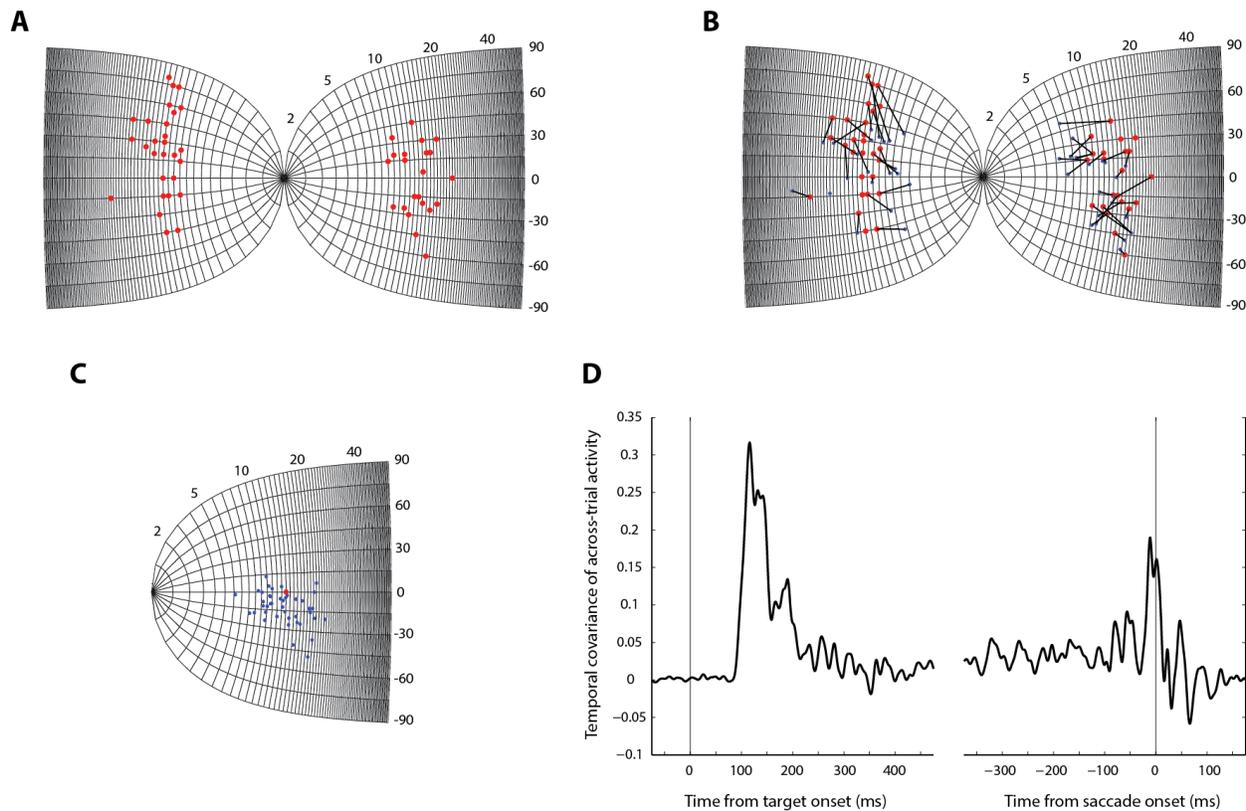
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518 **Extended Data Figures**



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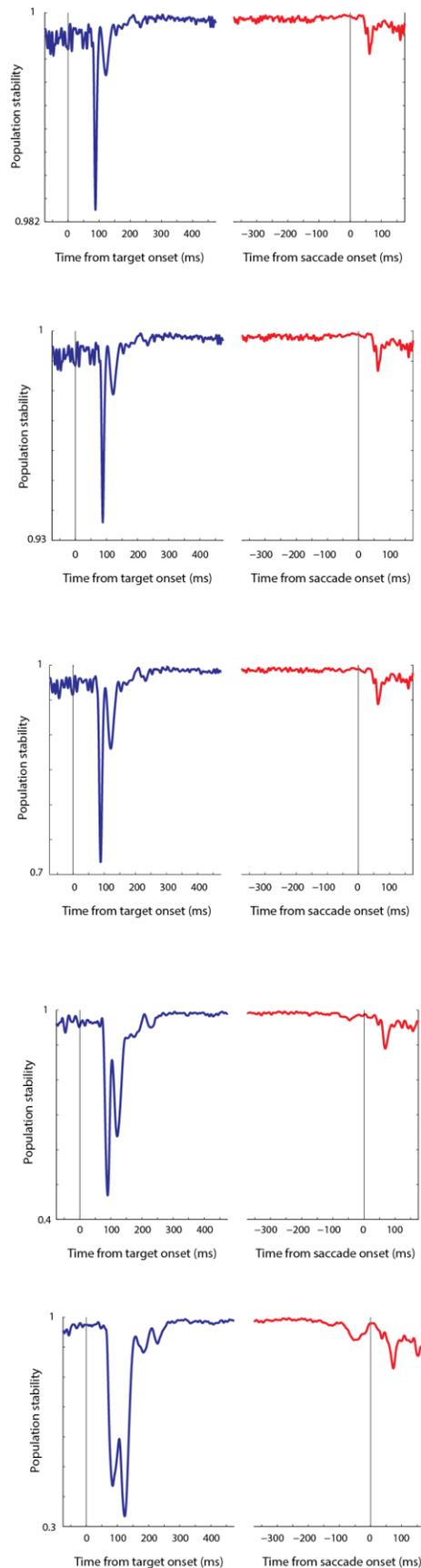
522 **Extended Data Figure 1. Inferring population dynamics from single-unit recordings.** A. Point images
523 (red) of target locations on the SC map across all experimental sessions. The locations on the SC were
524 computed using known transformations between visual space and SC tissue coordinates (Online
525 Methods). B. Same as in A, with the endpoint of the stimulation-evoked saccade vector at the recording
526 site shown in blue. The stimulation vector provides a proxy for the RF center of the recorded neuron.
527 Neuron-target pairs (blue-red) from individual sessions are connected using black lines. C. The active
528 pseudo-population on the SC map. The red locations from A and B were referenced to an arbitrary
529 location on the SC map (here, $R=15$, $\theta = 0$) and the blue locations appropriately translated. Points
530 from both colliculi have been shown on a single SC for the sake of clarity. D. Covariance of activity over
531 time and across trials averaged across all visuomovement neurons in SC aligned on target (left) and
532 saccade (right) onsets. For a derivation of how using the average activity for each neuron sets limits on
533 the expected stability on single trials, see Supplementary Material.

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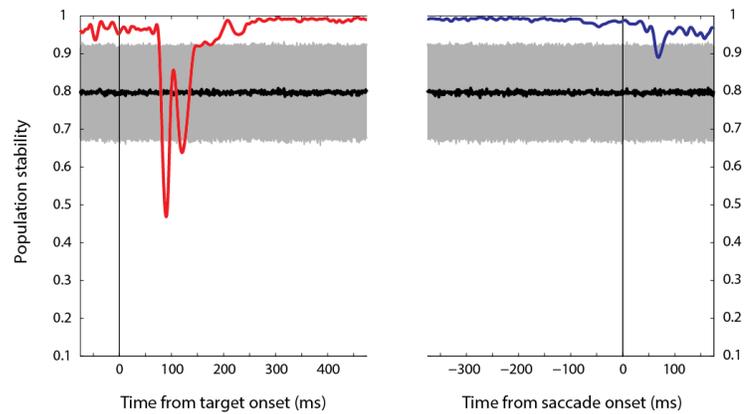
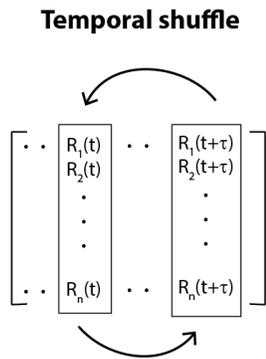
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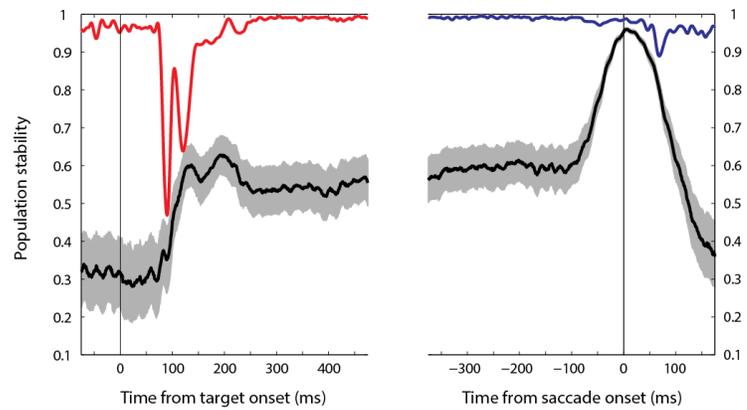
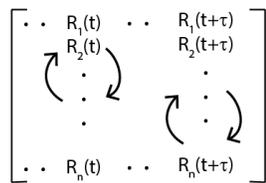
Extended Data Figure 2. Temporal stability as a function of τ . In order to ensure that the choice of τ did not have an undue effect on our results, we computed stability for several values of τ . The panels show temporal stability profiles for SC as in Figure 2C, for (from top to bottom), $\tau = 1, 2.5, 5, 10,$ and 20 , respectively. As seen, the absolute magnitude of stability was inversely related to τ - a consequence of the smooth and continuous nature of the trajectory. In other words, since the state of the neural population evolves smoothly, it must traverse intermediate states in order to move from one state to another, resulting in greater similarity between state vectors close together in time than those further apart. However, the relative shape of stability profile was largely preserved across τ (except for some deviation for values of τ as high as 40 ms, possibly a consequence of comparing population vectors from different epochs). Thus, the relative instability during the visual epoch and stability during the premotor epoch persists across all time separations.

A



B

Instantaneous neuron shuffle



539

540

541 **Extended Data Figure 3. Temporal stability profile is specific to the recorded population structure.** A.
 542 Left - The population code was altered by randomly shuffling the population vectors between different
 543 time points, i.e., the activity vectors are taken from the population trajectory in the original data but in a
 544 different temporal sequence. Right - The black trace is the mean temporal stability across 1000 shuffles,
 545 and the gray region is the standard deviation across these samples. The stability profile flattens at what
 546 is effectively the average value of the dot product between any two vectors in the trajectory. This
 547 bootstrap approach indicates that the observed stability profile (red and blue traces) is specific to the
 548 true population dynamics. B. Left - The population code was scrambled by randomly shuffling the
 549 neuron's activities at each time point. This shuffle preserves the average population activity at each
 550 instant while removing any ordering between individual neurons. Right - The observed stability profile
 551 (red and blue traces) is well outside the distribution of profiles obtained across 1000 shuffles (mean –
 552 black trace, standard deviation – gray region).

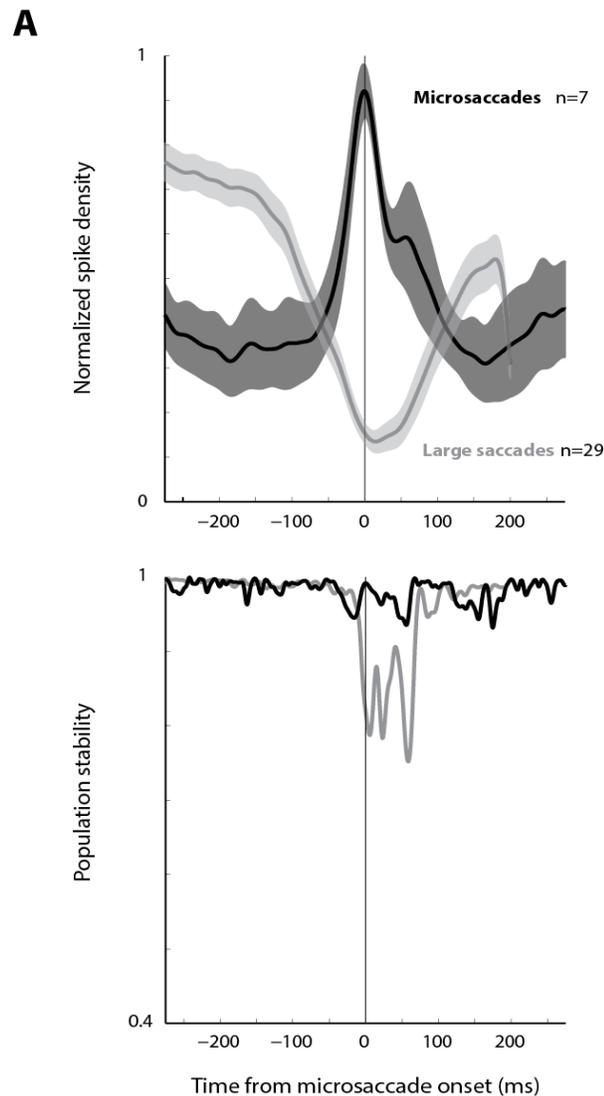
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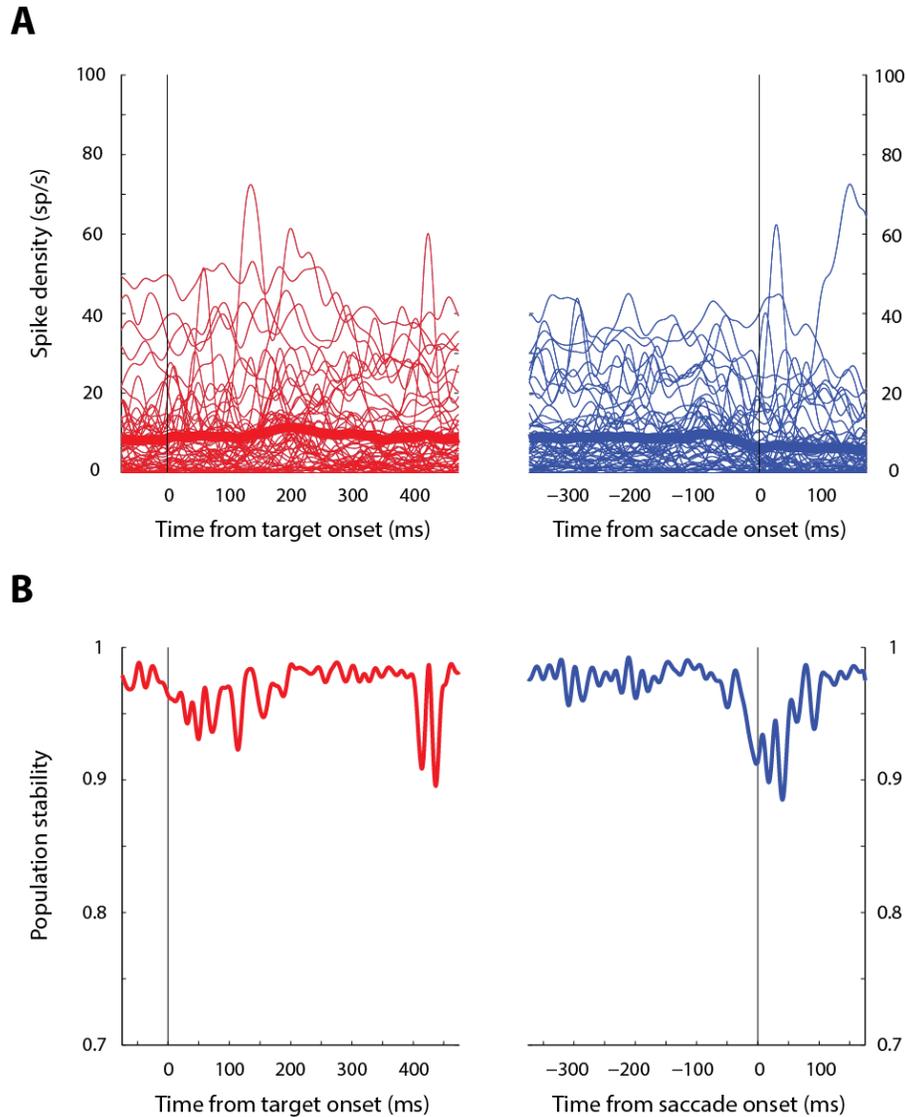
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560 **Extended Data Figure 4. Population activity of rostral SC neurons is stable during microsaccades.** Top –
561 Normalized population activity of microsaccade-related neurons in rostral SC (black). Note the strong
562 burst compared to the suppression in fixation-related neurons in the same region for large saccades
563 (gray). Bottom – Temporal stability of the rostral SC population for microsaccades shown in comparison
564 to large saccades. Neurons which burst for microsaccades remain stable during these fixational eye
565 movements, consistent with the hypothesis that both an increase in firing rate and high stability is
566 required for movement generation.



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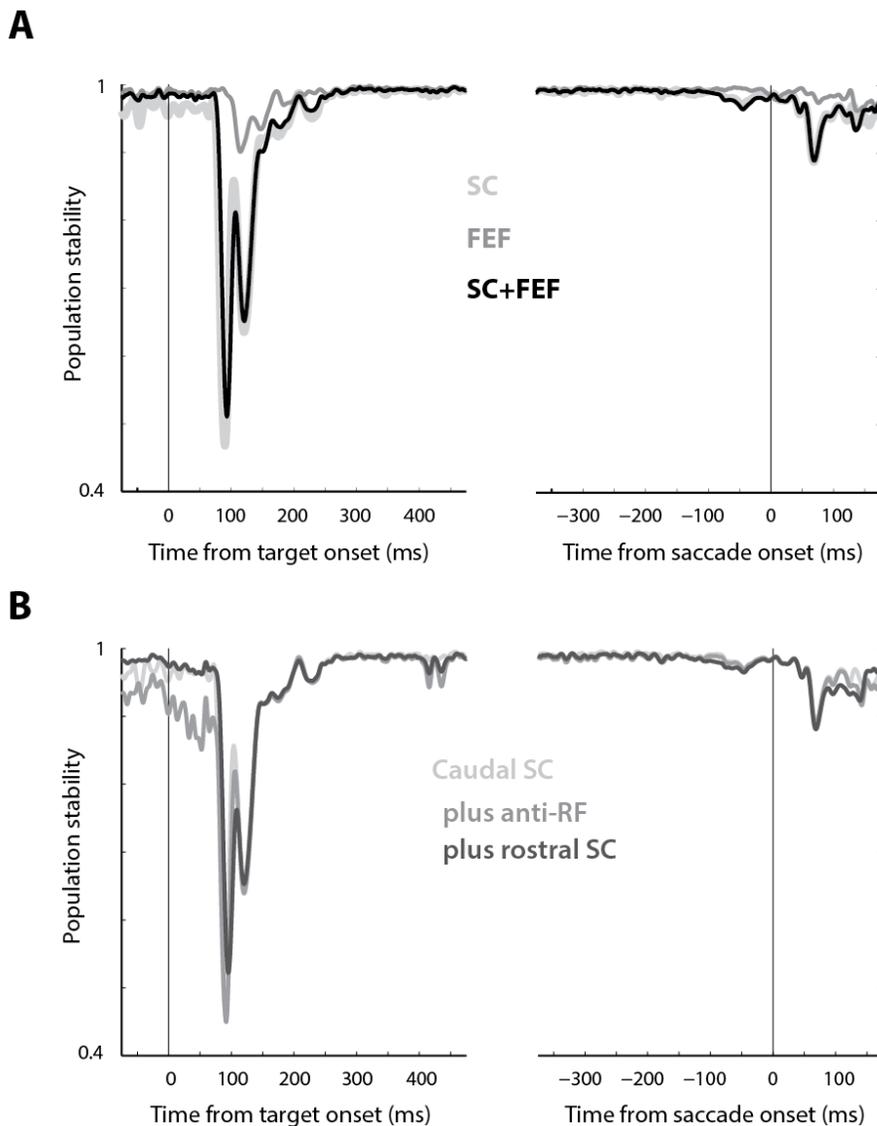
569 **Extended Data Figure 5. Characteristics of the population in ipsilateral SC.** A. Spike densities of
570 individual neurons (thin traces) and the population average (thick trace) when the target (left panel) and
571 saccade (right panel) were in the ipsilateral hemifield. These neurons show little to no modulation in
572 their firing rate when neurons in the contralateral SC actively produce bursts. B. Temporal stability of
573 the ipsilateral population aligned on target (left) and saccade (right) onset. Like the rostral SC (Figure 3),
574 the ipsilateral population is relatively more unstable during the saccade, suggesting that stability may be
575 an actively controlled variable designed to reduce the effective drive from competing neuronal
576 populations during movement generation.

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582 **Extended Data Figure 6. Temporal stability of combined populations.** A. Temporal stability aligned on
583 target (left) and saccade (right) onset for neurons constituting the individual “active populations” in
584 caudal SC and FEF (same as Figure 2) in the two shades of gray, and the combined active population in
585 black. Since the burst generators receive input from both sets of neurons, it is important to verify that
586 the characteristics of population temporal structure is preserved for the whole population. B. Temporal
587 stability for the active caudal SC population (light gray, same as above and Figure 2), combined caudal SC
588 population (including the ipsilateral SC from EDF4, medium gray), and all neurons in SC (dark gray). Since
589 the ipsilateral and rostral SC populations contribute little to the firing rate vector, their contribution to
590 the dot product is also low – therefore, any difference between these stability profiles is small.

591

592

593 **Supplementary text**

594 **S1. Inferring population dynamics from single-unit recordings**

595 We previously described in the main text and methods how we reconstructed a pseudo-population from
596 unit recordings. Using knowledge of the RF centre of a given session's local population obtained from
597 microstimulation, and known transformation from visual space to SC tissue coordinates, here we
598 reconstruct the pseudo-population on the SC map.

599 We first transferred target locations (R_T, θ_T) inside the RF for each neuron onto the SC map using the
600 following transformations³⁷ –

601
$$x_T = \frac{B_x}{2} \ln \frac{(H+A)^2 + V^2}{A^2}, y_T = B_y \tan^{-1} \frac{V}{H+A}, \text{ where,}$$

602
$$H = R_T \cos \theta_T, = R_T \sin \theta_T, \text{ and } A = 3, B_x = 1.4, B_y = 1.8.$$

603 The transformed locations (x_T, y_T) on the bilateral SC map are shown in EDF1, panel A. In order to move
604 these target locations to one pseudo-population, we need to identify where these points reside in the
605 RF of each neuron or local population. Previous studies have demonstrated a strong correspondence
606 between the saccade vector evoked by microstimulation and the centre of the RF of neurons at the site
607 of microstimulation. We used the endpoints of the site-specific microstimulation-evoked saccades as a
608 proxy for the respective RF centres. The transformed endpoints, with their locations relative to the
609 corresponding target locations, are shown in EDF1, panel B. We picked an arbitrary location in the visual
610 field as the RF centre of the pseudo-population, $(R_c, \theta_c) = (15, 0)$. We used the fact that the size of the
611 active SC population is invariant regardless of the encoded saccade to preserve relative distances in
612 tissue space between a site's target location and RF centre, and translated the transformed
613 microstimulation endpoints to the common pseudo-population centre, along with the respective target
614 locations. To construct one active population from sites gleaned from both hemifields (and therefore
615 both colliculi), we reflected the coordinates from one colliculus onto the other. The resulting pseudo-
616 population (EDF1, panel C) shows a fairly representative sampling of neurons from the pseudo-
617 population, with its extent consistent with a large (25% of SC tissue) active population, albeit one that is
618 biased to one side of the population.

619 A fundamental assumption behind treating the expected population trajectory as a proxy for an
620 arbitrary single-trial trajectory is that relevant features of single-trial population dynamics are captured
621 by the expected population trajectory. However, it is possible that correlated dynamics between
622 neurons, such as noise correlations on individual trials, may cause measures based on single-trial
623 trajectories to deviate significantly from the dynamics of the average. We show here that our measure
624 of the structure of the trial-specific population code – temporal stability – is indeed preserved in the
625 expected trajectory.

626 Let us denote by $\hat{R}(t)$ the population unit vectors comprising the expected trajectory. Let us also denote
627 the population unit vectors making up individual trial trajectories by $\widehat{kR}(t)$, where k is a trial index. The
628 expectation of the inner product stability measure across trials is given by,

629
$$E \left[\widehat{kR} \left(t - \frac{\tau}{2} \right) \cdot \widehat{kR} \left(t + \frac{\tau}{2} \right) \right] =$$

$$\begin{aligned}
 630 \quad & E\left[\sum_i kR_i\left(t - \frac{\tau}{2}\right) kR_i\left(t + \frac{\tau}{2}\right)\right] = \\
 631 \quad & \sum_i E\left[kR_i\left(t - \frac{\tau}{2}\right) kR_i\left(t + \frac{\tau}{2}\right)\right] = \\
 632 \quad & \sum_i E\left[kR_i\left(t - \frac{\tau}{2}\right)\right] E\left[kR_i\left(t + \frac{\tau}{2}\right)\right] - Cov\left[kR_i\left(t - \frac{\tau}{2}\right), kR_i\left(t + \frac{\tau}{2}\right)\right] < \\
 633 \quad & \sum_i E\left[kR_i\left(t - \frac{\tau}{2}\right)\right] E\left[kR_i\left(t + \frac{\tau}{2}\right)\right] = \\
 634 \quad & \sum_i R_i\left(t - \frac{\tau}{2}\right) R_i\left(t + \frac{\tau}{2}\right) = \\
 635 \quad & \hat{R}\left(t - \frac{\tau}{2}\right) \cdot \hat{R}\left(t + \frac{\tau}{2}\right)
 \end{aligned}$$

637 Therefore, $E\left[\widehat{kR}\left(t - \frac{\tau}{2}\right) \cdot \widehat{kR}\left(t + \frac{\tau}{2}\right)\right] < \hat{R}\left(t - \frac{\tau}{2}\right) \cdot \hat{R}\left(t + \frac{\tau}{2}\right)$, i.e., the stability profile obtained using
 638 individual trial trajectories and then averaging across trials will be lower than that obtained from the
 639 expected single-trial trajectory (as we do here). The inequality holds only if the second term in the sum,
 640 the covariance between normalized time-separated activity across trials, is positive. Intuitively, this
 641 seems likely because of the fact that the activity of these neurons evolves smoothly. We verified this by
 642 computing the evolution of temporal covariance across trials for each neuron. Since we do not have
 643 access to the population unit vectors for individual trials, we simply used the unnormalized activity,
 644 reasoning that covariance should be unaffected by normalization scaling. The average covariance profile
 645 is shown in EDF1, panel D. Note that the covariance is largely positive, with minor deviations late during
 646 the trial. Importantly, note that the covariance is higher during the visual epoch compared to the
 647 premotor epoch. This will produce a greater reduction in stability (step 4 in derivation) during the visual
 648 epoch in the true single-trial trajectories, only strengthening our result.

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