

Research Articles | Systems/Circuits

Pooled versus structured inhibition drives stimulus competition across distinct spatial scales in the superior colliculus

<https://doi.org/10.1523/JNEUROSCI.2296-25.2026>

Received: 17 December 2025

Revised: 29 September 2026

Accepted: 1 October 2026

Copyright © 2026 the authors

This Early Release article has been peer reviewed and accepted, but has not been through the composition and copyediting processes. The final version may differ slightly in style or formatting and will contain links to any extended data.

Alerts: Sign up at www.jneurosci.org/alerts to receive customized email alerts when the fully formatted version of this article is published.

Title: Pooled versus structured inhibition drives stimulus competition across distinct spatial scales in the superior colliculus

Abbreviated title: Rules of competition in the superior colliculus

Author Names and Affiliations: Arunima Banerjee^{1,#}, Ninad B. Kothari^{1,#}, Shreesh P. Mysore^{1,2,3,*}

¹Department of Psychological and Brain Sciences, Johns Hopkins University, Baltimore, MD, 21218

²The Solomon H. Snyder Department of Neuroscience, Johns Hopkins University School of Medicine, Baltimore, MD, 21205

³Kavli Neuroscience Discovery Institute, Johns Hopkins University, Baltimore, MD, 21218

[#]Equal contribution

***Corresponding author email address:** mysore@jhu.edu

Number of pages: 45

Number of figures: 5

Number of Words:

Abstract: 214

Significance statement: 119

Introduction: 616

Discussion: 1490

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

Acknowledgements: This work was supported by NIH R34NS111653 (SPM), NSF 2047298 (CAREER award, SPM), NIH F32EY032776 (NBK), and start-up funds from the Johns Hopkins University (SPM). We thank Gokul T. Anandan for assistance with setting up the eye-tracking

camera, Jacob Bedke for help in establishing the preprocessing pipeline for spiking data, and James Garmon for engineering support.

Abstract

Competitive stimulus interactions across the space map of the midbrain superior colliculus (SC) are critical for spatial decision-making among multiple options. Here, using electrophysiological recordings from the intermediate and deep layers of the SC (SCid) in mice of either sex, we discovered that the rules and spatial profiles of these competitive interactions are different between local within-receptive field (RF), versus global across-RF-boundary, spatial scales. When one visual stimulus was centered in the RF and a second one was located within the RF's classical inhibitory surround, we found that stimulus interactions followed an averaging rule. This classical surround was spatially restricted, and the strength of inhibition underlying these within-RF interactions decreased with distance from the RF center. By contrast, when the second stimulus was located outside the RF, stimulus interactions followed a divisive rule. Strikingly, this extra-classical inhibitory surround was spatially global, and the strength of competitive inhibition underlying these across-RF-boundary interactions was distance-invariant while being dependent on the relative strengths of the competing stimuli. Computational modeling revealed that whereas within-RF interactions are well-explained by a normalization-like mechanism driven by pooled inhibition, across-RF-boundary interactions are not, and instead, are well-explained by a winner-take-all-like mechanism driven by structured, donut-like inhibition. Combined, our results reveal the mechanistic logic of stimulus competition across the space-map of mammalian SCid.

Significance statement: Selecting one target among many is essential for diverse cognitive functions, and requires neural circuits to compare priorities of competing stimuli. Using

electrophysiological recordings in the mouse superior colliculus, a critical brain area for spatial decision-making, we show that stimulus competition follows fundamentally different rules at local versus distant spatial scales. Nearby stimuli within a receptive field (RF) engage spatially restricted, decaying inhibition, enacting an averaging rule. By contrast, distant stimuli separated by the RF boundary engage spatially global, distance-invariant inhibition, enacting a divisive, relative strength-dependent rule. Computational modeling reveals that local interactions are explained by a pooling normalization mechanism, whereas global interactions are explained by a donut-like winner-take-all mechanism. Results resolve long open questions about stimulus competition in the SC.

INTRODUCTION

Animals are exposed to a barrage of diverse inputs from multiple locations in the sensory field. Selecting the location of the most salient (or behaviorally relevant) stimulus from this array is essential for behaviors involving spatial attention, perceptual decision-making, and orienting movements. Experimental studies as well as computational models have suggested that such selection emerges from competitive interactions among the representations of stimuli in the sensory field (Desimone & Duncan 1995, Knudsen 2007, Koch & Ullman 1987). These interactions create a distributed representation of stimuli in the form of a spatial map of relative stimulus salience (or more generally, of relative priority; priority = salience + relevance) that guides the locus of selection (Fecteau & Munoz 2006, Itti & Koch 2001, Mysore & Kothari 2020).

Topographically organized maps of stimulus salience (and more generally, of priority) are known to exist in several interconnected brain regions: the frontal eye field (FEF), the lateral intraparietal area (LIP), and the superior colliculus (SC) (Goldberg et al 2006, McPeck & Keller 2004, Thompson & Bichot 2005). Among these, the superior colliculus (SC), a layered midbrain

structure that is evolutionarily conserved across vertebrates, plays a central role in mediating spatial target selection (Basso & May 2017, Krauzlis et al 2013). Its intermediate and deep layers (SCid) integrate multisensory inputs into topographic representations of sensory space, and drive orienting behaviors, including saccades in primates and head movements in rodents (Allen et al 2021). Importantly, SCid activity reflects the relative priority of the stimulus, and focal silencing of SCid disrupts spatial target selection among distractors (Lovejoy & Krauzlis 2010, McPeck & Keller 2004, Wang et al 2020).

A prominent signature of competitive selection in the SC occurs in the form of response suppression. Studies across species have shown that when multiple visual stimuli are presented simultaneously, SC responses to a given stimulus are often reduced compared to when it is presented alone (cats: (Rizzolatti et al 1973); primates: (Basso & Wurtz 1998); barn owls: (Mysore et al 2010); mice: (Wang et al 2020)). This suppression occurs for spatially distant stimuli (Mysore et al 2010, Rizzolatti et al 1974), implicating long-range competitive interactions, as well as for stimuli within a single receptive field (Alvarado et al 2007, Li & Basso 2005), suggesting local interactions. Mechanistically, lateral inhibition—the suppression of activity in one spatial channel arising from neighboring channels—is thought to play a key role in mediating these competitive interactions (Munoz & Istvan 1998, Mysore & Knudsen 2012). However, the spatial and computational principles underlying these inhibitory interactions remain poorly understood in the mammalian SC.

In this study, with extracellular neural recordings in the mouse SCid, we investigate the rules of visual stimulus competition across the SCid space map. We find that the computational rules governing paired stimulus responses, as well as the underlying spatial structure, are markedly different across two regimes: inside versus outside the classical inhibitory surround. When two stimuli fall within the RF of a neuron, the joint responses follow an averaging rule, consistent with the classical center-surround RF structure. The underlying inhibition drops off in

strength with distance from the RF center. By contrast, when the competing stimulus lies outside the RF, the joint responses are suppressed with respect to the response to the RF stimulus alone, following a divisive rule. This suppression is global in scope - spanning nearly the entire azimuthal and elevational space, and its strength is space-invariant - showing no dependence on the relative distance between the stimuli. Computational modeling to account for the observed results from stimulus competition experiments reveals the necessity of two separate inhibitory circuit mechanisms operating over distinct spatial scales in order to mediate stimulus competition across the SCid space map.

MATERIALS AND METHODS

Animals

All mice used for electrophysiological studies were adult (8+ weeks old; 4 male and 4 female) wild-type mice (C57BL6/J strain, Jackson Laboratory). Upon arrival, all mice were housed in a colony where temperature (~74°F) and humidity (~50%) were controlled on a 12:12 hr light:dark cycle, and all procedures were performed after allowing mice to acclimate for at least 7 days in the new environment. All procedures followed NIH guidelines and were approved by the Johns Hopkins University Animal Care and Use Committee.

Stereotactic surgery

Surgical procedures were followed as described previously (Kothari et al 2026). Briefly, animals were anesthetized with isoflurane (3% induction, 0.5–2% maintenance) and were secured using standard earbars in a stereotaxic frame (Kopf Instruments). A feedback-controlled heating pad (FHC) was used to maintain the body temperature at 36.8°C, and eye lubricant was applied to

the eyes to prevent them from drying. Meloxicam (0.1 mg/kg) was administered as an analgesic. After the head of the animal was leveled in the stereotaxic frame, fur was gently trimmed away on top of the head, exposed skin was cleaned using betadine (antiseptic) with sterile wipes, and bupivacaine (0.75 mg/kg) was injected subcutaneously for perioperative analgesia. A scalp incision was made along the midline, followed by skin retraction, skull cleaning and preparation. Once bregma and lambda were exposed, the skull was zeroed by adjusting the earbars and bitebar of the stereotax. A craniotomy was made above the SC (centered at AP -3.5 mm; ML +/- 0.8 mm from bregma). All coordinates are based on a standard mouse brain atlas (Franklin & Paxinos 2019). Craniotomies were covered with KwikSil (World Precision Instruments) for the protection of the underlying tissue. A custom-made titanium headbolt was stereotactically affixed to the skull using Metabond (Parkell), and the bregma location was kept exposed. Subsequently, mice received meloxicam (0.1 mg/kg) daily for up to 3 days.

Neurophysiology

Neurophysiological protocols followed those that have been described previously (Kothari et al 2026). Briefly, acute single-shank silicon probes (Neuronexus 0.3 – 1.5M Ω at 1 kHz probes: A1x32-poly3-10mm-25s-177-A32, A1x32-poly3-10mm-50s-177-A32, A1x32-poly2-5mm-50s-177-A32) were used to record single and multi-units extracellularly in head-fixed, awake mice. The apparatus allowed targeting coordinates of the brain stereotactically. Neural activity that was synchronized with visual stimulus presentation times was collected via a 32-channel headstage (Intan Technologies), sampled at 30 kHz, and stored using a data acquisition system (OpenEphys).

All recordings were made in the intermediate to deep layers of the SC (SCid). Electrodes were lowered into the brain using measurements from the Bregma as well as from the pial surface.

Physiological signatures were used to further guide the recordings: superficial layers (SCs) exhibited characteristic bursting activity, which was absent in deeper SCid layers. All recordings were made in the SCid by targeting depths more than 1.5 mm below the pial surface, and more than 400 μm below the surface of the SC. In a subset of mice, probes were covered with Dil, and the mice were perfused after the experiment to histologically verify the probe track and recording depth (Fig. S4A).

Preprocessing and spike sorting

Multi-unit spike waveforms were sorted offline into putative single units using Waveclus (Quiroga et al 2004) and analyzed using custom MATLAB code. Briefly, a custom script was first used to detect and remove motion artifacts that were correlated across channels, using correlated responses evoked from potentially distinct units across channels that spiked concurrently to the stimulus. Following this, spikes were detected for individual channels at time points where the membrane potential crossed a threshold of 6-7 times the median absolute deviation. Channels exhibiting multi-unit visually evoked activity greater than 7 Hz were retained for further analysis. To minimize the likelihood of including duplicate units recorded on neighboring channels, only units from channels separated by $>50 \mu\text{m}$ were included for spike sorting. Spike waveforms were extracted from a 1.5-ms window centered on the detected peak and sorted in Waveclus. The fraction of visually responsive units varied across experiments (18.75%–100%), with greater variability observed among recordings from deeper SC layers. Visually evoked activity was quantified as the baseline-subtracted firing rate during stimulus presentation.

Experimental design

The critical variables for this study were the stimulus-evoked responses of superior collicular neurons to the presentation of single and two simultaneously presented visual stimuli. For each question (figure), experiments were performed in 3-6 mice.

Visual stimuli: Visual stimuli were created using customized MATLAB software (Kothari et al 2026, Mahajan & Mysore 2022, Mysore et al 2010, Schryver et al 2020). Mice were head-fixed in the apparatus with the monitor 12.5 cm from the center of the eyes, in either of two positions: (#1) *Head-on/Symmetric position*: In this configuration, the tangent monitor was positioned directly ahead of the mouse so that the monitor was exposed symmetrically to both eyes; this yielded a viewing angle of $\pm 65^\circ$ azimuth and $\pm 35^\circ$ elevation (Fig. S1A); (#2) *Rotated/Asymmetric position*: In this configuration, the tangent monitor was within $\pm 45^\circ$ around a vertical axis centered between the eyes so that the monitor was more visible to one of the eyes; this provided a maximum viewing angle of $-20/+110^\circ$ azimuth and $\pm 35^\circ$ elevation (Fig. S1A). In all cases, positive azimuthal angles denote contralateral locations. The visual grid was corrected for distortions resulting from presenting the stimuli on a flat tangent screen. In the schematics of all experimental protocols, the tangent monitor is shown in head-on position for simplicity, even though data were collected from both positions. We confirmed that the rotation of the mouse relative to the screen did not affect the response properties of SCid neurons (Fig. S1B).

Expanding dots. Expanding visual stimuli consisted of a white or black dot on a gray background that increased linearly in size over the period of stimulus presentation (250 ms), starting from a size of 3° diameter. The center of an expanding stimulus remained constant throughout the duration of stimulus presentation, and expansion speed was defined as the (linear) rate of change of the angular size of the dot, $d\theta/dt$, where θ is the visual half-angle subtended by the object at the eye. Different expansion speeds were achieved by changing the final size of the dot, while

keeping the initial size and the duration of presentation fixed (as above). Linearly expanding dots, used extensively in previous work (Kothari et al 2026, Mysore et al 2010, Mysore et al 2011, Schryver & Mysore 2019, Schryver et al 2020), evoked stronger responses than stationary dots of the same final size (Fig. S1C), indicating that a major driver of their responses is their dynamics (speed of expansion) than simply the final size. They also evoked stronger responses for faster expansion speeds (Fig. S1C), allowing for expansion speed to serve as a metric of stimulus salience (Kothari et al 2026, Mysore et al 2010, Mysore et al 2011), and produced responses that were more resistant to trial-to-trial adaptation than stationary dots when presented repeatedly at the same spatial location (Kothari et al 2026, Mysore et al 2010). In our experiments, expansion speeds (final dot sizes) were chosen such that the responses remained within the linear range of the response curve (Fig. S1C; bottom, black curve): final diameters of the RF stimulus were 6°-10°, while those of the competitor stimulus were 12°-20°.

Spatial tuning curves. Spatial tuning to visual stimuli was measured by presenting expanding dots of 5° final radius, duration 250-500 ms, ISI 2.5-3.5 s, white or black, on a gray background. Tuning to visual azimuth (or elevation) was first estimated by presenting 10–15 repetitions of stimuli at randomly interleaved azimuthal (elevation) locations, with the elevation (azimuth) held constant at the manually estimated best value for the site. These measurements were iteratively performed if the azimuthal or elevation responses were different from the manually estimated centers.

Bar stimuli. We used bar stimuli to estimate the extent of the excitatory and inhibitory components of an SCid neuron's classical receptive fields (Schryver & Mysore 2023). Briefly, bar stimuli were of fixed thickness (3°), but varying lengths such that the center of the bar was displayed at the receptive field center of the measured neuron in the SCid.

Stimulus adaptation. Responses of neurons in the SCid habituated (weakened) to the repetition of any stimulus (light, dark, moving, or stationary dots) (Kothari et al 2026, Lee et al 2020). To

minimize these effects, we used long interstimulus intervals (up to 3.5 s) and used expanding dot stimuli instead of stationary dot stimuli (Kothari et al 2026, Mysore et al 2010).

Two-stimulus protocols. One of the main goals of this study was to study the neural responses of SCid neurons when two stimuli were present. To achieve this, visual stimuli were created following previously established protocols (Mysore et al 2010). Expanding dots were used for both stimuli. To estimate the display locations, first, the center of the receptive field (RF) was measured as described above, and a stimulus S1 was placed at the center. The competing stimulus, S2, was chosen to be stronger than S1 (see above) as weaker stimuli often failed to produce reliable suppression of S1 responses when S2 was presented outside the RF (Fig. S5E; see also Kothari et al 2026). When both S1 and S2 were presented inside the RF, choosing S2 to have a higher physical strength (e.g., higher expansion rate) enabled us to sample conditions in which the effective strength of S2 (quantified by the firing rate evoked by S2 alone) was either weaker or stronger than that of S1, depending on S2's spatial position within the RF. Additionally, choosing S2 to have a higher physical strength than S1 had the advantage of the S2 stimulus being identical to the one used in the outside-RF interaction experiments.

For two-stimulus protocols with a stimulus outside the classical receptive field, this second stimulus (S2) was typically placed $>30\text{-}40^\circ$ from the center (median = 38.4°). This was to ensure that S2 was sufficiently outside the region influenced by the classical surround. Post-hoc analysis ensured that S2 locations that were overlapping with the estimated RF boundary were eliminated from the analysis. The median distance of S2 from the RF center was well beyond half the median RF size in our population (half of the median value of full-width at half-max, FWHM= 24.3° ; Fig. S3A). When both stimuli were presented inside the RF, it was ensured that their spatial locations were non-overlapping.

Eye tracking: To track eye positions during stimulus presentations, a camera (Arducam UC-844) was placed approximately 11 cm from the contralateral eye. Each eye was illuminated using an array of four infrared LEDs (940 nm). Video streams were collected with an average frame rate of 100 Hz and stored within a Raspberry Pi 4 module, along with TTL pulses synchronized to the onset of stimulus presentations. To extract pupil positions, a neural network was trained with four landmarks for pupil position and one for the center of the corneal reflection, using DeepLabCut (Fig. S2B, (Mathis et al 2018)). Custom MATLAB scripts were used to fit a circle to the estimated pupil coordinates and estimate the pupil centers at any instant. Pupil angular positions with respect to the eye center were calculated from the two-dimensional image using previously published methods that use the corneal reflection and a constant eyeball radius of 1.25 mm for C57BL6/J mice (Sakatani & Isa 2004). The small fraction of pupil displacements was restricted to 3.27° along the horizontal plane and 2.88° along the vertical plane (99-percentile values) (Fig. S2C,D). These results demonstrated that eye movements were small compared to the spatial extents of visual stimuli and were unlikely to modulate neural responses, particularly during two-stimulus protocols.

Statistical analysis

In all experiments, the net responses of an SCid neuron to a stimulus were computed by subtracting the mean firing rate during the pre-stimulus baseline period (averaged across all interleaved trials) from the mean firing rate within a post-stimulus analysis window ([70 ms, 250 ms] w.r.to stimulus onset).

Visuospatial receptive field (RF). The visual RF for each site was defined as the region of the visual field where a single visual stimulus elicited a significant increase in firing rate above baseline. A location was considered to be within the RF if the baseline-subtracted response at

that location was significantly greater than zero ($p < 0.05$, one-way ANOVA, followed by multiple comparison correction). The RF center was defined as the peak of a one-dimensional Gaussian fit to the tuning curve along azimuth or elevation. RF size was quantified as the full width at half maximum (FWHM) of the Gaussian fit (Fig. S3A). The goodness-of-fit (R^2) of the Gaussian model was used to assess the extent to which RF tuning deviated from a Gaussian profile, including potential asymmetries (Fig. S3B); the RFs were all well-fit by Gaussians and hence, symmetric.

Two-stimulus interactions with S2 outside the RF. To quantify competitive suppression from a second, spatially discrete stimulus (S2) *outside* the RF, we compared responses to the simultaneous presentation of S1 (centered in the RF) and S2, with responses to S1 alone (Fig. 4). A robust linear regression (*robustfit* in MATLAB) was applied to the paired response data. The intercept was considered significantly different from zero if the associated p-value was less than 0.05. The slope was considered significantly different from unity if its 95% confidence interval did not include 1.

To evaluate the effect of different locations of S2, we computed the percent change in response to S1 in the presence of S2 compared to the response to S1 alone. To identify locations where S2 had a significant modulatory effect ($p < 0.05$), a one-way ANOVA followed by multiple comparison correction was applied to the distribution of percent-change values. To visualize the spatial profile of suppression, we binned S2 locations into 10° - 30° bins. For each bin, the mean percent suppression was calculated and plotted $\pm$ SEM. Bin centers were computed as the mid-points of the S2 locations within each bin and shown with corresponding SEM error bars.

Difference of Gaussians (DoG) model. To characterize classical surround suppression, we analyzed responses to bar stimuli of increasing length, which followed a classic non-monotonic profile (Fig. 5). We fit a standard difference-of-Gaussians (DoG) model, widely used in retinal and cortical physiology (Mysore et al 2010, Sceniak et al 1999), which assumes spatially co-centered

excitatory and inhibitory Gaussian components that sum linearly to produce a net response $R(s)$ described as:

$$R(s) = R_0 + K_e \int_{-s}^s e^{-\left(\frac{2y}{a}\right)^2} dy - K_i \int_{-s}^s e^{-\left(\frac{2y}{b}\right)^2} dy$$

where R_0 denotes the baseline response, K_e and K_i denotes the strengths, while a and b denote the space constants of the excitatory and inhibitory Gaussians, respectively. The spatial extent of the classical inhibitory surround was estimated as the bar length at which the responses reached 5% of the steady-state value. The suppression index was calculated as the difference between the peak response and the steady-state response, normalized (divided) by the peak response.

Time course analysis. To measure any differences in the time course of responses, we computed the instantaneous firing rates from the peristimulus time histograms (binned at 10 ms) using a Gaussian filter ($\sigma = 12$ ms). For two-stimulus experiments within the RF, we compared the instantaneous firing rates of S1 and S2 presented together with either S1 or S2 presented alone, by performing an ANOVA across each time bin. The time-to-divergence (TTD) was estimated as the first time bin where the p-value of ANOVA dropped below 0.05 and remained below 0.05 for at least 30 ms. Similarly, when the second stimulus was outside the RF, we computed the time-to-suppression (TTS) as the time point when the instantaneous firing rate in response to S1 and S2 presented together dropped significantly below the response to S1 presented alone.

Statistics. All statistical analyses were carried out on MATLAB. Statistical significance was accepted as $p < 0.05$. Parametric tests (ANOVA, t-test) were used when the data were normally distributed (tested using Kolmogorov-Smirnov test), and non-parametric tests (sign test, Wilcoxon rank sum test) were performed otherwise. When testing across multiple groups, Holm-Bonferroni corrections for multiple comparisons were performed. Data is summarized as mean $\pm$ SEM unless otherwise specified.

Computational modeling of competitive stimulus interactions in SCid using spiking neurons

We used biophysically plausible neurons to simulate excitatory and inhibitory neurons for modeling. For each neuron, the membrane voltage $V(t)$ was modeled as a first-order leaky integrator with a passive membrane time constant (Burkitt 2006, Gerstner et al 2014, Mysore & Quartz 2005).

$$\tau_m \left(\frac{dV}{dt} \right) = -(V(t) - V_{rest}) + R_m * I(t) \quad \text{Eq 1}$$

where τ_m is the membrane time constant, V_{rest} is the resting potential, and R_m is the membrane resistance. When the membrane potential reached the spike-initiation threshold, V_{th} , it was clamped to V_{spike} (voltage at the peak of the spike) for one timestep, and then reset to V_{reset} . A refractory period $\tau_{refractory}$ prohibited spiking for a fixed duration following each spike.

Neuronal model parameters selection criteria. Excitatory neuron parameters were selected to match the loom-response curves and spike rates found in SC recordings (Fig. S5A). Inhibitory neuron parameters were generally chosen to be the same as those for excitatory neurons, with the exceptions of: membrane resistance, spiking threshold and refractory periods (Gerstner et al 2014):

Excitatory: $\tau_m=50$ ms, $R_m=4$ M Ω , $V_{th}=-50$ mV, $V_{reset}=-65$ mV, $V_{rest}=-65$ mV, $V_{spike}=30$ mV, $\tau_{refractory} = 2$ ms.

Inhibitory: $\tau_m=50$ ms, $R_m=5$ M Ω , $V_{th} = -52$ mV, $V_{reset}=-65$ mV, $V_{rest}=-65$ mV, $V_{spike}=30$ mV, $\tau_{refractory} =1.5$ ms

To simulate a population of neurons, we instantiated each excitatory and inhibitory neuron with randomly chosen values of the following four parameters from within the indicated ranges: $\tau_m = 48-52$ ms, $R_m = 3.8-4.5$ M Ω , $V_{th} = -52-49$ mV and $\tau_{refractory} = 1.5-3$ ms.

Synaptic Inputs. Synaptic inputs were modeled as exponentially decaying post-synaptic currents. Every spike caused a weight-modulated ($w_{synapse}$) step-rise in the current input to the post-synaptic neuron, which then decayed based on a time-constant τ_{decay} :

$$\tau_{decay} \left(\frac{dI}{dt} \right) = -I(t) \quad \text{Eq 2}$$

Consequently, for each spike event, a synapse would have instantaneous increments in input current as in Eq 3:

$$I(t) = I(t) + w_{synapse} \quad \text{Eq 3}$$

Synaptic weights and decay constants. The values for weights and decay constants were selected to be within the range of previously published work (Brunel 2000).

Excitatory to Inhibitory synaptic weights: $w_{exc \rightarrow inh} = 10$ a.u., $\tau = 12.0$ ms

Inhibitory to Excitatory synaptic weights: $w_{inh \rightarrow exc} = 20$ a.u., $\tau = 12.0$ ms

Inhibitory to Inhibitory synaptic weights: $w_{inh \rightarrow inh} = 60$ a.u., $\tau = 20.0$ ms

Note that the $w_{exc \rightarrow inh}$ weight was relevant only for the normalization circuit with pooling inhibition (Fig. 5A-C).

Synaptic weights were kept constant across conditions, and all excitatory post-synaptic currents contributed additively to each neuron's net synaptic current such that the input current of a neuron 'i' was given by :

$$I_i(t) = \sum_j I_{j \rightarrow i}(t) + I_{i_ext}(t) \quad \text{Eq4}$$

Here, I_{i_ext} is the external current stimulus applied to each neuron, and proportional to the strength of the stimulus (Fig. S5A)

Modeling the receptive field of input neurons. To model the Gaussian receptive field profile for input neurons, the external current I_{i_ext} was scaled by a Gaussian profile. For example, for neuron i with the azimuthal RF center located at position θ_{azim} and RF width σ_{RF} , the effective input current with a stimulus located at θ was computed as:

$$I_{i_ext}(t, \theta) = G_i(\theta_{azim}, \sigma_{RF}) * I_{i_ext}(t) \quad \text{Eq 5}$$

where $I_{i_ext}(t)$ is the unweighted external stimulus current, and $G_i(\theta_{azim}, \sigma_{RF})$ is the RF of neuron i :

$$G_i(\theta_{azim}, \sigma_{RF}) = e^{\left[\frac{-(\theta - \theta_{azim})^2}{2\sigma_{RF}^2} \right]} \quad \text{Eq 6}$$

Here, θ is the stimulus location in azimuthal space, and σ_{RF} is the receptive-field width. This multiplicative formulation in Equation 5 allowed us to evaluate the effect of the location of both S1 and S2 on every neuron's net input current.

Network circuit diagram. We modeled two circuit motifs in this study, first, the *normalization circuit with pooled inhibition* (Fig. 5A-C; (Mahajan & Mysore 2022, Wong & Wang 2006) and, second, the *donut-like mutual inhibition circuit* (Fig. 5D-F; (Mahajan & Mysore 2022, Mysore & Knudsen 2013, Wang et al 2004). Each circuit contained two input channels corresponding to the two excitatory inputs S1 and S2, each of which injected external currents (I_{i_ext} , see Eq 4) denoted respectively as I_{S1} and I_{S2} into the input neurons. The synaptic inputs to each neuron in the models were computed as described in Equations 2 and 3.

Simulating competitive responses with an outside-RF competitor, using the relative-strength dependent competition protocol. For our network simulations, we adapted the competition protocol based on previously published experimental protocols in both owls and mice (Kothari et

al 2026, Mysore et al 2011). Briefly, two stimuli (S1 and S2; simulated as input currents I_{s1} and I_{s2} ; Eqns 4 and 5) were simultaneously presented as inputs to the network. One stimulus, S1, was considered the inside RF stimulus and its strength was held constant at I_{i_extS1} , whereas the second stimulus, S2, was presented outside the receptive field and its strength was systematically varied (I_{i_extS2}). The resulting responses from this protocol have been termed previously as the competitor strength–response profile (CRP; (Kothari et al 2026, Mysore et al 2011)). The strength of the S2 stimulus was varied over 8-10 equal steps, ranging from below the strength of S1 to above it; S1's strength was chosen as the middle 6th or 7th value in this range.

Analysis of simulated responses to the competition protocol. All analyses were performed using custom Matlab code. Sigmoids were fit to the responses of neuron Ex1 (Fig. S5E,F), which receives the constant strength S1 stimulus. Best sigmoidal fits to CRPs were obtained by using a nonlinear least squares estimation procedure (*nlinfit* command in MATLAB). The transition range of a CRP (i.e., precision of the neural selection boundary) was defined as the range of S2 strengths over which responses dropped from 90% to 10% of the total range of responses. The transition point of a CRP was defined as the strength of the S2 stimulus at which responses to the stimulus pair changed abruptly from high to low values, and was numerically equal to the midpoint of the transition range of the CRP. The categorization index was computed in the standard manner, per previously published work (Freedman & Assad 2006, Kothari et al 2026, Mahajan & Mysore 2022, Mysore & Knudsen 2011).

RESULTS

Two-stimulus interactions within the classical RF: computational rule

As a first step in investigating stimulus competition across space in the SCid, we asked what computational rules underlie short-range interactions between two stimuli occurring

simultaneously within the classical RF. Studies in monkeys have shown that distinct stimuli presented inside the RF of SCid neurons interact competitively, with subsequent behavioral cues modulating this competition (Li & Basso 2005). To understand the computational rule(s) underlying such short-range competition in the mouse SCid, we examined how the responses of a neuron to a stimulus centered in its receptive field (S1) are affected by a second stimulus (S2) also located inside the receptive field. We used expanding dot stimuli as they produced robust responses compared to stationary dots of the same radius (Fig. S1C). In these experiments, S1 was presented at the center of the RF, and S2, with a faster expansion rate than S1, was presented at 2-4 locations also within the RF, always ensuring that the two stimuli did not overlap physically in space. These conditions were interleaved with trials where either S1 or S2 was presented alone at the same respective locations (Fig. 1A).

We observed that the responses to the simultaneous presentation of S1 and S2 were consistently lower than the linear sum of the responses to each stimulus alone, i.e., were sub-additive, and followed an averaging rule (Fig. 1B right bottom; [S1] = 23.59 sp/s, [S2] = 64.07 sp/s, [S1&S2] = 47.88 sp/s). The time course of response to [S1&S2] diverged significantly from the responses to each of the stimuli (Fig. 1B, right-top; time-to-divergence (TTD) = 85 ms w.r.to S1 alone; TTD = 225 ms w.r.to S2 alone; Methods). Across the population, sub-additive responses were found in 77/78 of paired locations. Additionally, the averaging rule held true across the population: a linear regression model fit to the responses across all stimulus location pairs (Fig. 1C) revealed a slope of 0.53, while the intercept was 4.65 sp/s. To determine whether averaging could be explained by response saturation (rather than inhibitory interactions), we compared responses in the two-stimulus condition with the maximum response evoked by either S1 or S2 presented alone at any location within the RF. We found that for the overwhelming majority of cases (89.7%), the two-stimulus responses were lower than this maximum single-stimulus response (Fig. S2A), indicating

that neurons were capable of firing at rates higher than those observed in the two-stimulus condition. Thus, the observed averaging cannot be readily explained by response saturation, and is instead consistent with suppressive interactions between the stimuli. During these experiments, eye (in orbit) movements were measured and found to be negligible in size, thereby offering no confounds with respect to the actual retinotopic positions of the stimuli (Fig. S2B-D, see Methods).

Overall, these findings reveal that the net response to two stimuli presented within the RF of mouse SCid neurons is well-approximated by an averaging rule operating on the responses to the individual stimuli.

Two stimulus interactions across the RF boundary: the computational rule

Beyond short-range competitive interactions, SCid neurons across vertebrate species also show competitive suppression of their evoked responses by a second stimulus presented far outside the classical RF (Basso & Wurtz 1998, Mysore et al 2010, Rizzolatti et al 1974). However, the computational rules underlying such long-range interactions between distant stimuli are not known in mammals. To address this question, we examined how the responses of a neuron to a stimulus inside its RF (S1) are affected by a second stimulus (S2) outside the RF. Specifically, we presented a stimulus, S1, at different azimuthal or elevational locations spanning the RF, and the second, stronger stimulus, S2, well outside the RF, (Fig. 2A, median distance of S2 from the RF center = 38.4°). We used expanding dot stimuli again, and fixed S2 to be stronger than S1 (expansion rates for S1: 19 ± 0.8 deg/s, S2: 43.6 ± 1.3 deg/s). Note that, by definition of the RF, S2, when presented alone, does not evoke responses different from baseline.

We observed that the responses to the simultaneous presentation of S1 and S2 were lower than the responses to S1 alone (Fig. 2B,C; and therefore, also to the sum of the responses to S1 and

S2). In other words, the introduction of S2 suppressed the responses to the RF stimulus (Fig. 2B,C). To quantify this sub-additive relationship, we regressed the responses in the paired condition (S1 & S2) against those to S1 alone for spatial positions of S1 within the RF. The slope of the regression line was significantly less than 1, while the intercept was not significantly different from zero (Fig. 2C, bottom), indicating a divisive interaction rule. Across the population, the divisive rule was consistent, with the slope of the linear fit to the normalized data being 0.66 (1/divisive factor; Fig. 2D), and y-intercept being 7% (Fig. 2D); a statistically significant, but small, additive factor. During these experiments as well, eye (in orbit) movements were measured and found to be negligible in size, thereby offering no confounds with respect to the actual retinotopic positions of the stimuli (Fig. S2B-D, see Methods).

Overall, these findings revealed that the net response to two stimuli occupying locations across the RF boundary (with one inside and one outside) was well-approximated by a divisive rule operating on the responses to S1.

Spatial extent and profile of inhibition in the classical surround

Our results, thus far, show that stimulus interactions within the RF follow an averaging rule, whereas those across the RF boundary (with one stimulus outside the RF) follow a divisive rule. However, both produced two-stimulus responses that were sub-additively related to the individual responses, suggesting the involvement of inhibitory mechanisms. Might these two types of stimulus interactions utilize a shared inhibitory mechanism? To begin addressing this question, we examined the spatial organization (extent and profile) of the inhibition underlying each.

We first examined the spatial organization of inhibition operating within the classical RF, i.e., of the classical inhibitory surround. For this, we adopted a standard protocol used in the literature to study the classical surround: we presented bars of increasing length at the center of the RF (Fig. 3A). We found that as bar lengths increased, responses first increased to a peak value, followed by a steady decline, eventually saturating to a steady-state value for longer bars (Fig. 3B).

The shape of such response profiles has been well-explained by a classic combination of a narrow, strong excitatory field and a spatially wider, but weaker inhibitory surround (Mysore et al 2010, Sceniak et al 1999). Such a combination would predict that for smaller bar lengths, an increasing amount of the excitatory field would get engaged as bar length increased, leading to an increase in response. For intermediate lengths, the positive effect of the spatially restricted excitatory field would be overcome by the negative effect of the wider suppressive surround, resulting in a decline in the net response. For very large bar lengths, the net contributions of the excitatory and inhibitory fields would become constant, producing a steady-state response.

To test for this “center-surround” structure, we fit the data with a classic difference of Gaussians model (see Methods). The best fit indeed resulted in a narrow excitatory Gaussian and a wider, inhibitory Gaussian, resulting in a Mexican-hat receptive field profile (Fig. 3C). The radius of the inhibitory surround for the example site was estimated to be 30° (see Methods). Across a population of 22 neurons, the median radius was 34° (Fig. 3D left). We also quantified the strength of asymptotic suppression, i.e., constant suppression at large bar lengths, using a suppression index (SI), defined as the normalized reduction of the asymptotic response from the peak response. Across the population, the median SI was 0.51 (Fig. 3D right).

Overall, these results revealed that a spatially restrictive classical surround provides inhibition inside the RF, with its strength decreasing with distance from the RF center.

Spatial extent and profile of inhibition in the extraclassical surround

Next, we examined the spatial organization of inhibition operating outside the classical RF. The divisive modulation of evoked responses by a stimulus outside the RF suggests a more expansive extraclassical suppressive field. To characterize the extent and spatial profile of this inhibition, we measured the effect of varying the location of a second stimulus outside the RF on the responses to an RF stimulus. Briefly, we presented an expanding dot stimulus, S1, at the center of the RF of SCid neurons, and a second, stronger, expanding dot stimulus, S2, at various locations outside the RF. We systematically varied S2's location along the azimuthal axis, both in contralateral and ipsilateral space (relative to the recording site), while maintaining its elevation the same as that of S1 (Fig. 4A,B). In addition, we measured single-stimulus spatial tuning by presenting either S1 or S2 alone at different azimuth locations (Fig. 4C top). The single-stimulus and two-stimulus conditions were randomly interleaved. For each outside-RF location of S2, we computed the percentage change in response relative to the condition when the RF stimulus, S1, was presented alone at the RF center (Fig. 4C bottom).

As expected, we found that the presentation of S2 produced a strong suppression in the responses to S1. Notably, we found this suppression for all locations of S2 outside the RF (Fig. 4B,C): suppression was significant at locations right outside the RF (approx. 30° from the RF center) and extended up to the tested limit of 100° (Fig. 4D).

To identify any patterns of suppression across the azimuthal space, we pooled responses from all the recorded SCid neurons in different ways. First, we examined whether suppression strength depended on the distance between S1 and S2. To this end, we aligned the RF centers of all the neurons and measured the change in response as a function of the distance of S2 from the RF center (Fig. 4D): we found that suppression strength remained constant across all distances ($p = 0.279$, one-way ANOVA with Holm-Bonferroni correction), with a net average suppression of 35.82%.

Next, we investigated if there were any inherent asymmetries in the suppressive interactions along the frontal $\rightarrow$ peripheral (versus peripheral $\rightarrow$ frontal) axis (Fig. 4E). To this end, we examined the suppression exerted by peripheral stimuli onto neurons with frontal RFs, and vice versa. Frontal RFs were defined as those whose centers were $\leq 30^\circ$ in azimuth (Fig. 4E inset – orange zone), whereas peripheral RFs had centers $> 30^\circ$ in azimuth (Fig. 4E inset – blue zone). For each group, we calculated the suppression from S2 locations in the complementary region of space. We found that neurons with frontally located RFs experienced marginally weaker suppression than those with peripherally located RFs (Fig. 4E, $p < 0.001$, t-test).

Finally, we tested for any systematic differences in the strength of competitive suppression experienced by neurons in the intermediate versus deep layers of the SC. We identified the putative layer of a neuron based on anatomical recording depth as well as functional signatures (Fig. S4AB), and then grouped neurons based on the identified layer. We found no discernible difference in the strength of suppression among sub-layers in the SCid (Fig. S4C).

Together, these findings reveal a widespread, effectively global, suppressive field extending across the entire azimuthal space. This extraclassical field is characterized by a lack of distance-dependence of the suppression by S2 and a consistent asymmetry in which frontal stimuli exert stronger suppression than peripheral ones.

Given the constancy and far-reaching nature of the suppression across azimuth, we next tested if these were true across elevation as well. To this end, we varied the location of S2 along the elevation axis while keeping its azimuth center at the same value as the azimuth of S1. We found no significant differences in the amount of suppression with distance from the center (Fig. 4F, $p = 0.184$, one-way ANOVA with Holm-Bonferroni correction). Additionally, we tested for any asymmetry in the strength of suppression from the upper versus lower elevation locations. Since elevations less than 0° are encoded by a relatively small portion of the SC (Drager and Hubel, 1976), we classified units with RF centers at an elevation of $> 10^\circ$ as having 'upper' elevational RFs, and those with RF centers $\leq 10^\circ$ as having 'lower' elevational RFs. In each case, we measured the amount of suppression from the complementary region of elevation space and found no significant differences between the two groups (Fig. 4G, $p = 0.851$, t-test).

Together, this distance-invariant, global competitive inhibition suggests the presence of an extraclassical surround mechanism distinct from that underlying the decaying-with-distance, local inhibition observed within the classical RF.

Computational modeling of two-stimulus interactions across the SCid space map

Our experiments have revealed different computational rules and interaction profiles for the two spatial scales of stimulus interactions across the SCid space map of mice (Figs. 1-4). A critical open question, then, is whether a common circuit mechanism underlies competitive interactions both within and outside the receptive field (RF), or whether two distinct mechanisms are needed to account for the observed results. To gain insights into potential underlying mechanisms, we turned to computational modeling (Methods). Briefly, our core network model had two 'channels', each corresponding to a spatial location and each consisting of excitatory and inhibitory neurons (Fig. 5). The neurons were modeled as spiking (leaky integrate-and-fire) neurons, and the values for various model parameters were grounded in published experimental findings (Methods; (Burkitt 2006, Gerstner et al 2014, Mysore & Quartz 2005)). To address questions of circuit mechanisms, we implemented different connectivity diagrams in our network model, and examined whether (and which ones) accounted for the observed experimental findings on stimulus competition.

As a first step, we first considered our experimental results showing that competitive interactions within the RF follow the averaging rule. These results are consistent with prior reports of response averaging across vertebrate species, including the primate SCid (Li & Basso 2005), bird OTid (Mysore et al 2010), and primate visual cortex (Reynolds et al 1999). Because such averaging in the visual cortex has been previously modeled using normalization circuits (Reynolds et al 1999), we tested whether our SCid data in mice (Fig. 1) could also be explained by a normalization mechanism. We implemented a well-established circuit diagram for normalization model with pooled inhibition in our network (Fig. 5A; Methods; (Mahajan & Mysore 2022, Wong & Wang 2006)).

To simulate within-RF stimulus interactions in our model (Fig. 5A left; Fig. 1A), we set the spatial RFs of the neurons in each channel to overlap significantly with those of the neurons in the other channel (Fig. 5A, center). Channel 1 was designated the 'recording' channel, and S1 was 'presented' at the center of the RF of its excitatory neuron (Ex1). S2 was 'presented' to channel 2, which encoded an adjacent location to S1. Notably, by virtue of our design choice of overlapping RFs between the two channels, S2 was also located within the RF of Ex1 (the neuron being recorded). When S1 and S2 were 'presented' simultaneously to each of the two channels, respectively, thus mimicking the within-RF experiment (Fig. 1A), we found that the responses of the 'recorded' model neuron, Ex1, closely reproduced within-RF experimental results from Fig. 1 (Fig. 5A right). Specifically, Ex1's responses to simultaneous S1 & S2 presentation were consistently lower than the sum of the individual responses, and followed an averaging rule (Fig. S5B, example neuron; Fig. 5A, right; population summary).

We next tested whether the same normalization model could also account for two-stimulus interactions **across the boundary of the RF** (Fig. 5B, left). To do so, we set the spatial RFs of the neurons in the two channels to be far away from each other (i.e., no spatial overlap; Fig. 5B, center). We found, again, that the responses of Ex1 closely reproduced outside-RF experimental results from Fig. 2 (Fig. 5B). Specifically, introducing S2 outside the RF caused suppression of Ex1's responses to the RF stimulus, and followed a divisive rule (Fig. S5C: example model neuron; Fig. 5B right: population summary of the slope and intercept from modeling). These results (Figs. 5AB) indicate that a classic, pooled normalization circuit **could, in principle**, serve as a common mechanism for competitive interactions both within and across the boundary of the RF of SCid neurons, in line with previous suggestions (Basso & May 2017).

However, if normalization is the common mechanism governing both spatial scales of competition, the same circuit should also explain another recent finding about across-RF-boundary competitive interactions in the mouse SCid (Kothari et al 2026) (Fig. S5E). In that experiment, responses of SCid neurons were measured to a fixed-strength stimulus inside the RF (S1) while simultaneously presenting a second, competing stimulus (S2) of varying strength outside the RF. In other words, this experiment involved varying the relative strength between the competitor stimulus and the RF stimulus while maintaining both their locations (i.e., relative distance) fixed. Using this competition protocol that tested the encoding of relative stimulus strength, SCid neurons were shown to signal categorically the strongest stimulus across the SCid space map (Fig. S5E). Thus, these results showed that the strength of competitive inhibition depends (categorically) on the relative strengths of the competing stimuli. Here, we tested if these results as well could be accounted for by the normalization model. We did so by presenting to our model, S1 of fixed strength, and a distant S2 of varying strength (Fig. 5C, left).

We found that the normalization circuit model **failed to produce categorical signaling**. Responses of the SCid RF neuron (Ex1; Fig. 5C center) decreased gradually with increasing S2 strength, rather than exhibiting an abrupt, categorical drop (Fig. S5F: example neuron). To quantify the categoricalness of the response profile, we computed the categorization index (CI; (Freedman & Assad 2006, Kothari et al 2026, Mahajan & Mysore 2022); Methods). The categorization index (CI) was low (Fig. 5C right: CI population summary, blue data) compared to the experimental data from Kothari et al, 2026 (Fig. 5C, right, gray data), demonstrating that pooled normalization cannot discriminate categorically the strongest stimulus from *other* weaker competitors. These results agree with recent modeling of experimental data from avians, which also showed that normalization with pooled inhibition is ineffective for producing categorical selection (Mahajan & Mysore 2022). Thus, the normalization model **cannot** account for a key

finding on across-RF-boundary competition, indicating that it cannot serve as a common mechanism for within as well as across RF boundary stimulus interactions.

What, then, might be a viable mechanism for long-range, across-RF-boundary stimulus interactions in the mouse SCid? Previous work in avians provides a potential avenue of investigation. A specialized, donut-like inhibitory motif implemented in the avian SC (called optic tectum, OT) by a satellite inhibitory nucleus called the Imc (nucleus isthmus pars magnocellularis; (Mysore & Knudsen 2013)) has been identified as being critical for categorical stimulus selection by OTid neurons in avians (Majahan & Mysore 2022). Recently, the mouse analog of the avian Imc, called PLTi (parabigeminal lateral tegmental inhibitory complex), was identified, shown to be connected bidirectionally with the SC, and was demonstrated to be involved in target selection for spatial attention in freely behaving mice (Kothari et al 2026). We, therefore, decided to test whether this circuit motif can account for both key aspects of across-RF-boundary stimulus interactions observed above in the mouse SCid, namely: the divisive inhibitory rule due to a fixed strength S2 (Fig. 2; Fig. 5B), and the categorical profile of responses due to a varying strength S2 (i.e., of competitive suppression as a function of relative stimulus strength) (Kothari et al 2026) (Fig. S5E and Fig. 5C).

We first tested the donut-like motif's ability to account for the relative-strength dependent responses (Fig. 5D left and center). We found that, unlike the normalization model, it successfully generated **categorical, switch-like responses** when the strength of an outside-RF S2 was varied systematically (Fig. S5F: example neuron). The categorization index of these response profiles was high (Fig. 5D right, blue data: CI population summary), revealing the ability of this circuit to robustly discriminate the strongest stimulus from weaker competitors. We then tested

the donut-like inhibitory motif's ability to account for the observed divisive suppression (Fig. 5E left and center). We found that, similar to the normalization circuit, the donut-like circuit successfully reproduced divisive suppression of S1 responses by an outside-RF S2, matching the experimental results in Fig. 2 (Fig. S5D: example neuron; Fig. 5E right: population summary of the slope and intercept). These findings indicate that a donut-like mutual inhibition circuit can account for both the key signatures of stimulus competition in the SCid involving a second stimulus that lies outside the RF: namely, a divisive inhibitory rule, with the strength of divisive inhibition depending on the relative strengths of the competing stimuli.

As a final step, we wondered whether this structured, donut-like inhibitory circuit might also be able to account for within-RF interactions, thereby serving as a potential common model for all two-stimulus interactions across the SCid space map. To address this question, we first considered the anatomical connectivity of the donut-like inhibitory motif: it is defined by the self-sparing pattern of spatial inhibition that it implements. That is, the inhibitory neuron of each spatial channel delivers stimulus-evoked inhibition to all other spatial channels except itself. By contrast, the construction of the classical inhibitory surround, which governs the within-RF interactions, is driven by inhibition which peaks at the center of the spatial channel and decays with distance from it, as revealed by the bar-length experiments (Fig. 3). In other words, the inhibitory mechanism for within-RF interactions necessarily involves each spatial channel inhibiting itself. This anatomical argument predicts that the donut-like inhibitory motif is fundamentally incompatible with within-RF interactions. For completeness, we tested directly with model simulations, the efficacy of the donut-like model for within-RF interactions. As expected, results from our simulation of the donut-like motif for within-RF interactions did **not** match experimental results (Fig. 5F, right panel, $p=9.5e-10$, *two-sample T-test* between experimental and simulated values of the ratio $[S1 \& S2] / [S1] + S2]$). Further examination of the simulation results revealed

an additional insight regarding this mismatch. Specifically, that when $S1 > S2$ (i.e., when $S1$ was stronger), model outcomes did align very well with experimental ones (Fig. S5G first and second panels from right; green plotted data). However, it was when $S2 > S1$ (i.e., when $S2$ was stronger) that model outcomes were starkly different from experimental ones (Fig. S5G first and second panels from right; blue plotted data). In other words, the donut-like (mutually suppressing) motif causes $S2$ to overwhelmingly suppress $S1$, consistent with the categorization it produces. Taken together, these results demonstrate that the donut-like inhibitory motif is not a viable mechanism for the observed within-RF interactions.

Combined, our results indicate that two distinct inhibitory circuit mechanisms govern stimulus interactions across the SCid space map: (1) a pooled normalization-like mechanism for within-RF stimulus competition (Fig. 5GH), but (2) a distinct, donut-like inhibitory mechanism for long-range, across-RF-boundary competition (Fig. 5GH).

Discussion

In this study, we investigated the principles underlying competitive stimulus interactions across two spatial scales in the SCid, namely those governed by a classical inhibitory surround and those governed by an extraclassical inhibitory surround.

Computations underlying stimulus interactions within the classical versus the extraclassical surrounds

Computationally, stimulus interactions within the RF as well as across the RF boundary were sub-additive, signaling the involvement of inhibitory influences (Basso & May 2017). However, the

nature of these sub-additive interactions turned out to be different. Within the classical surround, stimulus interactions were best described by an averaging rule, where responses to two stimuli approximated the mean of their individual responses. Similar sub-additive interactions have been reported previously in the primate SC (Li & Basso 2005), the barn owl OT (Mysore et al 2010), and cortical areas (Reynolds et al 1999), and such averaging is consistent with normalization models in which responses are balanced by pooled inhibition driven by all the inputs (Carandini et al 2005, Carandini & Heeger 2011, Reynolds & Heeger 2009).

By contrast, interactions of an RF stimulus with competitors outside the RF followed a divisive suppression rule. These interactions across the RF boundary, that do not involve changes in relative strength, seemed well accounted by normalization mechanisms: both by our modeling (Fig. 5B), and as suggested by past experimental work (Basso & Wurtz 1998, Vokoun et al 2014). However, the inclusion of relative-strength-dependent two-stimulus interactions revealed a different picture. SCid neurons across vertebrate species have been shown to exhibit categorical response profiles, switching abruptly from one stimulus representation to another as relative salience (or priority) of the competing stimuli changes (Kothari et al 2026, Mysore et al 2011, Mysore & Knudsen 2014). Such categorical response profiles aid winner-take-all selection among distant competing stimuli across the SCid space map, especially when their relative salience or priority are similar (Kothari et al 2026, Mahajan & Mysore 2022, McPeck & Keller 2004, Mysore et al 2011, Mysore & Knudsen 2014), and are critical for the function of SCid (Kothari et al 2026, Lovejoy & Krauzlis 2010, McPeck & Keller 2004, Mysore & Kothari 2020). These relative-strength-dependent categorical dynamics, however, could not be accounted for by standard pooling normalization models (Fig. 5C). Instead, they required a self-sparing inhibitory motif (Fig. 5D-F) in which inhibitory neurons driven by one stimulus suppress the representations of the competing stimuli but not their own; a finding consistent with the recent discovery of self-sparing inhibition as the circuit mechanism underlying categorical responses in the owl OTid (Mahajan & Mysore

2022). (This motif, incidentally, also implements gain control, one of the classic functions ascribed to pooled normalization mechanisms (Mahajan & Mysore 2022)). Taken together, our results reveal the necessity of distinct circuit mechanisms for mediating local versus global competitive interactions in the SCid.

Classical versus extraclassical surrounds in visual processing and stimulus selection

We show that the classical and extraclassical surrounds in SCid exhibit fundamentally different properties. Classical surrounds are spatially localized, with the strength of inhibition decreasing with distance from the center. Typically, such surrounds are thought to shape single stimulus responses: receptive field sizes and tuning to certain features (Ito et al 2017). However, when multiple stimuli are presented in the RF, the resulting responses could be either integrative or competitive: cross-modal interactions within the RF often produce additive or super-additive effects, while unimodal interactions often produce sub-additive effects. Considering that neurons in the SCid play a major role in relative priority computations, it is reasonable to suppose that sub-additive interactions between spatially resolvable stimuli of the same modality are competitive rather than integrative, even when they are presented within the same RF (Alvarado et al 2007). Indeed, experiments in the primate SC have shown that cueing one of two simultaneously presented stimuli as the target for a subsequent saccade can shift neural responses from averaging to a winner-take-all-like representation (Li & Basso 2005), just as in cortical areas (Reynolds et al 1999). Thus, classical inhibitory surrounds may support both cross-modal integration and short-range, within-modal competition, allowing local networks to flexibly integrate multiple nearby stimuli or to prioritize just one.

By contrast, extraclassical surrounds are spatially expansive and distance-invariant, enabling global suppression across the visual field. Notably, this distance-invariant competitive

suppression is distinct from the position-invariant encoding of stimuli in the deep SC (Lee et al 2020), where neurons exhibit short-latency responses to single stimuli across a wide range of locations within the RF, likely owing to feedforward excitatory connections. By contrast, the invariance reported in our study relates to the invariance in suppression strength from a competing stimulus, likely arising from lateral inhibitory connections. Additionally, these competitive interactions also extend across the two hemispheres, consistent with reports of global, intra- and cross-hemispheric inhibitory surrounds reported in the cat SC (Rizzolatti et al 1973, Rizzolatti et al 1974) and owl OT (Mysore et al 2010). We also found that frontal stimuli exert marginally stronger suppression on peripheral representations, potentially reflecting a weak, but ethologically-relevant, asymmetry in rodents for prioritizing threatening stimuli occurring in the frontal space. Overall, the observed pattern of global inhibition is consistent with requirements for constructing a map of relative stimulus strengths (or priorities) wherein stimulus strengths (priorities) across all spatial locations need to be evaluated to locate the highest priority stimulus (Knudsen 2018, Mahajan & Mysore 2018).

Circuit mechanisms for local versus global inhibition

How might these surrounds be constructed in the SCid? The SC contains a large complement of GABAergic neurons (Mize & Luo 1992). Spatially localized inhibition consistent with that required for the construction of a classical surround has been reported in rodent SC slices (Lee & Hall 2006, Phongphanphnee et al 2011). Functionally, local GABAergic inhibition in the SCid has been demonstrated to influence visually guided behavior, as its activation impairs behavioral performance in orientation change detection tasks (Hu et al 2019, Wang et al 2020). Thus, short-range inhibition for shaping within-RF stimulus interactions likely originates within the SCid, although the extent to which they are produced locally versus inherited from earlier processing stages remains unclear.

The SCid, however, lacks extensive, long-range inhibitory projections required for the construction of a global surround (Lee & Hall 2006). Although long-range horizontal GABAergic neurons have been described in mouse SCid, their relatively limited axonal projections (mean length $\approx 325 \mu\text{m}$; range 228–586 μm) appear insufficient, by themselves, to provide inhibitory coverage across the full collicular map (Sooksawate et al., 2011). Moreover, projections were characterized primarily along the mediolateral axis, and the spatial extent of inhibition along the rostrocaudal axis remains less understood. Additionally, collicular projections in mice are reported to be patchy and restricted to specific spatial zones rather than being globally uniform: studies have reported rostro-caudal asymmetry (Bayguinov et al 2015) or exclusive rostral-rostral or caudal-caudal connectivity patterns (Doykos et al 2020). Thus, currently described intra- and inter-collicular inhibitory circuits are unlikely to account for the spatially uniform extraclassical suppression observed here, raising the possibility that the underlying inhibitory signals originate largely outside the SCid.

What sources extrinsic to the SC potentially fit the bill? Although long-range competitive interactions have been previously reported in the retina (Baccus 2007, Lee 1996), these cannot explain inhibition resulting from the opposite hemisphere or from multisensory stimuli (as shown also in birds; (Mysore et al 2010)). Similarly, inputs from the substantia nigra are also unlikely to serve as the source for global competitive inhibition in the SCid as they function by a focal, stimulus-driven disinhibition mechanism that is suited for gating rather than broad suppression (Hikosaka & Wurtz 1985). In our recent work, we identified another candidate inhibitory nucleus called the PLTi, situated in the brainstem tegmentum, that provides widespread inhibition to the SC (Kothari et al 2026). Bilateral silencing of this nucleus disrupts target selection in a spatial attention task, without any impairments to motor movements. In previous works, the avian homolog of the PLTi, called the lmc, has been demonstrated not only as a source of competitive suppression in the owl optic tectum (Mysore & Knudsen 2013) but also as an active substrate for the construction of

extraclassical surrounds (Schryver & Mysore 2023). Furthermore, our results, here, from bar-length tuning experiments show that broad, symmetrical activation of the extraclassical surround of SCid neurons does not produce unlimited suppression. Rather, suppression plateaus out, suggesting the presence of reciprocal inhibition among the inhibitory neurons mediating the extraclassical surround (Mysore et al 2010, Mysore & Knudsen 2012, Mysore & Knudsen 2013, Goddard et al 2014). Together, these converging pieces of evidence suggest that local SCid circuits mediate classical surround suppression, whereas extrinsic, long-range sources - likely the PLTi - implement extraclassical suppression and generate the global inhibitory field needed for distance-invariant competition. Investigating the molecular identity and microcircuit connectivity of the local inhibitory neurons in the SC that may help construct the classical inhibitory surround, as well as the role of PLTi in constructing the extraclassical surround of SCid neurons, are two exciting directions of future work.

Author Contributions: SPM, AB and NBK conceived the project. AB and NBK performed the experiments and analyses. NBK performed the computational modeling. AB and NBK generated the figures. AB, NBK and SPM wrote the paper.

Data and Code Availability: The data and software code used to generate the figures in this study are available at this Zenodo repository: <https://doi.org/10.5281/zenodo.21842129>

References

- Allen KM, Lawlor J, Salles A, Moss CF. 2021. Orienting our view of the superior colliculus: specializations and general functions. *Curr Opin Neurobiol* 71: 119-26
- Alvarado JC, Vaughan JW, Stanford TR, Stein BE. 2007. Multisensory versus unisensory integration: contrasting modes in the superior colliculus. *J Neurophysiol* 97: 3193-205
- Baccus SA. 2007. Timing and computation in inner retinal circuitry. *Annu Rev Physiol* 69: 271-90
- Basso MA, May PJ. 2017. Circuits for Action and Cognition: A View from the Superior Colliculus. *Annu Rev Vis Sci* 3: 197-226
- Basso MA, Wurtz RH. 1998. Modulation of neuronal activity in superior colliculus by changes in target probability. *J Neurosci* 18: 7519-34
- Bayguinov PO, Ghitani N, Jackson MB, Basso MA. 2015. A hard-wired priority map in the superior colliculus shaped by asymmetric inhibitory circuitry. *J Neurophysiol* 114: 662-76
- Brunel N. 2000. Dynamics of sparsely connected networks of excitatory and inhibitory spiking neurons. *J Comput Neurosci* 8: 183-208
- Burkitt AN. 2006. A review of the integrate-and-fire neuron model: I. Homogeneous synaptic input. *Biol Cybern* 95: 1-19
- Carandini M, Demb JB, Mante V, Tolhurst DJ, Dan Y, et al. 2005. Do we know what the early visual system does? *Journal of Neuroscience* 25: 10577-97
- Carandini M, Heeger DJ. 2011. Normalization as a canonical neural computation. *Nat Rev Neurosci* 13: 51-62
- Desimone R, Duncan J. 1995. Neural mechanisms of selective visual attention. *Annu Rev Neurosci* 18: 193-222

853 Doykos TK, Gilmer JI, Person AL, Felsen G. 2020. Monosynaptic inputs to specific cell types of
854 the intermediate and deep layers of the superior colliculus. *J Comp Neurol* 528: 2254-68

855 Fecteau JH, Munoz DP. 2006. Saliency, relevance, and firing: a priority map for target selection.
856 *Trends Cogn Sci* 10: 382-90

857 Franklin KB, Paxinos G. 2019. *Paxinos and Franklin's the Mouse brain in stereotaxic coordinates,*
858 *compact: The coronal plates and diagrams*. Academic press.

859 Freedman DJ, Assad JA. 2006. Experience-dependent representation of visual categories in
860 parietal cortex. *Nature* 443: 85-8

861 Gerstner W, Kistler WM, Naud R, Paninski L. 2014. *Neuronal dynamics: From single neurons to*
862 *networks and models of cognition*. Cambridge University Press.

863 Gingras G, Rowland BA, Stein BE. The differing impact of multisensory and unisensory integration
864 on behavior. *J Neurosci*. 2009 Apr 15;29(15):4897-902. doi: 10.1523/JNEUROSCI.4120-
865 08.2009. PMID: 19369558; PMCID: PMC2678542.

866 Goddard CA, Mysore SP, Bryant AS, Huguenard JR, Knudsen EI. 2014. Spatially reciprocal
867 inhibition of inhibition within a stimulus selection network in the avian midbrain. *PLoS One*
868 9: e85865

869 Goldberg ME, Bisley JW, Powell KD, Gottlieb J. 2006. Saccades, saliency and attention: the role
870 of the lateral intraparietal area in visual behavior. *Prog Brain Res* 155: 157-75

871 Hikosaka O, Wurtz RH. 1985. Modification of saccadic eye movements by GABA-related
872 substances. II. Effects of muscimol in monkey substantia nigra pars reticulata. *J*
873 *Neurophysiol* 53: 292-308

874 Hu F, Kamigaki T, Zhang Z, Zhang S, Dan U, Dan Y. 2019. Prefrontal Corticotectal Neurons
875 Enhance Visual Processing through the Superior Colliculus and Pulvinar Thalamus.
876 *Neuron* 104: 1141-52 e4

877 Ito S, Feldheim DA, Litke AM. 2017. Segregation of Visual Response Properties in the Mouse
878 Superior Colliculus and Their Modulation during Locomotion. *J Neurosci* 37: 8428-43

879 Itti L, Koch C. 2001. Computational modelling of visual attention. *Nature reviews. Neuroscience*
880 2: 194-203

881 Knudsen EI. 2007. Fundamental components of attention. *Annu Rev Neurosci* 30: 57-78

882 Knudsen EI. 2018. Neural Circuits That Mediate Selective Attention: A Comparative Perspective.
883 *Trends Neurosci* 41: 789-805

884 Koch C, Ullman S. 1987. Shifts in Selective Visual Attention: Towards the Underlying Neural
885 Circuitry In *Matters of Intelligence*, pp. 115-41

886 Kothari NB, Banerjee A, Zhang Q, You WK, Mysore SP. Evolutionarily old brainstem neurons are
887 required for the control of selective spatial attention. *Nat Commun.* 2026 Apr
888 28;17(1):5849. doi: 10.1038/s41467-026-72340-9. PMID: 42050354; PMCID:
889 PMC13333841.

890 Krauzlis RJ, Lovejoy LP, Zenon A. 2013. Superior colliculus and visual spatial attention. *Annu*
891 *Rev Neurosci* 36: 165-82

892 Lee BB. 1996. Receptive field structure in the primate retina. *Vision Res* 36: 631-44

893 Lee KH, Tran A, Turan Z, Meister M. 2020. The sifting of visual information in the superior
894 colliculus. *Elife* 9

895 Lee P, Hall WC. 2006. An in vitro study of horizontal connections in the intermediate layer of the
 896 superior colliculus. *J Neurosci* 26: 4763-8

897 Li X, Basso MA. 2005. Competitive stimulus interactions within single response fields of superior
 898 colliculus neurons. *J Neurosci* 25: 11357-73

899 Lovejoy LP, Krauzlis RJ. 2010. Inactivation of primate superior colliculus impairs covert selection
 900 of signals for perceptual judgments. *Nat Neurosci* 13: 261-6

901 Mahajan NR, Mysore SP. 2018. Combinatorial Neural Inhibition for Stimulus Selection across
 902 Space. *Cell Rep* 25: 1158-70 e9

903 Mahajan NR, Mysore SP. 2022. Donut-like organization of inhibition underlies categorical neural
 904 responses in the midbrain. *Nat Commun* 13: 1680

905 Mathis A, Mamidanna P, Cury KM, Abe T, Murthy VN, et al. 2018. DeepLabCut: markerless pose
 906 estimation of user-defined body parts with deep learning. *Nat Neurosci* 21: 1281-89

907 McPeck RM, Keller EL. 2004. Deficits in saccade target selection after inactivation of superior
 908 colliculus. *Nat Neurosci* 7: 757-63

909 Meyer AF, O'Keefe J, Poort J. Two Distinct Types of Eye-Head Coupling in Freely Moving Mice.
 910 *Curr Biol.* 2020 Jun 8;30(11):2116-2130.e6. doi: 10.1016/j.cub.2020.04.042. Epub 2020
 911 May 14. PMID: 32413309; PMCID: PMC7284311.

912 Michael AM, Abe ET, Niell CM. Dynamics of gaze control during prey capture in freely moving
 913 mice. *Elife.* 2020 Jul 24;9:e57458. doi: 10.7554/eLife.57458. PMID: 32706335; PMCID:
 914 PMC7438109.

915 Mize RR, Luo Q. 1992. Visual deprivation fails to reduce calbindin 28kD or GABA
 916 immunoreactivity in the rhesus monkey superior colliculus. *Vis Neurosci* 9: 157-68

917 Munoz DP, Istvan PJ. 1998. Lateral inhibitory interactions in the intermediate layers of the monkey
918 superior colliculus. *J Neurophysiol* 79: 1193-209

919 Mysore SP, Asadollahi A, Knudsen EI. 2010. Global inhibition and stimulus competition in the owl
920 optic tectum. *J Neurosci* 30: 1727-38

921 Mysore SP, Asadollahi A, Knudsen EI. 2011. Signaling of the strongest stimulus in the owl optic
922 tectum. *J Neurosci* 31: 5186-96

923 Mysore SP, Knudsen EI. 2011. Flexible categorization of relative stimulus strength by the optic
924 tectum. *J Neurosci* 31: 7745-52

925 Mysore SP, Knudsen EI. 2012. Reciprocal inhibition of inhibition: a circuit motif for flexible
926 categorization in stimulus selection. *Neuron* 73: 193-205

927 Mysore SP, Knudsen EI. 2013. A shared inhibitory circuit for both exogenous and endogenous
928 control of stimulus selection. *Nat Neurosci* 16: 473-8

929 Mysore SP, Knudsen EI. 2014. Descending control of neural bias and selectivity in a spatial
930 attention network: rules and mechanisms. *Neuron* 84: 214-26

931 Mysore SP, Kothari NB. 2020. Mechanisms of competitive selection: A canonical neural circuit
932 framework. *Elife* 9

933 Mysore SP, Quartz SR. *Proceedings. 2005 IEEE International Joint Conference on Neural*
934 *Networks, 2005.2005*, 5: 2766-71. IEEE.

935 Phongphanphanee P, Mizuno F, Lee PH, Yanagawa Y, Isa T, Hall WC. 2011. A circuit model for
936 saccadic suppression in the superior colliculus. *J Neurosci* 31: 1949-54

937 Quiroga RQ, Nadasdy Z, Ben-Shaul Y. 2004. Unsupervised spike detection and sorting with
938 wavelets and superparamagnetic clustering. *Neural Comput* 16: 1661-87

939 Reynolds JH, Chelazzi L, Desimone R. 1999. Competitive mechanisms subserve attention in
940 macaque areas V2 and V4. *J Neurosci* 19: 1736-53

941 Reynolds JH, Heeger DJ. 2009. The normalization model of attention. *Neuron* 61: 168-85

942 Rizzolatti G, Camarda R, Grupp LA, Pisa M. 1973. Inhibition of visual responses of single units in
943 the cat superior colliculus by the introduction of a second visual stimulus. *Brain Res* 61:
944 390-4

945 Rizzolatti G, Camarda R, Grupp LA, Pisa M. 1974. Inhibitory effect of remote visual stimuli on
946 visual responses of cat superior colliculus: spatial and temporal factors. *J Neurophysiol*
947 37: 1262-75

948 Sakatani T, Isa T. 2004. PC-based high-speed video-oculography for measuring rapid eye
949 movements in mice. *Neuroscience research* 49: 123-31

950 Sceniak MP, Ringach DL, Hawken MJ, Shapley R. 1999. Contrast's effect on spatial summation
951 by macaque V1 neurons. *Nat Neurosci* 2: 733-9

952 Schryver HM, Mysore SP. 2019. Spatial Dependence of Stimulus Competition in the Avian
953 Nucleus Isthmi Pars Magnocellularis. *Brain Behav Evol* 93: 137-51

954 Schryver HM, Mysore SP. 2023. Distinct neural mechanisms construct classical versus
955 extraclassical inhibitory surrounds in an inhibitory nucleus in the midbrain attention
956 network. *Nat Commun* 14: 3400

957 Schryver HM, Straka M, Mysore SP. 2020. Categorical signaling of the strongest stimulus by an
958 inhibitory midbrain nucleus. *J Neurosci*

959 Thompson KG, Bichot NP. 2005. A visual salience map in the primate frontal eye field. *Prog Brain*
960 *Res* 147: 251-62

961 Vokoun CR, Huang X, Jackson MB, Basso MA. 2014. Response Normalization in the Superficial
962 Layers of the Superior Colliculus as a Possible Mechanism for Saccadic Averaging.
963 *Journal of Neuroscience* 34: 7976-87

964 Wang L, McAlonan K, Goldstein S, Gerfen CR, Krauzlis RJ. 2020. A Causal Role for Mouse
965 Superior Colliculus in Visual Perceptual Decision-Making. *J Neurosci* 40: 3768-82

966 Wang Y, Major DE, Karten HJ. 2004. Morphology and connections of nucleus isthmi pars
967 magnocellularis in chicks (*Gallus gallus*). *J Comp Neurol* 469: 275-97

968 Wong KF, Wang XJ. 2006. A recurrent network mechanism of time integration in perceptual
969 decisions. *J Neurosci* 26: 1314-28

970

Figure legends

Fig. 1. Computational rule underlying two-stimulus interactions within the RF. **A**, Schematic of the experimental setup and stimulus protocol. One visual stimulus (S1, black dot) was presented at the center of the recording neuron's RF (dotted line), and a second stimulus (S2, blue) was presented at a non-overlapping location within the RF. **B**, Responses from an example SCid neuron; Black: S1 alone; Blue: S2 alone; Red: S1 & S2. (Left) Raster plots of responses to S1 presented alone at 0° azimuth (top), S2 presented alone at 14° azimuth (middle), and both presented simultaneously (bottom). (Right, top) Average instantaneous firing rate responses to S1 alone, S2 alone, or S1 and S2 presented together. Time-to-divergence (TTD, see Methods) of response to S1 and S2 presented together from S1 presented alone (black arrow), or S2 presented alone (blue arrow). (Right, bottom) Mean $\pm$ SEM of the responses corresponding to the rasters in Left. **C**, Population summary of responses to paired stimuli [S1 & S2] plotted against the sum of mean responses to individual stimuli ([S1]+[S2]) for all paired locations of S1 and S2 ($n = 26$ neurons, 78 paired locations, 5 mice). Red line indicates best-fit from a linear regression model with slope = 0.53 (not significantly different from 0.5, $p = 0.107$), and an intercept of 4.65 sp/s (significantly different from 0, $p < 0.05$). (Inset) Distribution of the ratio of responses to [S1 & S2] and the sum of individual responses ([S1]+[S2]).

Fig. 2. Computational rule underlying two-stimulus interactions across the RF boundary. **A**, Schematic of the experimental setup and stimulus protocol. One visual stimulus (S1, black dot) was presented at different azimuthal (or elevational) locations within the RF to measure the azimuthal (or elevational) tuning curve. S1 was presented either without (left) or with (right) a second, stronger stimulus (S2, red) at a distant, contralateral location outside the RF. **B**, Raster plots of responses to stimulus S1 presented alone at different azimuths (left) or along with stimulus S2 presented 35° away from the RF center (right). **C**, (Top) Responses of the example neuron in

B plotted as a function of azimuthal location of S1, with (red) or without (black) the second stimulus, S2. Data indicate mean $\pm$ SEM. Solid lines indicate the best-fit Gaussian to the responses in each condition. (Inset) Average instantaneous firing rate responses to S1 alone (black) or S1 and S2 presented together (red). Time-to-suppression (TTS, see Methods) of the best response to S1 by S2, as indicated by the blue arrow, is 155 ms. (Bottom) Scatter plot comparing responses to S1 and S2 presented together ([S1 & S2]) against responses to S1 presented alone ([S1 alone]). Responses for only those locations at which S1 was inside the RF (i.e., at which S1 elicited above-baseline activity; see Methods) are plotted. The dashed line indicates the best-fit regression line, with a slope of 0.67, significantly different from 1 ($p = 0.0067$) and an intercept of 4.44 sp/s that is not significantly different from 0 ($p = 0.238$). **D**, Population summary ($n=40$ neurons, 6 mice; 35 azimuthal tuning curves, and 5 elevational tuning curves). The responses at each neuron were normalized to its maximum response when S1 was presented alone. Data from all the neurons were binned along the x-axis (normalized responses to S1 alone; bin size = 0.1), and the average y-value (paired responses) within each group was plotted (mean $\pm$ SEM). The best fitting line to these data was determined by linear regression to have a slope (1/divisive factor) of 0.66 (significantly different from 1, $p = 2.2e-24$; see Methods), and a y-intercept (additive factor) of 7% (significantly different from 0, $p = 3.9e-4$; see Methods). The inset shows the distributions of the slope and intercept values across the population. Darker dots indicate data significantly different from 1 (for slopes) or 0 (for intercepts).

Fig. 3. Classical inhibitory surrounds of neurons in the SCid. **A**, Schematic of experimental setup. Bars of varying lengths were presented at the center of the recording neuron's RF. **B**, Responses of an example SCid neuron to increasing bar lengths. The solid black line indicates the best-fit line estimated using a difference of Gaussian (DoG) model shown in C. Suppression index (SI, see Methods) = 0.38. **C**, Excitatory (blue) and inhibitory (red) Gaussians obtained by

fitting a DoG model to the responses in B. The net “Mexican-hat” response is shown in black. Estimated inhibitory radius for this neuron = 30° (see Methods). **D**, Boxplots and population summaries of inhibitory radii (left) and suppression indices (right) for $n=22$ neurons across $N=3$ mice. Median inhibitory surround radius = 34° and median SI value = 0.51 ($p < 0.001$).

Fig. 4. Extraclassical inhibitory surround in the SCid. **A**, Schematic of the experimental setup and stimulus protocol. In the first condition (S1 alone, black), an expanding dot stimulus (16 deg/s) was presented alone at the center of the RF. In the second, interleaved condition, S1 was accompanied by a second stimulus (52 deg/s, red) presented at different azimuthal locations outside the RF. **B**, Raster plots for an example neuron. Top: responses to S1 presented alone at the RF center (35° azimuth). Bottom: responses to paired presentation of S1 at the RF center and S2 at different azimuthal locations spanning -5° to 55° azimuth. Positive values denote contralateral location. **C**, Top: Azimuthal tuning curves for the example neuron measured independently using S1 (black) or S2 (blue). The dashed line indicates the extent of the RF (see Methods). Bottom: Percent change in responses to paired presentation of S1 and S2 compared to responses to S1 alone, plotted as a function of the location of S2 for the example neuron. Data indicate mean $\pm$ SEM. The dashed line denotes the extent of RF, estimated from the spatial tuning curve measured with S2 (top; black curve; Methods). Spatial profiles of suppression for different azimuthal locations of a competitor. **D,E** Population summary of extraclassical profiles of suppression for different azimuthal locations of S2 outside the RF (data from ipsi and contra locations have been merged). **D**, Percent change of the paired (S1 & S2) response plotted as a function of the distance of S2 from the RF center of the recording neuron. **E**, Percent change in response to frontal S2 stimuli (black dot) for peripheral RF locations (blue dot: S1, blue dashed circle: peripheral RF, blue curve: range of RF locations deemed to be “peripheral”; and in response to peripheral S2 stimuli (black dot) for frontal RF locations (orange dot: S1, orange

dashed circle: frontal RF, orange curve: range of RF centers deemed to be “frontal”), orange curve indicates RFs of neurons encoding frontal locations). All data plotted as mean $\pm$ SEM ($n = 40$ neurons, $N = 6$ mice). ‘**’ indicates $p < 0.05$, w.r.to 0. **F,G** Population summary of extraclassical profile of suppression for different elevational locations of S2 outside the RF. F, Same as D, but for different elevation locations of S2. G, Percent change in response to lower elevational S2 stimuli for upper elevation RF locations, and in response to upper elevational S2 stimuli for lower elevation RF locations. All data plotted as mean $\pm$ SEM ($n = 26$ neurons, $N = 5$ mice).

Fig. 5. Computational modeling of two-stimulus interactions across the SCid space map.

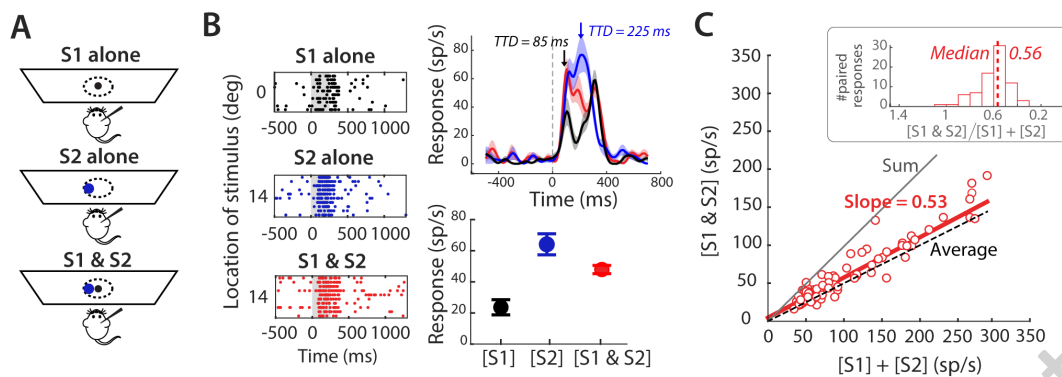
A-C, Normalization model with pooled inhibition. **(A)** Within RF competitive interactions. (Left) Schematic of stimulus protocol for simulating within RF competitive interactions using the network model; these protocols are identical to those presented to the mouse (dotted outline) in Fig. 1A. (Center) Schematic of normalization model with pooled inhibition. Ex1 and Ex2 are the input excitatory neurons that receive S_{1in} and S_{2in} input stimuli. The solid and dotted line Gaussian curves represent the overlapping receptive fields of Ex1 and Ex2. In1 and In2 are inhibitory neurons that send inhibition to both excitatory neurons (Methods). (Right) Blue: Population summary of responses to paired stimuli [S1 & S2] plotted against the sum of mean responses to individual stimuli ($[S1] + [S2]$) for all paired locations of S1 and S2 ($n = 30$ model neurons). Blue line: linear fit; dashed line: represents average ($p > 0.2$; test of slope being different from 0.5). (Inset) Blue: Distribution of the ratio of responses to [S1 & S2] and the sum of individual responses ($[S1] + [S2]$). Blue dotted line: median. Gray: distribution of experimental data from Fig. 1C, reproduced here for comparison. Simulated distribution is not significantly different from experimental distribution (1C); $p = 0.2$, two-sample t -test. For the simulation, fraction of paired responses is equal to fraction of neurons. **(B)** Across-RF-boundary competitive interactions – modulation of spatial tuning curve. (Left) Schematic of simulation protocol for modeling across

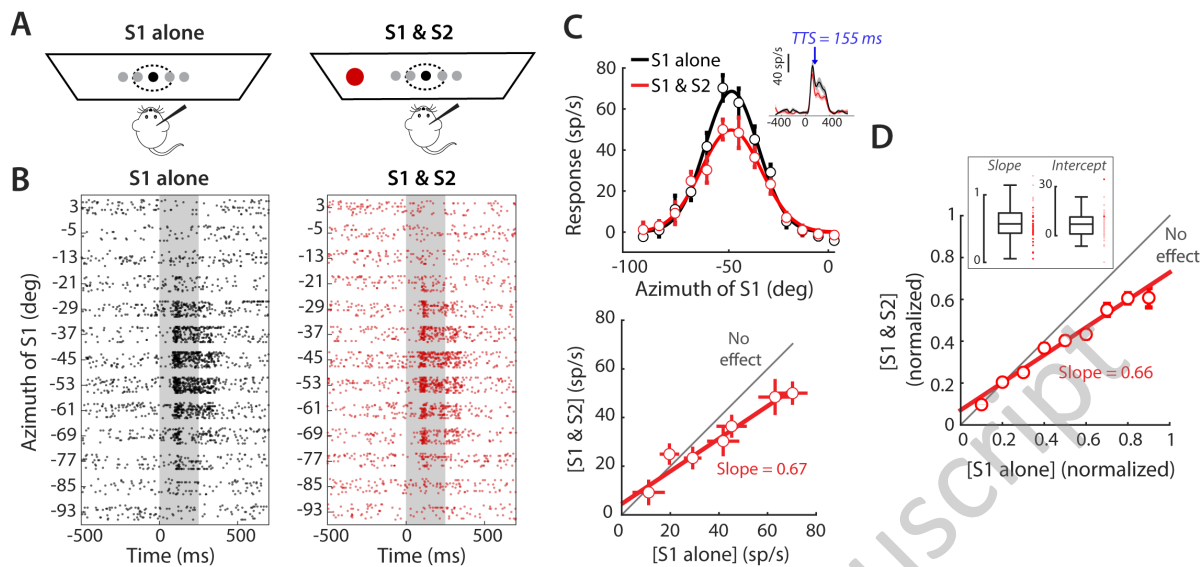
RF-boundary competitive interactions with constant relative strength between competitors (same as Fig. 2). (Center) Schematic of normalization model with pooled inhibition. Same as in panel A, except that the receptive fields of Ex1 and Ex2 do not overlap. (Methods). (Right) Blue: Population summary of the slope and intercept ($n=20$ model neurons; Methods; Slope: median = 0.38, slope significantly different from 1, $p < 1.0183e-16$, t-test against 1, results show strong divisive component, similar to experimental results from Fig. 2D; Intercept: median = 0.68, intercept not significantly different from 0, $p=0.3$, t-test against 0. Gray solid line: median of slope and intercept of experimental results (Fig. 2D) shown here for comparison purposes. **C**) Across-RF-boundary interactions dependent on relative-strength: categorical responses. (Left) Schematic of simulation protocol for modeling across RF-boundary relative strength dependent competitive interactions (same as in (Kothari et al 2026)). The varying strength of the outside RF stimulus (S_2) is depicted by three partially overlapping solid circles of increasing size. (Center) Schematic of normalization model with pooled inhibition. Same as in panel B. (Right) Blue: Population summary of the categorization index ($n=20$ model neurons; CI; mean=0.33, SD=0.023; Methods). Gray: Experimental data from Fig. S5E (Kothari et al, 2026); reproduced here for comparison ($n=16$ neurons). Horizontal bars: mean $\pm$ sem. $p<0.001$; test of model distribution (blue) being different from experimental distribution (grey). **D-F**, Donut-like inhibition model (**D**) Across-RF-boundary competitive interactions dependent on relative-strength: categorical responses. (Left) Schematic of simulation protocol (same as panel C). (Center) Schematic of model with donut-like inhibition. Ex1 and Ex2 are the input excitatory neurons and In1 and In2 are input inhibitory neurons, that receive S_{1in} and S_{2in} input stimuli. The solid and dotted line gaussian curves represent the receptive fields of Ex1, Ex2, In1 and In2. The critical difference between the donut model and normalization model is that there is no self-inhibition in the donut model (i.e. In1 and In2 do not inhibit Ex1 and Ex2, respectively; Methods). (Right) Blue: Population summary of the categorization index ($n=20$ model neurons; CI; mean=0.57, SD=0.09; Methods). Gray: Experimental data from Fig. S5E (Kothari et al, 2026), reproduced here for comparison ($n=16$

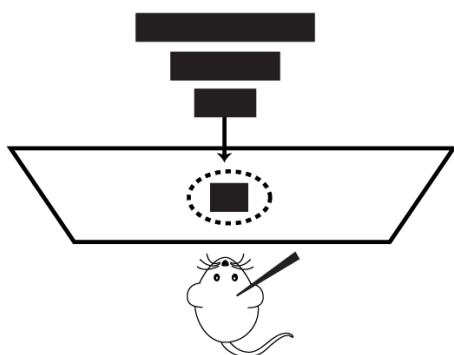
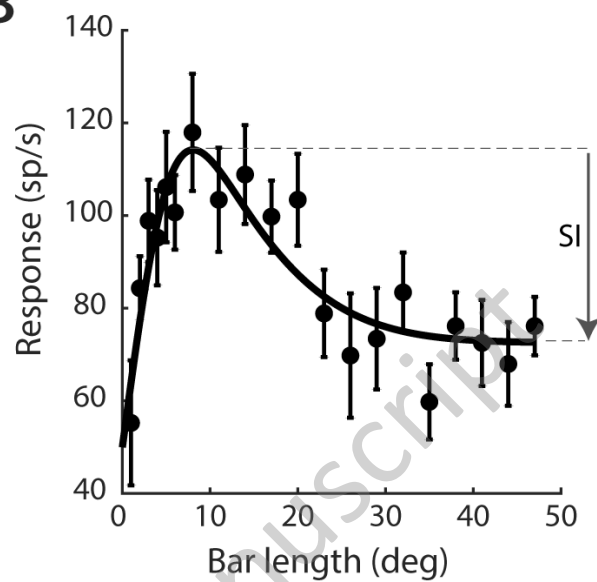
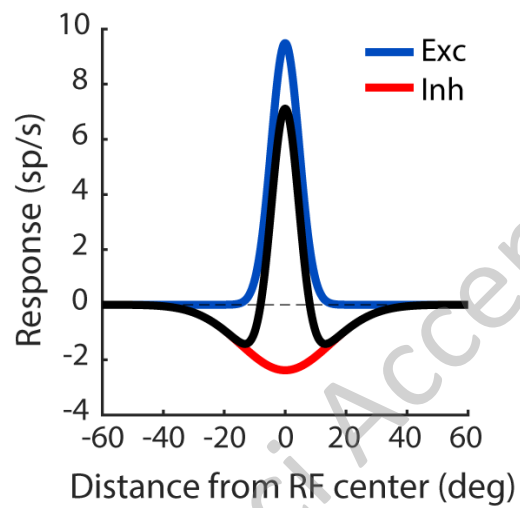
neurons), reproduced here for comparison. $p=0.38$; test of model distribution (blue) being different from experimental distribution (grey). **(E)** Across-RF-boundary competitive interactions – modulation of spatial tuning curve. (Left) Schematic of simulation protocol (same as panel B). (Center) Schematic of model with donut-like inhibition. Same as in panel D. (Right) Blue: Population summary of the slope and intercept ($n=20$ model neurons; Methods; Slope: median=0.42, slope significantly different from 1, $p < 0.001$, t-test against 1, results show strong divisive component, similar to experimental results from Fig. 2D; Intercept: median=0.12 not significantly different from 0, $p=0.31$, t-test against 0, results show small additive component, similar to experimental results from Fig. 2D. Gray solid line: median of slope and intercept of experimental results (Fig. 2D) shown here for comparison purposes. **(F)** Within RF competitive interactions. (Left) Schematic of stimulus protocol, same as in panel A. (Center) Schematic of model with donut-like inhibition (Methods). (Right) Population summary of responses to paired stimuli $[S1 \text{ \& } S2]$ plotted against the sum of mean responses to individual stimuli $([S1]+[S2])$ for all paired locations of $S1$ and $S2$ ($n = 30$ model neurons). Blue line: linear fit; dashed line: represents average ($p>0.2$; test of slope being different from 0.5). (Inset) Blue: Distribution of the ratio of responses to $[S1 \text{ \& } S2]$ and the sum of individual responses $([S1]+[S2])$. Blue dotted line: median. Gray: distribution of experimental data from Fig. 1C, reproduced here for comparison. Simulated distribution is significantly different from experimental distribution (1C); $p=5.8e-10$, two-sample t-test. For the simulation % paired responses is equal to % neurons. **(G)**, Schematic of the unified model of competitive interactions across the SCid space map. For simplicity, this schematic has just three input channels (S_{1in} , S_{2in} and S_{3in}). The normalization model (orange lines) operates on the local (within-RF) spatial scale: between $Ex1$ and $Ex2$ excitatory neurons, with $In1$ and $In2$ representing the pooled inhibition. The donut-like inhibition model (pink lines) operates on the global (across-RF-boundary) scale: between $Ex1$ and $Ex3$, with $In1$ and $In2$ implementing self-sparing, mutual inhibition. **(H)**, Simplified summary of the unified model for competitive interactions across the SCid space map. This involves two distinct mechanisms operating on the

1123 *short, within-RF spatial scale (orange; normalization), versus the longer range, global, across-*
1124 *RF-boundary spatial scale (pink; donut-like or mutual inhibition).*

JNeurosci Accepted Manuscript





A**B****C****D**