



OPEN The dynamics of stimulus selection in the nucleus isthmi pars magnocellularis of avian midbrain network

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The nucleus isthmi pars magnocellularis (Imc) serves as a critical node in the avian midbrain network for encoding stimulus salience and selection. While reciprocal inhibitory projections among Imc neurons (inhibitory loop) are known to govern stimulus selection, existing studies have predominantly focused on stimulus selection under stimuli of constant relative intensity. However, animals typically encounter complex and changeable visual scenes. Thus, how Imc neurons represent stimulus selection under varying relative stimulus intensities remains unclear. Here, we examined the dynamics of stimulus selection by in vivo recording of Imc neurons' responses to spatiotemporally successive visual stimuli divided into two segments: the previous stimulus and the post stimulus. Our data demonstrate that Imc neurons can encode sensory memory of the previous stimulus, which modulates competition and salience representation in the post stimulus. This history-dependent modulation is also manifested in persistent neural activity after stimulus cessation. We identified, through neural tracing, focal inactivation, and computational modeling experiments, projections from the nucleus isthmi pars parvocellularis (lpc) to "shepherd's crook" (Shc) neurons, which could be either direct or indirect. These projections enhance Imc neurons' responses and persistent neural activity after stimulus cessation. This connectivity supports a Shc-lpc-Shc excitatory loop in the midbrain network. The coexistence of excitatory and inhibitory loops provides a neural substrate for continuous attractor network models, a proposed framework for neural information representation. This study also offers a potential explanation for how animals maintain short-term attention to targets in complex and changeable environments.

Keywords Stimulus competition, Dynamics, Excitatory loop, Sensory memory, Attractor network

Abbreviations

Imc	Isthmi pars magnocellularis
lpc	Isthmi pars parvocellularis
OT	Optic tectum
FR	Firing rate
RF	Receptive field
PCRP	Previous competitor response profiles

The midbrain network, most elaborated and conserved across evolution in birds, controls spatial attention¹. It contains circuits that continuously compute the highest priority stimulus location (stimulus selection) and route sensory information from the selected location to forebrain networks that make cognitive decisions¹. Animals like birds frequently encounter dynamic visual scenes caused by self-motion or target movement, leading to challenges such as shifting luminance patterns and partial occlusions. Therefore, investigating the dynamic properties of stimulus selection in midbrain network is critical for understanding its functional mechanisms in natural environments.

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The midbrain network consists of the superior colliculus (SC, in mammals) or optic tectum (OT, in nonmammals), which encodes a topographic map of multisensory and motor space, as well as a spatial map of stimulus priority^{2,3}, and satellite brain areas (the isthmus nuclei) in the midbrain tegmentum that are interconnected with the SC/OT^{4–8}. OT is a laminar structure, divided into superficial (retinal neuronal projection area⁹), intermediate and deep layers¹⁰. The isthmus nuclei consist of the nucleus isthmi pars magnocellularis (Imc, GABAergic), pars parvocellularis (Ipc, cholinergic), and pars semilunaris (SLu)¹¹. A central node of the avian midbrain selection network is the “shepherd’s-crook” neurons (Shc), which relay retinal inputs arriving to the superficial tectal layers to the Imc, Ipc and SLu^{7,11–13}. The Imc has a specialized pattern of antitopographic connectivity with the intermediate and deep layers of OT (OTid) and Ipc^{7,11,14}, and reciprocal inhibitory projections among Imc neurons (inhibitory loop)¹⁵. The Ipc neurons maintain precise reciprocal retinotopic connection with the OT^{7,11}.

Stimulus competition protocols have been widely employed to investigate stimulus selection mechanisms in midbrain networks, typically involving two components: a fixed-intensity visual stimulus (S1) presented within a neuron’s receptive field (RF) and a competing stimulus (S2, visual or auditory) with graded intensities presented at distal locations^{16,8,15–27}. Neural responses were analyzed by averaging firing rates, with paired S1–S2 presentations generating characteristic competitor response profiles (CRPs) that quantify intensity-dependent interactions^{8,15,18,20,22–24,26}. Stimulus competition in OTid and Imc exhibits global properties: any stimulus outside a neuron’s RF suppresses or eliminates responses to an RF-internal visual stimulus^{16,17}. The neural representation of stimulus competition depends on relative physical stimulus intensity^{8,19,20}, and is mediated by Imc-derived inhibitory projections^{14,23,27}. Notably, Imc neurons signal the strongest stimulus more categorically, and earlier than the OTid neurons⁸. Additionally, the responses of Imc neurons encode the salience of stimuli, including spatial and temporal saliency^{28,29}.

Here, we performed in vivo extracellular recordings in pigeons, focusing on Imc. To simulate dynamic visual scenes encountered in natural environments, we designed two-segment spatiotemporally successive visual stimuli. Our electrophysiological data revealed that Imc neurons retain sensory memory of the previous stimulus, which modulates competition and salience representation for the post stimulus. This history-dependent modulation was reflected in persistent neural activity after stimulus cessation. Using anterograde neural tracing (viral injection in Ipc), focal Ipc inactivation, and computational modeling, we examined Ipc neuronal projections and their effects on Imc responses.

Materials and methods

Statement: (1) all experiments were conducted in accordance with the Animals Act, 2006 (China) for the care and use of laboratory animals and were approved by the Life Science Ethical Review Committee of Zhengzhou University; (2) all experiments were performed in accordance with ARRIVE guidelines (<https://arriveguidelines.org>).

Animal preparation

Given that sex differences in animals are rarely considered in researches that are conducted at the level of subcortical neurons and that most of the relevant works on midbrain network and Imc^{16,23,26,30–32} do not pay attention to animal sex, the neuronal recordings in this study were conducted in male and female pigeons (*Columba livia*, 350–450-g body weight). The pigeons were purchased from local feedlots and housed in individual wire mesh cages under a 12:12-h light–dark cycle with free access to water and food.

Surgery and neurophysiology

Experiments were performed following protocols that have been described previously^{28,33}. Briefly, surgery was initiated after the animals were anaesthetised with 20% urethane (1mL/100 g body weight). Most existing experiments on the Imc and OT were performed under anesthesia^{27,34}, therefore, experiments in this study were performed in anesthetized animals. When the birds closed their eyes and no longer responded to auditory or painful stimulation (mechanical stimulation such as pinching foot), meaning that they were in a state of deep anesthesia, they were transferred to a stereotaxic device. Their heads were placed in the stereotaxic holder, the right eye was kept open and moisturized with saline solution during the experiment, while the left eye was covered. A small hole was drilled in the bone to expose the left midbrain, allowing access to the Imc. A small slit was then made in the dura using a syringe needle, permitting dorsoventral penetration through the Imc. Throughout the experiment, a heating panel was used to maintain the pigeon’s body temperature at approximately 41 °C.

The Imc was targeted using previously described methods³³. Briefly, dorsoventral penetrations through the Imc were made at a medial-leading angle of five degrees from the vertical to avoid a major blood vessel in the path to the Imc. The identification of the recording site was based on the stereotaxic coordinates and expected physiological properties of Imc neurons: high firing rates and special visual receptive fields (to the best of our knowledge, Imc neurons are the only neurons in the midbrain that have a longitudinally distributed multi-lobed receptive fields structure³¹). The Ipc lies medial to the Imc and its targeting was confirmed based on the neural response characteristics of the neurons (characteristic bursty responses¹⁸). The targeting of the Imc and Ipc was finally validated at the beginning of this study by anatomical lesions as previously described^{27,35}.

Focal inactivation

To perform focal inactivation of Ipc neurons, we first implanted spatially aligned site-pairs in Imc and Ipc using single-channel electrodes. Stereotactic localization determined Ipc neuron coordinates. Neural responses of Imc and Ipc neurons to translational motion targets of varying sizes were recorded. After removing the single-channel electrode from the Ipc neurons, a single-barrel glass electrode was implanted at the same site. Physiological

saline or 3% lidocaine hydrochloride (100nL) was then injected using a microinfusion pump (Remote Infuse/Withdraw Pump 11 Elite Programmable Syringe Pump, Harvard Apparatus) and microsyringe (Hamilton 7000, 1μL) over a period of 10 min. The glass electrode was removed 20 min after completion of the injection to begin testing neural responses.

Neurotracer

Herpes simplex virus (HSV) belongs to the Herpesviridae family and α -herpesvirus subfamily. A modified virus (HSV-EGFP, 2.00×10^9 PFU/mL, BrainVTA, Wuhan, China) enables anterograde tracing of neural output circuits in mammalian and avian experimental animals by loading fluorescent proteins.

Surgical procedures for virus injection into the Ipc mirrored those used in local inactivation tests, except for anesthesia: 1.5% sodium pentobarbital (0.2 mL/100 g body weight) was administered. After implantation of the single-barrel glass electrode into the Ipc, HSV-EGFP (150nL) was injected using a microsyringe (Hamilton 7000, 1μL) and a microinfusion pump (Remote Infuse/Withdraw Pump 11 Elite Nanomite Programmable Syringe Pump, Harvard Apparatus). Following electrode removal, exposed brain tissue was sealed with dental cement, and the skin was sutured and disinfected. Post-surgery, animals were housed individually with ad libitum access to food and water. Seventy-two hours after virus injection, cardiac perfusion was performed sequentially with 100 mL normal saline followed by 100 mL 4% paraformaldehyde. Brain tissues were extracted, submerged in 4% paraformaldehyde at 4 °C overnight, and dehydrated using gradient sucrose solutions (15% and 30%). The brain tissues were embedded in optimal cutting temperature compound (OCT, 118 mL, Sakura, United States) and stored for 24 h. The brains were sectioned in coronal planes at 25 μm thickness using a cryomicrotome (Leica CM1950, Germany). The sections were washed with PBS solution (5 min*3 times) and coverslipped with SuperKine™ Enhanced Antibody Dilution Buffer (Abbkine Scientific, Wuhan, China). The sections were subjected to fluorescence scanning (Olympus VS200, Japan).

Recording and data analysis

The neural activity was recorded under urethane anesthesia using 16-channel microelectrode arrays (0.5~1 MΩ, 4×4, Clunbury Scientific, Bloomfield Hills, Michigan, USA) and single-channel microelectrode (0.5~1 MΩ, KedouBC, Suzhou, China), which were inserted into the Ipc using a micromanipulator.

The spike signals of the units were recorded with a sampling frequency of 30 kHz and extracted with a bandpass filter (250–5 kHz). Local field potential signals were amplified (4000×), filtered (0–250 Hz), and continuously sampled at 2 kHz using a Cerebus™ recording system (Blackrock Microsystems, Salt Lake City, UT, USA). All data recorded was analyzed off-line using customized MATLAB codes.

The “Wave_clus” spike sorting toolbox³⁶, a fast and unsupervised spike sorting algorithm, was used to sort the recorded data into single units. We included in the analysis only those units that had less than 5% of the spikes within 1.5ms of each other³¹. All analyses were performed using MATLAB. The responses of neurons were measured by counting spikes during a time window following stimulus onset. This window was chosen to capture well the evoked neural responses: for the neural response intensities during stimulation, the counting window was from 0ms to 500ms with respect to stimulus onset, for the persistence response intensities after stimulus cessation, the counting window was from 500ms to 750ms with respect to stimulus onset; the delay of the neural response was defined as the interval from stimulus onset to the time when the instantaneous firing rate of the neural response reached 10% of its maximum value; and the response persistence time after stimulus cessation was the interval from stimulus offset to the time when the instantaneous firing rate decayed to 10% of its maximum value. Average rates were calculated over all presentation repetitions.

Statistical analysis

Each stimulus combination (either single or paired) was repeatedly tested 5 times in a constantly interleaved fashion (10s). The data were analyzed, and the results were fitted with sigmoids or single-peak function, if necessary. All analyses were carried out with the custom MATLAB code. Parametric or non-parametric statistical tests were applied based on whether the distributions being compared were Gaussian or not, respectively (Lilliefors test of normality). Comparing differences between paired samples by Wilcoxon rank-sum test (not following Gaussian distribution) and paired-sample t-test (follow Gaussian distribution). Data shown as $a \pm b$ refer to mean $\pm$ MSE, unless specified otherwise. The “*”, “**”, “***” symbol indicates significance at the 0.05, 0.01, 0.001 level (after corrections for multiple comparisons, if applicable), respectively. Correlations were tested using Pearson’s correlation coefficient (“cori” command in MATLAB with the “Pearson” option).

Visual stimuli

Visual stimuli were generated using the MATLAB-based Psychophysics Toolbox (Psychtoolbox-3; www.psychtoolbox.org), running on Windows, and were synchronized with the recording system. An LED monitor (112 degrees vertical × 80 degrees horizontal, running at 100 Hz, PHILIPS, 558M1R) was placed tangentially to, and 40 cm from, the pigeon’s right eye to present monocular visual stimuli. The luminance of the gray screen background was 118 cd/m² and that of the black stimuli was 0.2 cd/m². The contrast of stimuli is
$$\text{contrast} = \frac{\text{background luminance} - \text{stimuli luminance}}{\text{background luminance}}.$$

To determine the extent of space encoded by a recorded site (in Ipc or Ipc), we collected two-dimensional receptive fields (RFs, azimuth × elevation) by presenting a translational motion stimuli at various azimuthal and elevational locations. The stimulus was a black square (0.9 degrees) against a gray background, moving randomly at 90 degrees/s along a series of parallel paths (time window for the vertical and horizontal paths: 1.24 and 0.89 s, respectively) over the tangent LED monitor in front of the pigeon to map the RF of Ipc units. The target exhibits four distinct motion directions: two horizontal directions (bottom-top and top-bottom) and two longitudinal

directions (left-right and right-left). Before the formal experiments started, the RFs of Imc units were estimated by manually sliding a dot delivered by a simple program around the screen while simultaneously monitoring the firing responses. Considering the large RF of Imc neurons, and to maximize the use of the monitor area, we then adjusted the position and angle of the monitor to ensure that its long axis was parallel with the long axis of the RF of the Imc. The integration of neuronal responses to horizontal motion stimuli establishes the vertical receptive field (RF) boundaries, with vertical motion stimuli conversely delineating horizontal boundaries. Through horizontal motion mapping, we observed a characteristic multi-lobed RF architecture. Based on this configuration, the longitudinal axis of the lobe exhibiting maximal responsiveness was designated as the target trajectory.

Studies on stimulus competition predominantly employ looming stimuli to establish experimental protocols, where stimulus intensity is modulated by adjusting looming velocity. Notably, under identical velocities, the frame-wise expansion magnitude of looming targets varies with initial conditions—this variability may act as a confounding factor in neural response modulation. Our study specifically focuses on abrupt changes in relative stimulus intensity during dynamic presentation. Translational motion stimuli offer methodological rigor: modifying target contrast or size at constant speed introduces no confounding variables, thus providing superior experimental control. For investigations requiring stable relative intensity, looming stimuli remain fully consistent with experimental requirements. We endorse the methodological validity demonstrated by the Mysore team in this particular research context and acknowledge their foundational contributions.

Constant Relative Intensity Spatial Competition Protocol (CRI-SCP). In each trial, visual stimulus S1 with fixed intensity was presented within the RF, paired with stimulus S2 of varying intensities at a distant location (typically, 30° away from S1). Both stimuli maintained constant intensity parameters throughout competitive interactions. Neural responses elicited by S1-S2 pairings were designated as competitor response profiles (CRPs). Target sizes included 0°, 0.15°, 0.33°, 0.6°, 0.9°, 1.5°, and 3°, contrasts included 0, 0.21, 0.37, 0.53, 0.68, 0.84, and 1. The target contrast is set to 1 when the target size is a variable, and the target size is set to 0.6° when the target contrast is a variable. Each parameter set was repeated five times, with stimulus parameters randomized across trials. Each trial had a 500ms stimulus duration and a 10s intertrial interval. Targets moved downward at 30°/s.

Constant Intensity Single-target Protocol (CISP). This stimulation protocol presents individual targets within the RF where stimulus intensity (modulated by size or contrast) remains invariant during individual trial. Target sizes included 0°, 0.09°, 0.15°, 0.21°, 0.27°, 0.33°, 0.39°, 0.45°, 0.6°, 1.5°, 3°, 6°, 15°, contrasts included 0, 0.21, 0.37, 0.53, 0.68, 0.84 and 1. Each parameter set was repeated five times, with stimulus parameters randomized across trials. Each trial had a 500ms stimulus duration and a 10s intertrial interval. Targets moved downward at 30°/s.

Variation Relative Intensity Spatial Competition Protocol (VRI-SCP). In these protocols, visual stimuli were divided into two spatiotemporally successive segments: a previous stimulus and a post-stimulus. A fixed-intensity visual stimulus (S1) was presented within the RF, with intensity defined by either size or contrast. The stimulus parameters of the previous segment (Pre-S2) of the competing stimulus (S2) were randomly selected from a specified parameter set (size or contrast) across trials. The intensity of the post segment (Pos-S2) of S2 remained identical to that of S1. S1 and S2 were separated by an angular distance of 30° and presented simultaneously, with each segment lasting 250 ms. Target sizes included 0°, 0.15°, 0.33°, 0.6°, 0.9°, 1.5°, and 3°, target contrasts included 0, 0.21, 0.37, 0.53, 0.68, 0.84, and 1. Target contrast was fixed at 1 when size was varied, and target size was fixed at 0.6° when contrast was varied. Each parameter set was repeated five times, with stimulus parameters randomized across trials. Each trial had a 500-ms stimulus duration and a 10-s intertrial interval. Targets moved downward at 30°/s.

Variation Intensity Single-target Protocol (VISIP). In these protocols, stimulus S1 was in the RF. The visual stimuli were divided into two spatiotemporally successive segments: a previous stimulus and a post stimulus. The stimulus parameter of the previous segment (Pre-S1) of the S1 was randomly selected from a specified parameter set (size or contrast) in different trials. The intensity of the post segment (Pos-S1) of the S1 remained constant. Target sizes included 0°, 0.15°, 0.33°, 0.6°, 0.9°, 1.5°, and 3°, contrasts included 0, 0.21, 0.37, 0.53, 0.68, 0.84, and 1. Each parameter set was repeated five times, with stimulus parameters randomized across trials. Each trial had a 500ms stimulus duration and a 10s intertrial interval. Targets moved downward at 30°/s.

Model

The leaky integrate-and-fire model is a neuronal discharge model that is frequently employed in neuroscientific research. It offers the benefits of a relatively simple model structure and straightforward debugging, while maintaining fidelity to the fundamental electrophysiological characteristics of neurons. Luksch et al. employed the leakage integral issuance model to simulate the issuance pattern of oscillatory bursts from Ipc neurons in response to input visual stimuli³⁷. In this study, a spike neural network model was constructed to verify the influence of the Shc-Ipc-Shc excitatory loop on the Imc neurons. This model involved three types of neurons: Shc, Ipc, and Imc.

All neurons in the network were modeled as leaky integrate-and-fire type. The membrane potential V_i of neuron i evolves according to the differential equation, $\tau_i \frac{dV_i}{dt} = E_i - V_i + R_i I_{in,i}$, when the membrane potential V_i reaches the threshold $V_{\theta,i}$, it is instantaneously reset to $V_{reset,i}$, which is interpreted as the occurrence of a spike. The basic cellular parameters are the threshold $V_{\theta,i}$, the reset potential $V_{reset,i}$, the resting membrane potential E_i , the membrane input resistance R_i , and the membrane time constant τ_i , postsynaptic currents $I_{in,i}$, $I_{in,i} = -g_d V_i$, synaptic conductance $g_{d,i} = g_{d,i} - \frac{g_{d,i}}{\tau_{g,i}} t_{step} + input_i$, $input_i$ are the input.

Shc neurons receive excitatory projections from RGC and Ipc neurons, $input_{Shc} = \omega_{RGC \rightarrow Shc} P_{RGC} + \omega_{Ipc \rightarrow Shc} P_{Ipc}$, where $\omega_{RGC \rightarrow Shc} = 1.3$ and $\omega_{Ipc \rightarrow Shc} = 2.3$ are the synaptic weights of RGC to Shc and

Ipc to Shc, respectively, $P_i = \begin{cases} 1, & \text{neuron } i \text{ fire spike} \\ 0, & \text{else} \end{cases}$, $V_{\theta,Shc} = -45$, $V_{reset,Shc} = -60$, $\tau_{Shc} = 10$,

$E_{Shc} = -55$, $\tau_{g,Shc} = 10$.

Ipc neurons receive excitatory projections from Shc neurons, $input_{Ipc} = \omega_{Shc \rightarrow Ipc} P_{Shc}$, where $\omega_{Shc \rightarrow Ipc} = 2$ the synaptic weights of Shc to Ipc, $E_{Ipc} = -55$, $V_{\theta,Ipc} = -50$, $V_{reset,Ipc} = -60$, $\tau_{g,Ipc} = 10$, $\tau_{Ipc} = 10$.

Imc neurons receive excitatory projections from Shc neurons, $input_{Imc} = \omega_{Shc \rightarrow Imc} P_{Shc}$, where $\omega_{Shc \rightarrow Imc} = 2$ the synaptic weights of Shc to Imc, $E_{Imc} = -55$, $V_{\theta,Imc} = -50$, $V_{reset,Imc} = -60$, $\tau_{g,Imc} = 10$, $\tau_{Imc} = 10$.

The model developed in this study is a simplified neuronal model consisting of only three types of neurons (Shc, Ipc, and Imc neurons). The parameter settings for these neurons were adopted from the neuronal model presented in Luksch's study³⁷.

Result

A total of 34 animals were used in the experiment, of which 4 animals were specifically used for Ipc neurons inactivation experiments and 3 animals were used for neural tracer experiments.

The relative salience of stimuli determines the neural representation of stimulus competition

Previous studies on stimulus selection often used looming stimuli to construct stimulus protocols. The results showed that the neural representation of stimulus selection depends on the relative physical intensity of the stimuli (i.e., looming speed). To further investigate the characteristics of stimulus selection in Imc neurons when the relative physical intensity of stimuli changes, we constructed a stimulus pattern based on translational motion stimuli (see “Materials and methods”). This study builds upon and extends the relevant experiments on stimulus selection conducted by the Mysore et al.⁸. By jointly analyzing the representation of salience^{28,29} and stimulus selection⁸ of Imc neurons, this study paves the way for subsequent research on spatial competition experiments with varying relative stimulus intensities.

Figures S1A and S2A depict the schematic of the Constant relative intensity spatial competition protocol (CRI-SCP, “Materials and methods”) in which paired competing stimuli (S1 within the Imc neuron's RF, S2 outside the RF) were presented. Competitor response profiles (CRPs) were calculated based on the mean firing rates during the 500ms stimulus period. Using this protocol, Imc neurons exhibited two distinct CRP patterns: “switch-like” (size: Fig. S1B, C; contrast: Fig. S2B, C) and “gradual-like” (size: Fig. S1D, E; contrast: Fig. S2D, E), consistent with the result of Mysore et al.⁸. These results establish that translational motion parameters (relative size or contrast) modulate the representation of stimulus selection, paralleling effects of looming velocity observed in prior studies.

For a single translational motion target, the response of the corresponding Imc neurons can characterize the salience of the target^{28,29}. In the constant intensity single-target protocol (CISP, “Materials and methods”), we quantified Imc responses to stimuli of varying intensity: size modulation in Fig. S3 and contrast modulation in Fig. S4. Figure S4B, C demonstrate contrast-dependent response enhancement (Pearson correlation, $p < 0.05$). This pattern persisted at the population level (Fig. S4D). Size-modulation experiments revealed non-monotonic responses in example Imc neurons (Fig. S3B and C), a pattern replicated population-wide (Fig. S3D). These findings align with observations reported in²⁹. A monotonic increase in neural response with target size occurred below 3° (Pearson correlation, $p < 0.05$), consistent with our previous demonstration that this threshold lies within Imc receptive field widths²⁹. Accordingly, all size-variation experiments in this study used targets $\leq 3^\circ$ unless specified.

To investigate the relationship between stimulus salience and selection representation in Imc neurons, we classified neural data from spatial competition and single-target paradigms into four categories: strong vs. weak competitor and strong vs. weak stimulus. This categorization was based on the size or contrast of S1 in the spatial competition stimuli. Within the Imc population (109 neurons for size; 55 for contrast; some neurons had two sizes of S1 in the spatial competition experiments). Population-averaged responses showed stronger suppression when $S2 > S1$ (strong competitor) versus $S2 < S1$ (weak competitor) (Fig. 1A, C; $p < 0.05$, Wilcoxon rank-sum test). Single-target stimuli confirmed higher salience for strong versus weak stimuli (Fig. 1B, D; $p < 0.05$, Wilcoxon rank-sum test). Integrated analysis of Fig. 1A–D data revealed an inverse relationship between relative stimulus salience (inside vs. outside RF) and the representation of stimulus selection. That is, the representation of stimulus selection in Imc neurons is dependent on the relative salience of stimuli.

Spatiotemporally proximal preceding stimuli modulate neural representations of current stimulus competition

To simulate the dynamically changing visual scenes faced by animals, we designed the variation relative intensity spatial competition protocol (VRI-SCP) (e.g., Fig. 2A; “Materials and methods”). In this protocol, stimulus competition occurred across two consecutive segments: the previous stimulus and the post stimulus. Within these two segments, a visual stimulus (S1) with fixed intensity (size and contrast) was presented within the RF of recorded Imc neurons. Additionally, a second visual stimulus (the previous stimulus Pre-S2 with varying intensities; the post stimulus Pos-S2 with constant intensities, identical to S1) was presented far outside the RF ($> 30^\circ$ away; see Fig. 3A). S1 and S2 were presented simultaneously, with each segment lasting 250ms. Responses to the paired S1 and S2 presentation in the post segment (mean firing rates within black bar-defined epochs in Fig. 2B–E) were collectively termed previous competitor intensity-dependent response profiles (or previous competitor response profiles, PCRPs) (Fig. 2F).

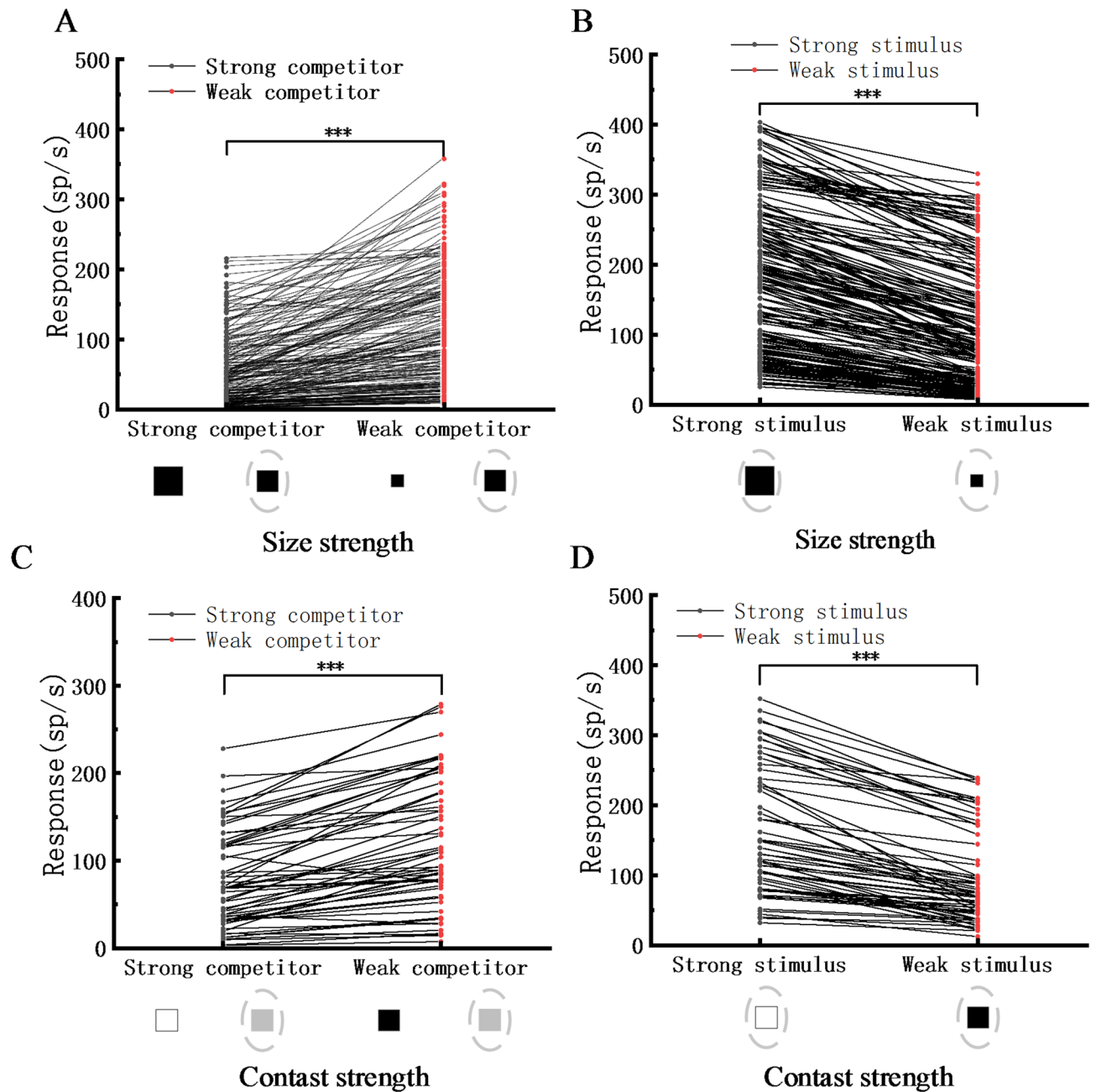


Fig. 1. Joint analysis of stimulus salience and stimulus selection representation. In the figure, ellipses represent the receptive fields of recorded Imc neurons, and squares indicate moving targets. The relative size of squares reflects the relative size in stimuli, the grayscale of squares represents contrast—darker colors indicate lower contrast (contrast experiments); A, C, The comparisons of neural responses to strong vs. weak competitors under the CRI-SCP paradigm. Stimulus within the receptive field is defined as S1, and stimulus outside as S2. Using the size or contrast of S1 as the threshold, neural responses when $S2 > S1$ are defined as strong competitors, and those when $S2 < S1$ as weak competitors, with mean values calculated for the two data types separately. The object intensity variable is size in A and contrast in C. Each pair of data represents a neuron. Neural responses under strong competitors were significantly weaker than those under weak competitors ($p < 0.001$, $n = 193$ for A; $p < 0.001$, $n = 61$ for C, Wilcoxon rank-sum test). Some neurons had two S1 sizes or contrasts in spatial competition experiments. B, D, The comparisons of neural responses to strong vs. weak stimulus under the CISP paradigm. The object intensity variable is size in B and contrast in D. The thresholds for strong/weak stimuli were identical to those in A and C. Target salience under strong stimuli was significantly higher than that under weak stimuli ($p < 0.001$, $n = 193$ for B; $p < 0.001$, $n = 61$ for D, Wilcoxon rank-sum test).

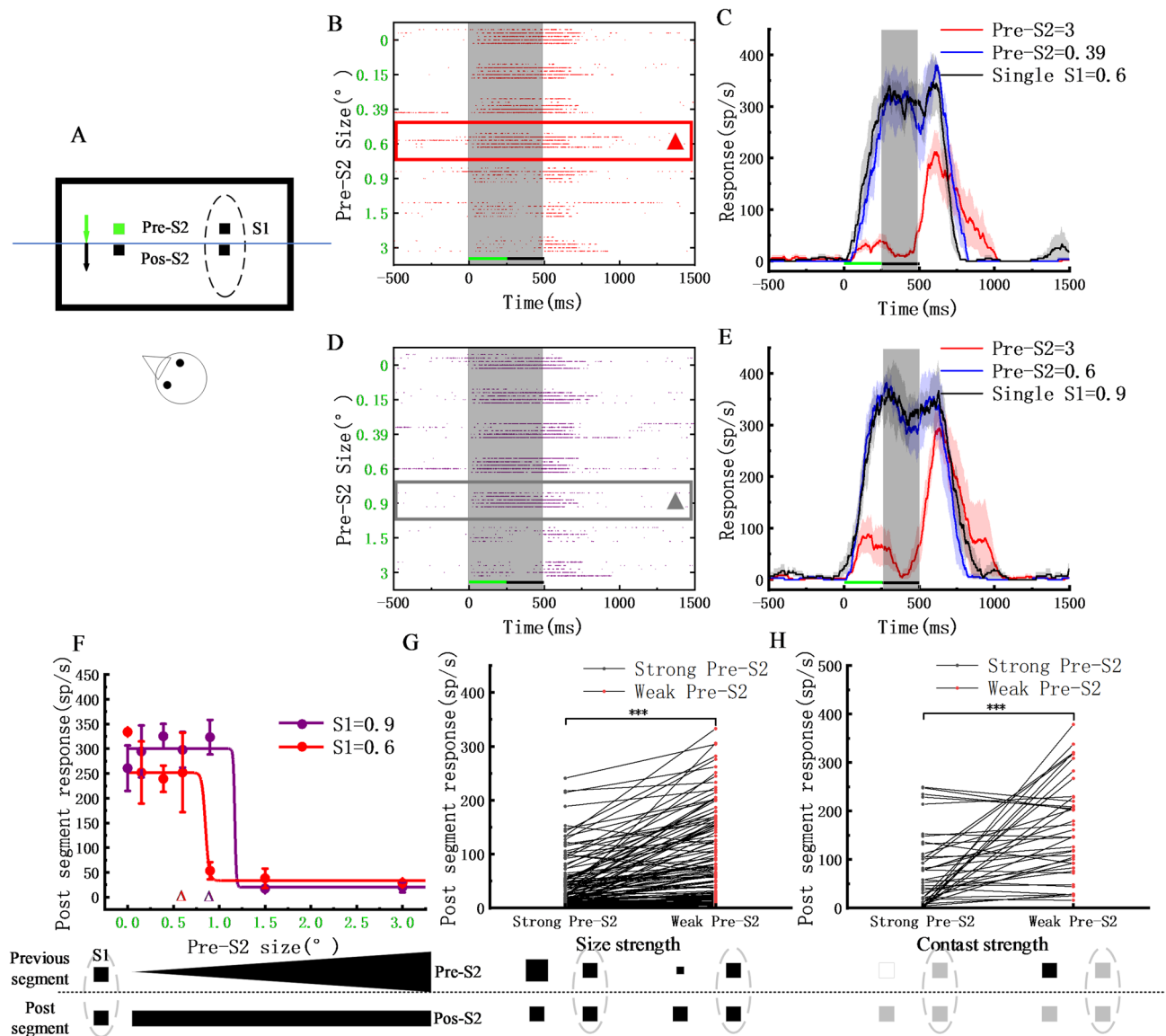


Fig. 2. The response of Imc neurons for variation relative intensity spatial competition protocol (VRI-SCP). In the figure, ellipses represent the receptive fields of recorded Imc neurons, and squares indicate moving targets. The relative size of squares reflects the relative size in stimuli, the grayscale of squares represents contrast—darker colors indicate lower contrast (contrast experiments); A, Diagram of VRI-SCP. Blue horizontal line is the dividing line between the two segments of stimulation, the up is the previous segment, the down is the post segment; Arrow, pointing in the direction of the stimulus motion; Square, the target intensity of the black square are the same and are fixed, green square are different in each trial; B, D, Raster plot of responses of the example Imc neuron to A protocols (the intensity variable of the object is the size). Shaded along the x-axis represents stimulus duration (500ms), green bar represent the previous segment, black bar represent the post segment during which response firing rates were calculated. S1, Pos-S2 = 0.6° (B), 0.9° (D). Red rectangle and triangle were the raster when S1, Pre-S1, Pos-S2 = 0.6° (B), 0.9° (D); C, E, PSTH of neurons to S1 and S2 presented together (C, Pre-S2 = 3°, 0.39° and Single S1 = 0.6°, corresponding to B, E, Pre-S2 = 3°, 0.6° and Single S1 = 0.9°, corresponding to D), computed by smoothing PSTHs (1 ms time bins) with a Gaussian kernel (SD = 20 ms; “Materials and methods”); F, Response mean firing rates corresponding to raster plots in B (red data) and D (purple data) during the post segment (black bar). Solid circle, response firing rates to paired presentation of S1 and S2 in the post segment (mean ± SEM). Correlation coefficient of responses in the post segment versus Pre-S2 intensity is -0.82 ($p = 0.02$, purple data), -0.82 ($p = 0.02$, red data), Pearson correlation test. Solid line, best fitting sigmoid to the competitor history response profile ($r^2 = 0.99$); red and black triangle, intensity of S1 (0.6°, 0.9°); G, H, The comparisons of the post segment neural responses to strong vs. weak Pre-S2 under the CRI-SCP paradigm. Using the size or contrast of S1 as the threshold, neural responses when Pre-S2 > S1 are defined as strong Pre-S2, and those when Pre-S2 < S1 as weak Pre-S2, with mean values calculated for the two data types separately. The object intensity variable is size in G and contrast in H. The neural response of the post segment under Strong Pre-S2 was significantly weaker than that of Weak Pre-S2 (G: $p < 0.001$, $n = 143$; H: $p < 0.001$, $n = 40$, Wilcoxon signed-rank tests).

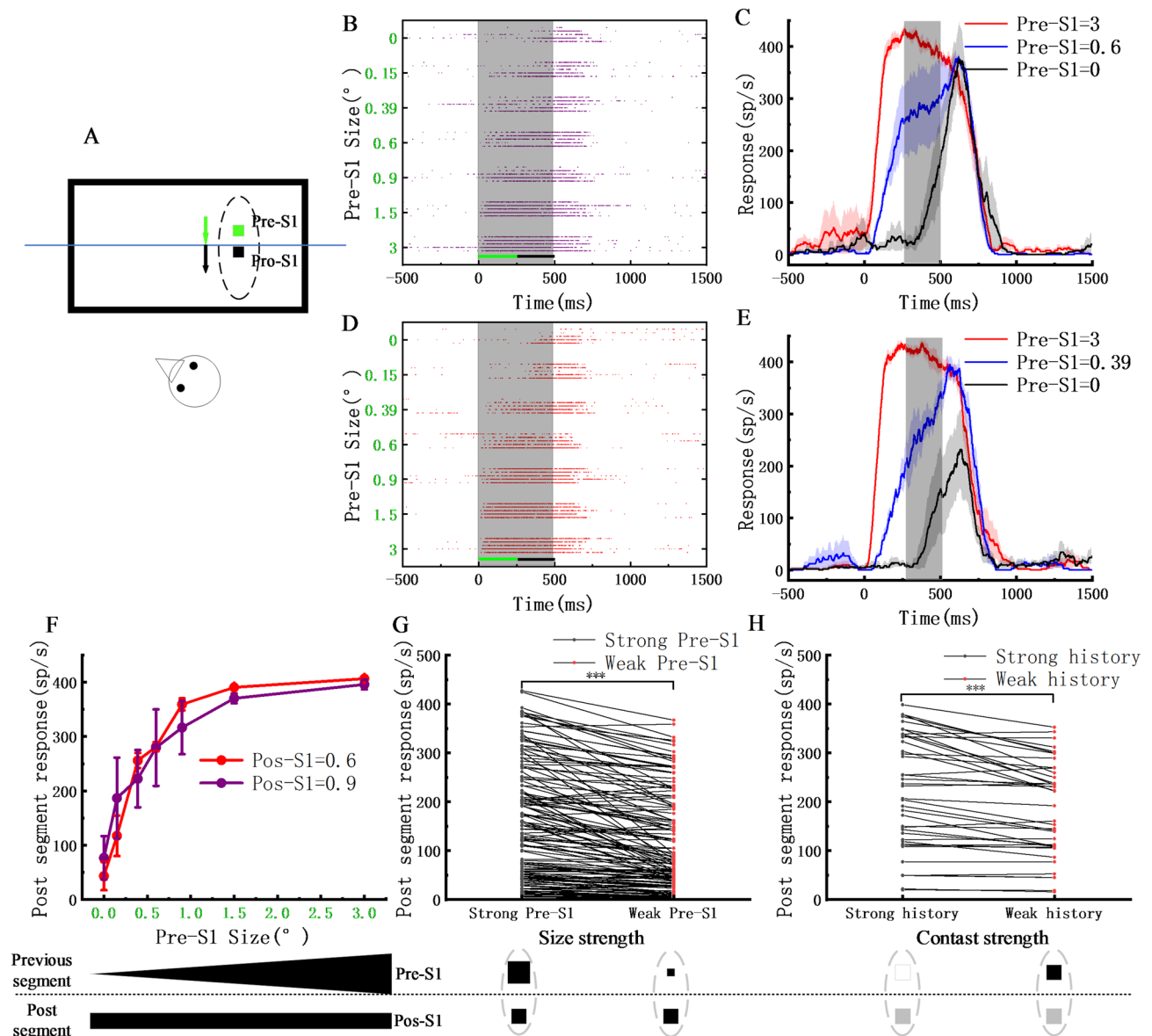


Fig. 3. The response of Imc neurons for variation intensity single-target protocol (VISP). In the figure, ellipses represent the receptive fields of recorded Imc neurons, and squares indicate moving targets. The relative size of squares reflects the relative size in stimuli, the grayscale of squares represents contrast—darker colors indicate lower contrast (contrast experiments); A, Diagram of VISP, conventions are as in Fig. 2A; B, D, Raster plot of responses of the example Imc neuron to A protocols (the intensity variable of the object is the size). Conventions are as in Fig. 2B, D. Pos-S1 = 0.9° in B, Pos-S1 = 0.6° in D; C, E, PSTH of neurons to A protocols. Conventions are as in Fig. 2C, E. Response firing rates corresponding to raster plots in B, D and F, Response mean firing rates corresponding to raster plots in (B) (red data) and (D) (purple data) during the post segment (black bar), correlation coefficient of responses versus size of the previous stimulus intensity is 0.78 (Pos-S1 = 0.6, $p = 0.03$, Pearson correlation test), 0.84 (Pos-S1 = 0.9, $p = 0.01$, Pearson correlation test); G, H, The response means of the post segment of each neuron are contrasted when the Pre-S1 > Pos-S1 (Strong Pre-S1) and Pre-S1 < Pos-S1 (Weak Pre-S1), G for different sizes of stimuli ($p < 0.001$, $n = 143$, Wilcoxon signed-rank tests), H for different contrast stimuli ($p < 0.001$, $n = 40$, Wilcoxon signed-rank tests).

We recorded 90 Imc neurons in size-modulated competition paradigms and 38 neurons in contrast-modulated paradigms (with some neurons tested with two S1 sizes or contrasts in spatial competition experiments). To analyze the response dynamics, we compared post-stimulus time histograms (PSTHs) across three conditions: Pre-S2 > S1, Pre-S2 < S1, and Single S1 (Fig. 2C, E). Notably, Imc neurons exhibited weaker transient activity when Pre-S2 > S1 compared to other conditions, despite Pos-S2 = S1 in the post segment. Figure 2F illustrates the post segment responses of an example Imc neuron under different Pre-S2 conditions, with color-coded data matching Fig. 2B and C. The red and purple curves are PCRP when S1 = 0.6° and S1 = 0.9°, respectively.

Intriguingly, these response profiles maintained neural representations of the previous segment stimulus competition despite equal stimulus sizes in the post segment.

To investigate how previous stimuli influences post stimuli processing, we compared mean responses of Imc neurons recorded during the post segment under two conditions: Pre-S2 > S1 (strong Pre-S2) and Pre-S2 < S1 (weak Pre-S2). Population-level analysis revealed significantly weaker neural responses in the strong Pre-S2 condition compared to the weak condition ($p < 0.05$, Wilcoxon signed-rank tests). Separate analyses for size and contrast modulation are presented in Fig. 2G and H, respectively.

As demonstrated in Fig. 1, during the previous segment of spatial competition, the neural responses showed stronger suppression when Pre-S2 > S1 compared to when Pre-S2 < S1, aligning with their physical intensity. Notably, in the post segment, Imc neurons maintained this suppression pattern despite equated stimulus intensities (Post-S2 = S1). Given that competitive neural representations are governed by relative salience, this phenomenon suggests that current stimulus salience might be influenced by spatiotemporally proximal preceding stimuli. To validate this hypothesis, we conducted further experiments.

Spatiotemporally proximal preceding stimuli modulate neural representations of current stimulus salience

To validate the proposed hypothesis, we developed the variation intensity single-target protocol (VISP) (see Fig. 3A; “Materials and methods”). In this protocol, stimulus competition occurred across two consecutive segments: the previous stimulus and the post stimulus. Within these two segments, visual stimulus S1 (the previous stimulus Pre-S1 with varying intensities; the post stimulus Pos-S1 with constant intensities) was presented within the RF, with each segment lasting 250ms. Responses to S1 presentation in the post segment (mean firing rates within black bar-defined epochs in Fig. 3B–E) were as shown in Fig. 3F.

To analyze the response dynamics, we compared PSTHs across three conditions: Pre-S1 > Pos-S1 (red data in Fig. 3C, E), Pre-S1 < Pos-S1 (blue data in Fig. 3C, E), and Only Pos-S1 (black data in Fig. 3C, E). In Fig. 3C and E, example Imc neurons showed the strongest post segment responses under Pre-S1 > Pos-S1 conditions, despite identical Pos-S1 sizes during this segment. Figure 3F illustrates the salience of Pos-S1 stimuli under varying Pre-S1 sizes. Results revealed a significant positive correlation between Pre-S1 stimulus size and the salience of Pos-S1 ($p < 0.05$, Pearson correlation test).

For population-level analysis of salience encoding dynamics in Imc neurons, we compared mean responses of Imc neurons recorded during the post segment under two conditions: Pre-S1 > S1 (strong Pre-S1) and Pre-S1 < S1 (weak Pre-S1). Population-level analysis revealed significantly stronger neural responses in the strong Pre-S1 condition compared to the weak condition ($p < 0.05$, Wilcoxon signed-rank tests). Separate analyses for size and contrast modulation are presented in Fig. 3G, H, respectively.

Our findings demonstrate that stimulus salience is determined not only by spatial, temporal salience and intrinsic physical intensity, but also by the physical intensities of spatiotemporally proximal preceding stimuli. This mechanism explains why spatiotemporally proximal preceding stimuli modulate neural representations of current stimulus competition—the representations of stimulus selection depend exclusively on the relative salience of stimuli. Notably, the neural responses in this study consistently exhibited persistent activity after stimulus cessation (e.g., Fig. S3). Whether such phenomenon reflect spatiotemporally proximal preceding stimuli modulate neural representations of current stimulus salience?

Sustained activity in Imc neuron after stimulus cessation

To analyze the properties of sustained activity in Imc neurons after stimulus cessation, we first defined key metrics. For each neuron, we set a threshold at 10% of the maximum firing rate (FR) across all target sizes. The response delay (delay time, indicated by the green region along the x-axis in Fig. 4B) was measured from stimulus onset to the FR reaching the threshold. The persistence time (indicated by the red region along the x-axis in Fig. 4B) quantified the duration from stimulus offset to the FR decaying below the threshold. We additionally analyzed the persistence intensity—defined as the mean FR during the 250ms after stimulus cessation.

Figure 4D–F display the delay time, persistence time, and persistence intensity of an example Imc neuron under varying stimulus sizes. The delay time showed a negative correlation with stimulus size ($c = -0.65$, $p = 0.04$, Pearson test). Conversely, persistence time exhibited a positive correlation with stimulus size ($c = 0.75$, $p = 0.01$). Persistence intensity also positively correlated with stimulus size ($c = 0.80$, $p = 0.005$).

Analysis of delay time, persistence time, and persistence intensity in the Imc neuron population revealed distinct correlations with stimulus size. Delay time showed negative correlation ($r = -0.74$, $p = 0.01$; Fig. 5G), while both persistence time ($r = 0.81$, $p = 0.003$; Fig. 5H) and persistence intensity ($r = 0.91$, $p < 0.001$; Fig. 5I) exhibited positive correlations.

Direct comparison at 3° stimulus size demonstrated significantly greater persistence time than delay time ($p < 0.001$, $n = 122$; Wilcoxon signed-rank test; Fig. 5C). These findings indicate that sustained poststimulus activity represents a distinct neural mechanism. This phenomenon extends beyond simple delays in neural transmission.

Imc neurons' persistence intensity positively correlates with target size. This aligns with the conclusion that stronger spatiotemporally proximal preceding stimuli enhances current stimulus salience. Thus, the sustained neural activity after stimulus cessation can be viewed as another manifestation of Imc neurons maintaining preceding stimulus memory. How is the sustained neural activity maintained after stimulus cessation?

lpc neurons modulate the response of Imc neurons

The hypothesis that bistable membrane properties and network feedback excitation sustain persistent neural responses has been proposed³⁸. Identification of an excitatory circuit in the midbrain network would hold

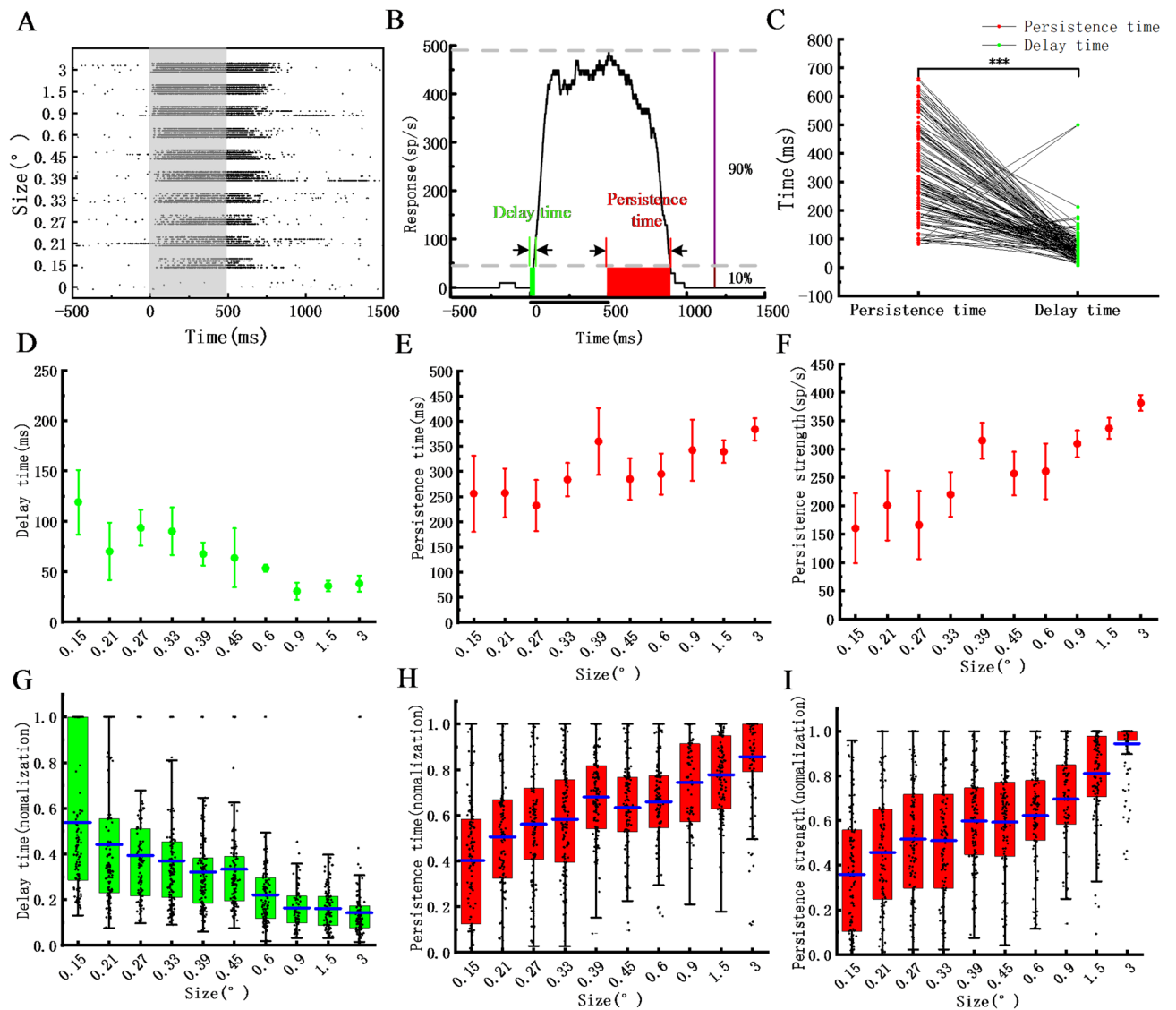


Fig. 4. Imc neurons' response dynamics; A, Raster plot of the example Imc neuron's responses to targets of different sizes. Shaded along the x-axis represents stimulus duration (500 ms); B, The definition of delay time and persistence time. The black curve is the PSTH of the Imc neurons' response, with stimuli presented at 0–500 ms. The bottom gray dashed line is the 10% of the maximum neural response (upper gray dashed line), it defines the dividing line at which the neural response begins and ends. The green interval is the delay time, the red interval is the persistence time; C, The contrast of the delay time and the persistence time of Imc neurons population. Size = 3°, $n = 122$, $P < 0.001$, Wilcoxon signed-rank tests; D, E, F, Statistics of delay time, persistence time, and persistence intensity of example Imc neurons to targets of different sizes. Correlation coefficient of delay time and persistence time versus size intensity = -0.65 ($p = 0.04$), 0.75 ($p = 0.01$), Pearson correlation test). Correlation coefficient of persistence intensity (means of FR 250ms after the stimulus cessation) versus size intensity = 0.8 ($p = 0.005$, Pearson correlation test); G, H, I, Statistics of delay time, persistence time, and persistence intensity of Imc neurons population to stimuli of different sizes, $n = 122$. Correlation coefficient of mean delay time and mean persistence time versus size intensity = -0.74 ($p = 0.01$), 0.91 ($p < 0.001$), Pearson correlation test. Correlation coefficient of mean persistence intensity versus size intensity = 0.91 ($p < 0.001$, Pearson correlation test).

significant implications. This could support the existence of continuous attractor networks (a candidate model for neural information representation³⁹) in midbrain circuitry⁴⁰.

In the midbrain network, both Imc and Ipc neurons receive excitatory inputs from Shc neurons. Ipc neurons send topographically organized projections to the OT, particularly targeting its superficial layers. Concurrently, Shc dendritic fields in these superficial layers integrate retinal afferents^{7,11,13}. We propose that Shc neurons reciprocally receive excitatory projections from Ipc neurons, thereby forming positive feedback loops (excitatory loops).

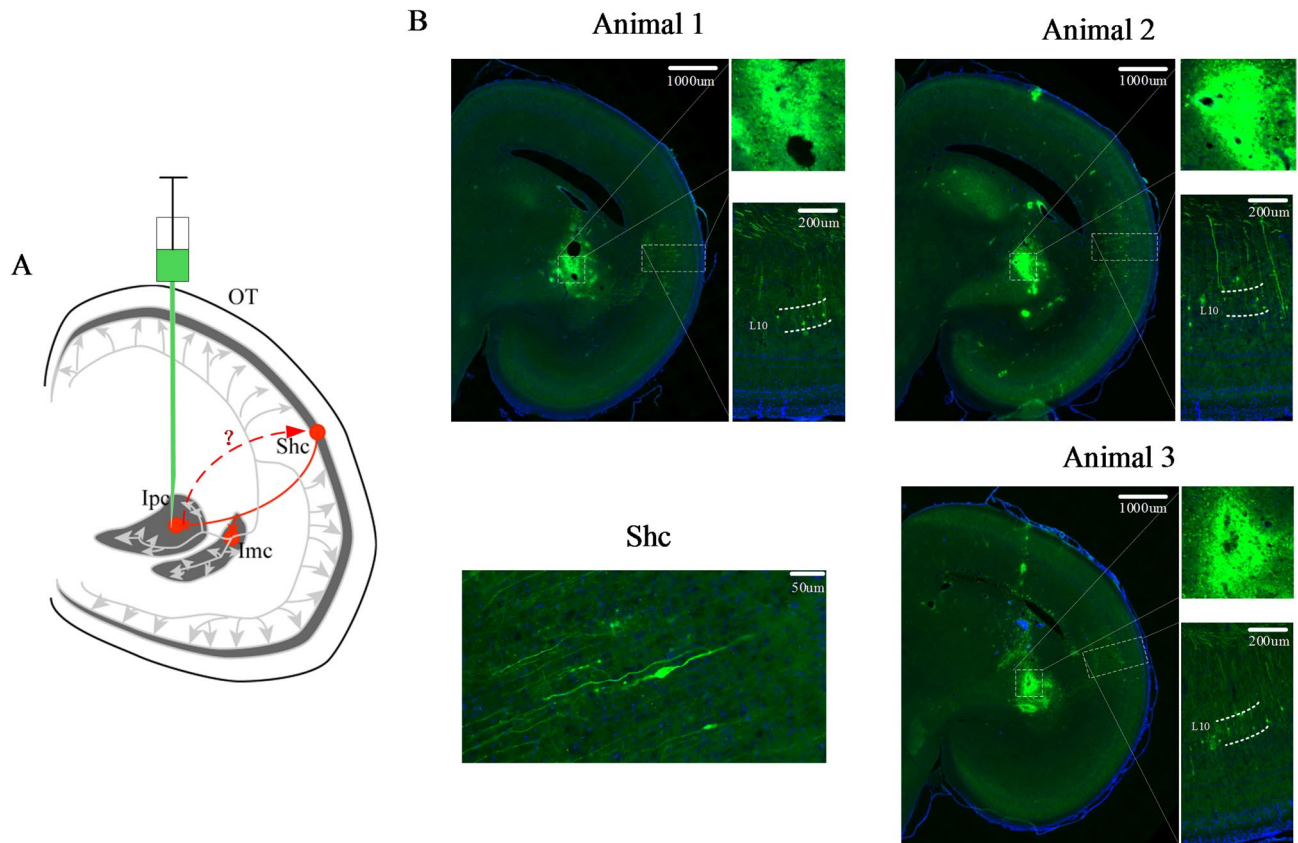


Fig. 5. In vivo injections of HSV-EGFP viral vectors in Ipc. **A**, Schematic diagram of the anterograde tracing, the red dotted line is the pathway to be verified; **B**, Brain slice scanning results of the anterograde tracer experiment. Experiments were conducted in three subjects, revealing robust fluorescence expression in OT layer 10 (L10). The lower-left panel shows a magnified view of Shc neurons within OT L10, whose cytoarchitecture matches documented morphological features.

To verify Ipc-to-Shc projections in the neural circuitry, we conducted anterograde tracing using HSV-EGFP viral vectors (“Materials and methods”). Viral injections (150 nl) targeted the Ipc (Fig. 5A), and the injection volume was 150nl. Brains were harvested for 3 days post-injection, sectioned, and imaged (Fig. 5B). In all three experimental subjects, fluorescence signals appeared at both the injection site and OT layer 10. The cytoarchitecture in OT L10 (Fig. 5B Shc) matched documented Shc neuron characteristics^{7,11,13}. These findings demonstrate excitatory Ipc-to-Shc projections, which may involve direct or indirect pathways. Notably, the precise topographic organization of Ipc-to-OT projections provides structural basis for excitatory loops between Ipc and Shc neurons with overlapping receptive fields.

In the midbrain network, Imc neurons exclusively receive excitatory inputs from Shc. To investigate whether Ipc-to-Shc (direct/indirect) projections modulate Imc responses, we performed Ipc inactivation experiments. Single-channel electrodes were implanted in Imc-Ipc neuron pairs with overlapping receptive fields (Fig. 6B). Neural responses of Imc neurons were recorded by presenting translational motion stimuli of various sizes at spatially aligned site-pairs in Imc and Ipc (Fig. 6C, black data, “Baseline”). Through glass electrodes, saline was microinjected into targeted Ipc regions (Fig. 6A). Imc neuronal responses were recorded starting 20 min post-injection (Fig. 6C, green data, “Saline”). Identical procedures were repeated using lidocaine for neural silencing (Fig. 6C, red data, “Lidocaine”). Experimental procedures are detailed in the “Materials and methods” section.

To assess Imc response modulation by Ipc inactivation, we employed three quantitative metrics: neural response intensity during stimulation (response intensity, Fig. 6D), persistence response intensity after stimulus cessation (persistence intensity, Fig. 6E), and persistence time after stimulus cessation (persistence time, Fig. 6F). As shown in Fig. 6D–F, example Imc neuron exhibited comparable response intensities between baseline and saline conditions. Lidocaine treatment, however, caused significant reductions in all three metrics.

To validate the phenomenon’s generalizability, we tested four pigeons across four experimental sessions. Differential responses (“Saline-Baseline” and “Lidocaine-Baseline”) were computed by subtracting baseline values from saline/lidocaine conditions. In Figs. 6G–I, each datapoint represents neural responses from individual neurons under specific stimulus sizes. Saline condition showed no significant differences from baseline in response intensity ($p=0.27$), persistence intensity ($p=0.25$), or persistence duration ($p=0.20$; one-sample Wilcoxon signed-rank test). Lidocaine-induced response reductions significantly exceeded saline effects across all parameters (response intensity, $P<0.001$; persistence intensity, $P<0.001$; persistence time, $P<0.001$;

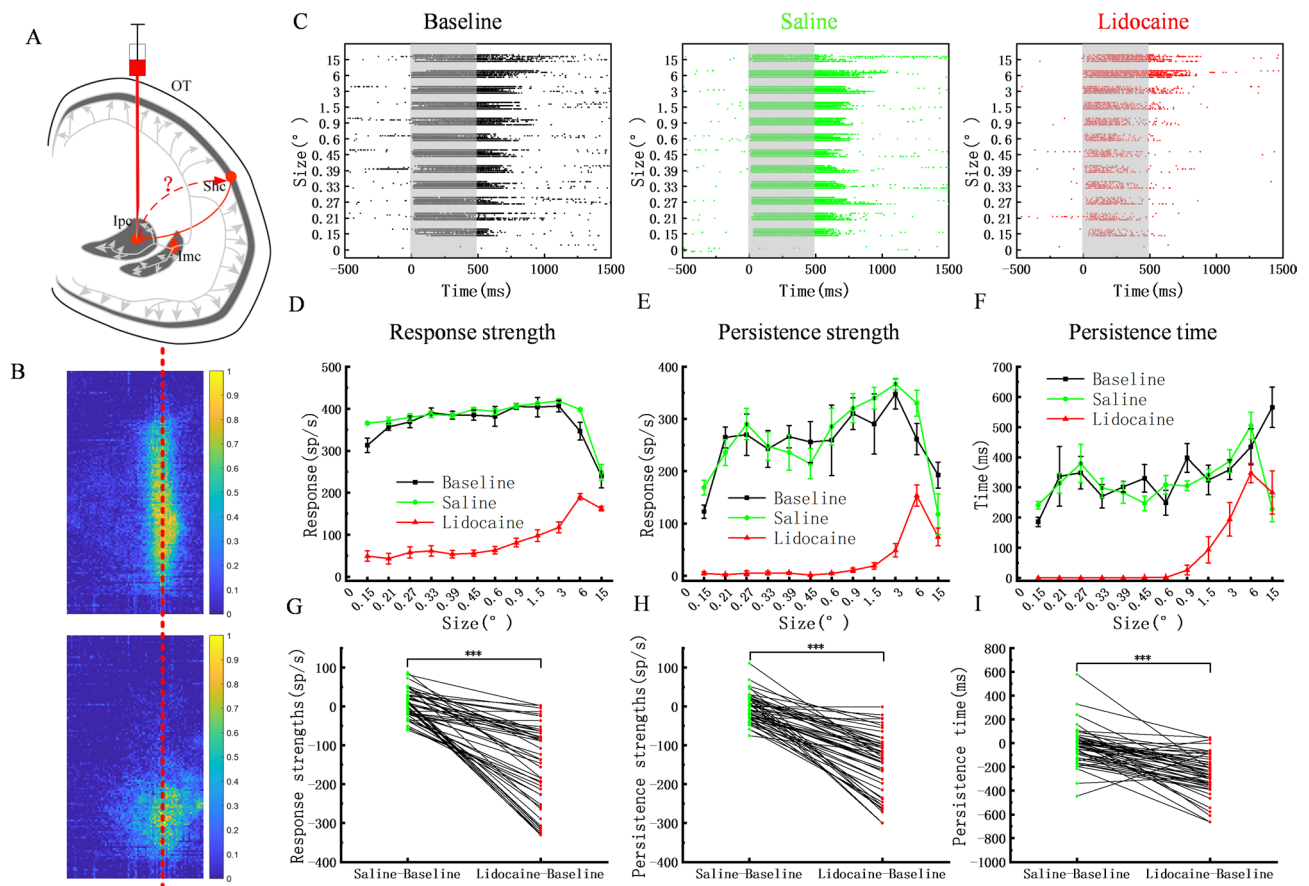


Fig. 6. Ipc inactivation effects on Imc neurons with overlapping receptive fields; A, Midbrain circuit diagram: red arrows denote excitatory projections, gray arrows inhibitory connections; B, Receptive field mapping: upper/lower panels show Imc/Ipc neuron receptive field locations on visual display; C, Raster plots of an exemplar Imc neuron under three conditions (left to right): pre-inactivation baseline, Ipc saline injection, and Ipc lidocaine administration. Stimulus duration (500 ms) marked by gray shading; D, E, F, Quantitative metrics for exemplar neurons: response intensity (D), persistence intensity (E), and persistence duration (F). Data points colored black (baseline), green (saline), red (lidocaine); G, H, I, Population-level comparisons: lidocaine-induced reductions in response intensity (G, $p < 0.001$), persistence intensity (H, $p < 0.001$), and persistence duration (I, $p < 0.001$) versus saline controls (Wilcoxon signed-rank tests).

Wilcoxon signed-rank tests). These data demonstrate Ipc's essential role in modulating both response magnitude and sustained activity dynamics in Imc.

Computational modeling serves as a pivotal tool for investigating neural mechanisms. To examine how Shc-Ipc (direct/indirect) projections modulate Imc neurons, we developed a midbrain network model ("Materials and methods"). In this model, Shc neurons receive direct excitatory inputs from Ipc neurons. We simulated Imc responses to retinal ganglion cell (RGC) inputs with varying intensities. Under Shc-Ipc-Shc loop conditions (Fig. 7, black traces), three metrics increased with stronger RGC inputs: response intensity (Fig. 7A), persistence intensity (Fig. 7B), and persistence time (Fig. 7C). Without loop, all parameters showed decreased (vs. loop condition). The model's output aligns with experimental data from Imc neurons. This confirms that the Shc-Ipc-Shc excitatory loop modulates both Imc neuronal responses and the maintenance of neural activity after stimulus cessation.

Collectively, this study reveals direct or indirect excitatory projections from Ipc to Shc neurons. These projections enhance Imc neuronal responses and facilitate the sustained activity in Imc neurons after stimulus cessation.

Discussion

This study employed spatiotemporally continuous two-segment visual motion stimuli to simulate dynamically changing visual scenes encountered by animals, investigating dynamic representations of stimulus selection in Imc neurons within midbrain networks. Using combined CRI-SCP and CISP protocols, we first demonstrated that Imc neuronal stimulus selection shifts from dependence on relative physical intensity to relative stimulus salience (Conclusion 1). Under VRI-SCP protocols, Imc neurons exhibited spatiotemporally proximal preceding stimuli modulated neural representations of current stimulus competition (Phenomenon 1). Integrating Conclusion 1 and Phenomenon 1, we hypothesized the existence of spatiotemporally proximal preceding stimuli

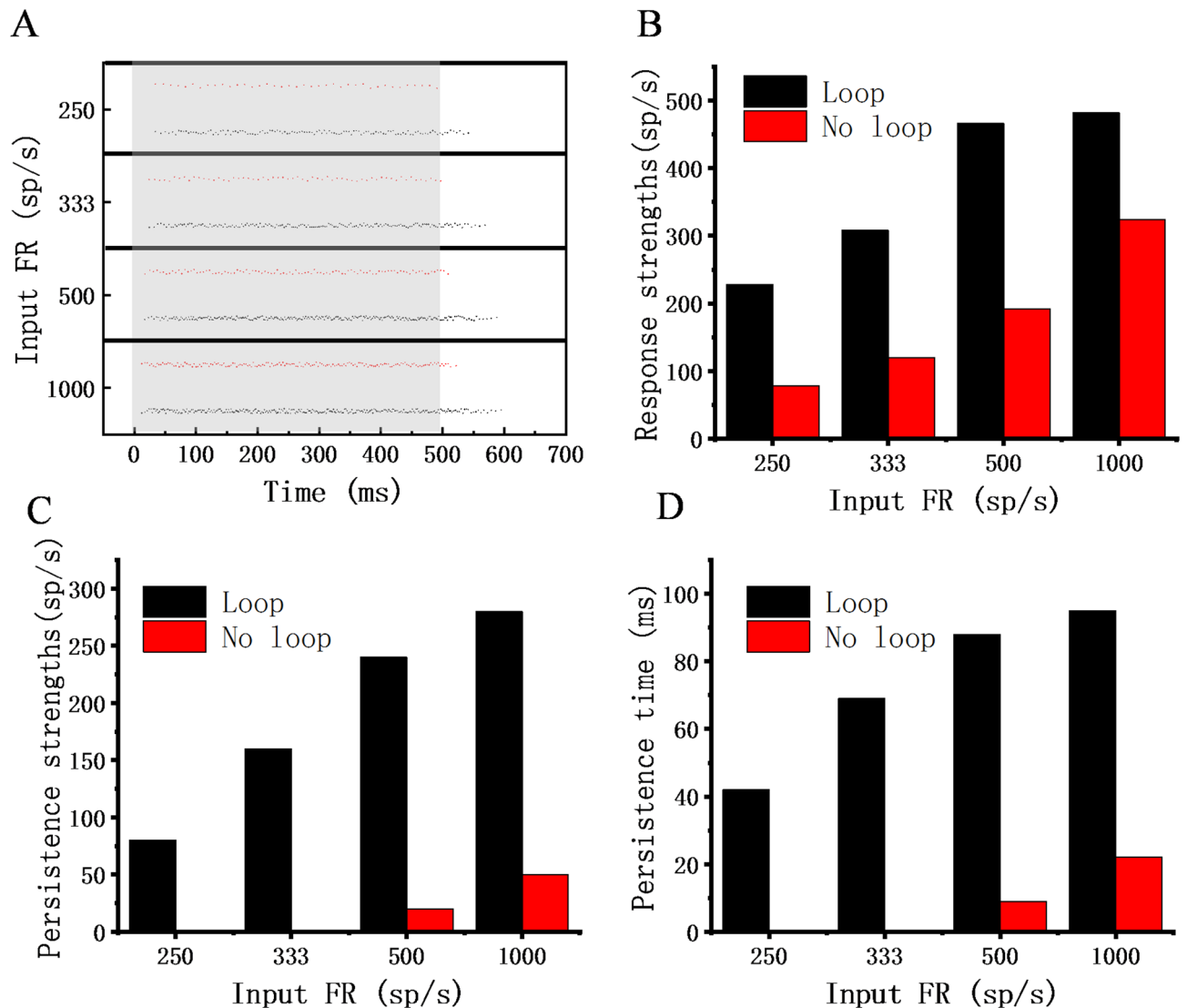


Fig. 7. Response of model Imc neurons with/without SC-Ipc-SC excitatory loops; A, Raster plots of model Imc activity under varying Shc input intensities (250–1000 spikes/s). Black/red data indicate loop presence/absence; B, C, D, Quantitative metrics across input frequencies (250–1000 spikes/s): response intensity (B), persistence intensity (C), and persistence time (D). Black/red data points represent loop-present/absent conditions respectively.

modulated neural representations of current stimulus salience (Phenomenon 2), subsequently validating this hypothesis via VISP protocols. Experiments revealed sustained neural activity in Imc neurons after stimulus cessation, with persistence time and intensity positively correlated to stimulus strength. Therefore, we propose this persistent neural activity and Phenomenon 2 share common neural mechanisms. Further investigation identified excitatory projections (direct/indirect) from Ipc to Shc neurons, which enhanced both Imc response magnitude and persistent neural activity after stimulus cessation. These findings, combined with midbrain connectivity patterns, suggest two functional circuits: an excitatory loop (Shc-Ipc-Shc) and an inhibitory loop (Imc-Ipc), collectively supporting continuous attractor networks in midbrain visual processing.

What factors influence the salience representations of Imc neurons? Without top-down modulation, the midbrain network selects stimuli based on salience^{1,16}. Avian optic tectum (OT) is widely regarded as encoding saliency maps^{41,42}, with Imc neurons receiving exclusive excitatory inputs from OT's Shc neurons^{11,13,27}. Our previous studies demonstrated that Imc neurons can encode both temporal- and spatial-domain salience^{28,29}, supporting downstream inheritance of salience signals from upstream circuits. Building on these results, we propose that the salience representations of Imc neurons depend not only on spatiotemporal stimulus uniqueness but also on two additional factors: physical intensity of stimulus (e.g., moving target size, contrast, approach speed) and the intensity of spatiotemporally proximal preceding stimuli.

What type of memory do Imc neurons exhibit for spatiotemporally proximal preceding stimuli? Atkinson and Shiffrin classified memory into sensory, short-term, and long-term stores⁴³. Incoming sensory information first enters the sensory memory, where it resides for a very brief period, then decays and is lost. The short-

term memory is the subject's working memory, it receives selected inputs from the sensory memory and also from long-term memory. The long-term store is a permanent repository for information transferred from the short-term memory. In Imc neurons, the modulation of current stimulus salience by spatiotemporally proximal preceding stimuli manifests as persistent neural activity after stimulus cessation (< 1s duration), indicating non-long-term memory mechanisms. Sensory-to-working memory transitions require selective attention—a process critically mediated by midbrain networks. Collectively, we propose that Imc neurons encode sensory memories of spatiotemporally proximal historical stimuli. This sensory memory likely helps animals sustain attention to stimuli over brief periods, effectively resisting noise interference to support higher cognitive functions.

Excitatory loops in the midbrain network. Excitatory loops in the midbrain network were hypothesized in earlier studies. Luksch et al.³⁷ modeled a Shc-Ipc-Shc excitatory loop to simulate oscillatory burst responses in Ipc neurons. Marin et al.⁴⁴ explained repetitive bursting waves in the optic tectum (OT) through this loop mechanism. Although these studies used Shc-Ipc-Shc loops to explain neural phenomena, direct anatomical evidence is still lacking. While our study cannot confirm direct Ipc-to-Shc projections, it demonstrates at least indirect connectivity. Furthermore, Marin et al.¹³ identified Shc neurons projecting to both Imc and Ipc. Combined with evidence that Ipc inactivation attenuates Imc responses and Ipc-OT projections exhibit topographic organization, we propose a functional Shc-Ipc-Shc excitatory loop, where Ipc-to-Shc connectivity may be direct or indirect.

The avian midbrain network contains attractor networks. An attractor network is a recurrent neural circuit whose dynamics drive network states toward stable equilibria⁴⁵, supporting cortical functions like memory, attention, and decision-making^{46,47}. The discovery of head direction circuits as ring attractor networks provides the most direct evidence for biological attractor dynamics^{48,49}. Such networks require two core components: (1) strong recurrent excitation to sustain persistent post-stimulus activity, and (2) lateral inhibition to prevent runaway excitation^{50,51}. In the midbrain, the excitatory Shc-Ipc-Shc loop and inhibitory Imc recurrent connections establish this structural framework. Sustained Imc activity after stimulus cessation further confirms attractor-like dynamics. These findings reveal previously unexplored neurodynamic mechanisms in avian midbrain function.

Data availability

The data that support the findings of this study are available on request from the corresponding author. email: wangsongwei_zzu@163.com.

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Author contributions

LLQ: Conceptualization, Data curation, Software, Investigation, Methodology, Writing – original draft. CCJ: Investigation, Methodology, Validation. JTW: Software, Validation. ZZW: Validation, Project administration. LS: Validation, Supervision. SWW: Conceptualization, Data curation, Visualization, Funding acquisition, Formal analysis. All authors read and approved the final manuscript.

Competing interests

The authors declare no competing interests.

Additional information

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