

Fig. S1 Microelectrode arrays packaged with the printed circuit board.

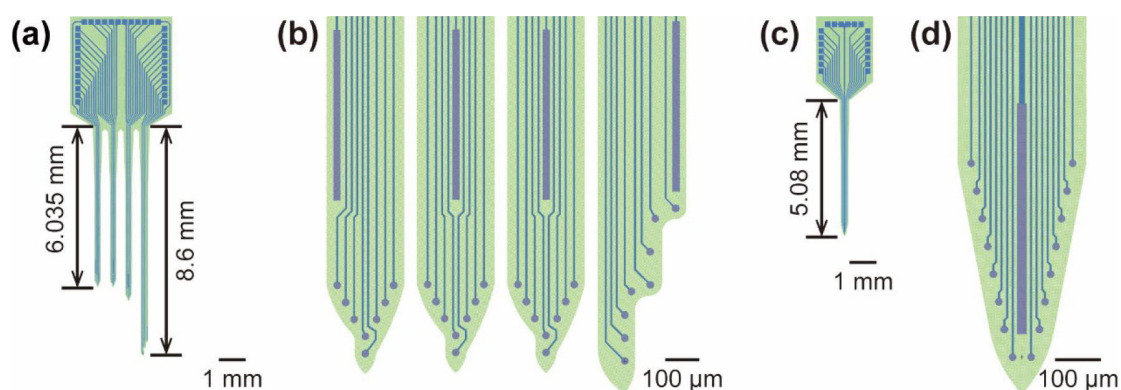


Fig. S2 Microelectrode shape design. (a) Microelectrode design drawing for the striatum. (b) Enlarged view of the microelectrode end for the striatum. On the shank of each electrode, purple circles represent detection sites, purple rectangles represent ground electrodes, and blue thin lines represent wires. (c) Microelectrode design drawing for the hippocampal CA1 region. (d) Enlarged view of the microelectrode end for the hippocampal CA1 region. CA1: cornu ammonis 1.

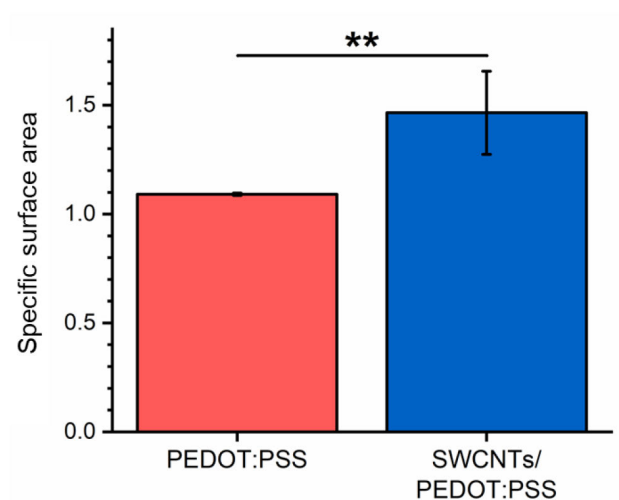


Fig. S3 Average specific surface area of electrode sites modified with PEDOT:PSS and SWCNTs/PEDOT:PSS. SWCNT: single-walled carbon nanotube; PEDOT:PSS: poly(3,4-ethylenedioxythiophene):poly(4-styrenesulfonate). Data are expressed as mean ± standard error of the mean (SEM), $n=5$. ** $P<0.01$.

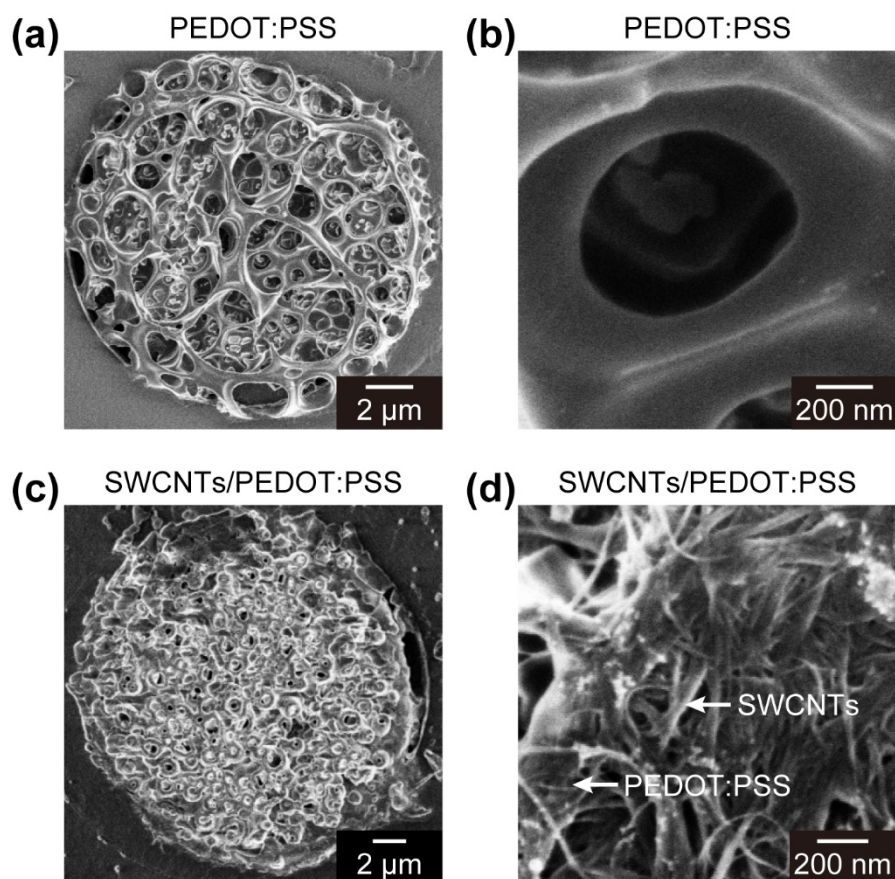


Fig. S4 Scanning electron microscopy images of electrode sites modified by nanomaterials. (a) PEDOT:PSS modified electrode sites at the micrometer scale. (b) PEDOT:PSS-modified electrode sites at the nanometer scale. (c) SWCNTs/PEDOT:PSS-modified electrode sites at the micrometer scale. (d) SWCNTs/PEDOT:PSS-modified electrode sites at the nanometer scale. SWCNT: single-walled carbon nanotube; PEDOT:PSS: poly(3,4-ethylenedioxythiophene):poly(4-styrenesulfonate).

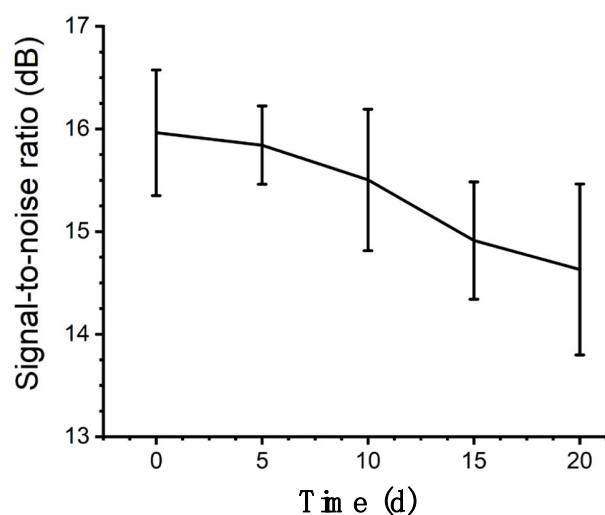


Fig. S5 Variation of neural signal-to-noise ratio with days after electrode implantation.

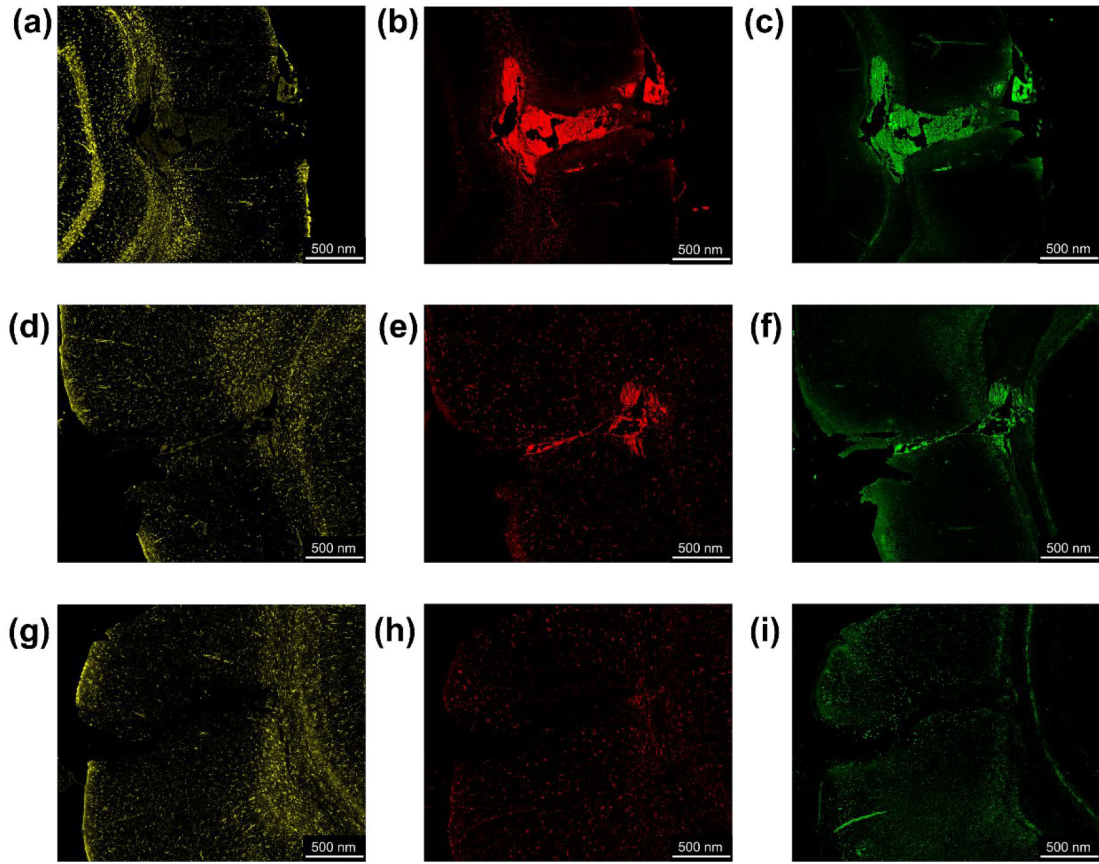


Fig. S6 Immunofluorescence staining of MEA, microwire array, and tungsten electrode in the hippocampal region. (a–c) Immunofluorescence staining of GFAP (a), IBA-1 (b), and NeuN (c) after microwire array implantation and explantation. (d–f) Immunofluorescence staining of GFAP (d), IBA-1 (e), and NeuN (f) after tungsten electrode implantation and explantation. (g–i) Immunofluorescence staining of GFAP (g), IBA-1 (f), and NeuN (i) after MEA implantation and explantation. GFAP: glial fibrillary acidic protein; IBA-1: ionized calcium-binding adapter molecule; NeuN: neuron-specific nuclear protein; MEA: microelectrode array.

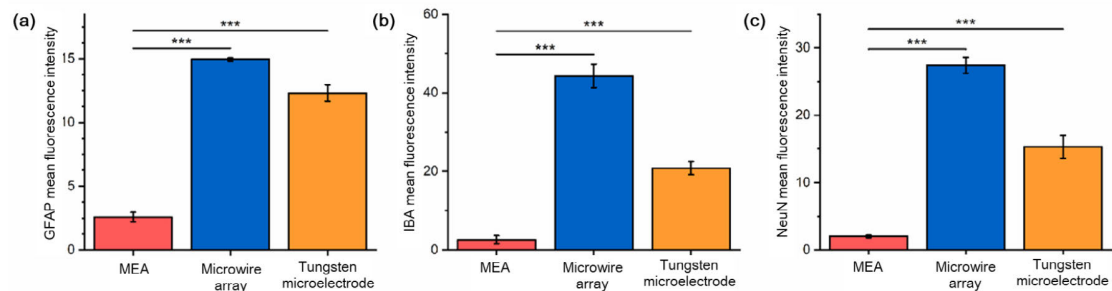


Fig. S7 Comparison of average fluorescence intensity after implantation of GFAP (a), IBA-1 (b), and NeuN (c). Data are expressed as mean±standard error of the mean (SEM), $n=6$. *** $P<0.001$. GFAP: glial fibrillary acidic protein; IBA-1: ionized calcium-binding adapter molecule; NeuN: neuron-specific nuclear protein; MEA: microelectrode array.

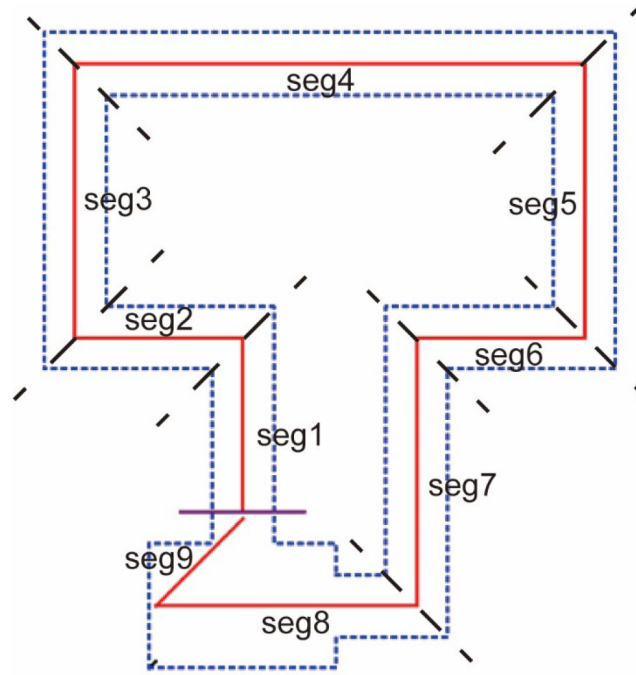


Fig. S8 Schematic diagram of converting two-dimensional positions to linear positions. The blue dotted line represents the outline of the maze. The red line represents the centerline of the maze used for linearization, and the purple line represents the starting point of the linearized trajectory. According to the shape of the maze, the linearized trajectory is divided into nine segments separated by black dotted lines. The two-dimensional trajectory is projected onto the linearized trajectory, and the distance from the projection point to the starting point is defined as the linearized trajectory. seg: segment.

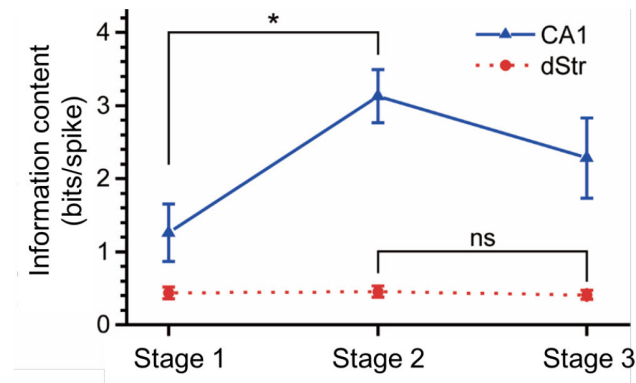


Fig. S9 Curves of spatial information content changes in two brain regions under three stages. The information content of the hippocampus increases rapidly with the emergence of new goal positions and decreases with the disappearance of rewards at the goal positions, while the spatial information content of the striatum does not show obvious changes. Data are expressed as mean±standard deviation (SD), $n=9$ for CA1 and $n=5$ for dStr. * $P<0.05$. ns: not significant. CA1: cornu ammonis 1; dStr: dorsal striatum.

Materials and methods

Fabrication of the microelectrode arrays (MEAs): the MEAs used for striatum (Str) have a total of four shanks and 32 sites. The diameter of each site is 22 μm , and the longest shank is 8.6 mm. The MEAs used for cornu ammonis 1 (CA1) have a total of 16 sites, with a length of 5 mm and a site diameter of 16 μm . The MEAs are fabricated based on silicon-on-insulator (SOI; 25 μm Si/1 μm SiO₂/350 μm Si) wafers. After thermally oxidizing 500 nm of silicon dioxide as the base insulating layer, a layer of photoresist is spin-coated. Then, 30 nm of Cr and 250 nm of Pt are sputtered as the metal layer. After lift-off, recording sites and wires are obtained. Subsequently, 300 nm of silicon oxide and 500 nm of silicon nitride films are deposited by plasma enhanced chemical vapor deposition (PECVD) as the top-layer insulating layer. Then, photolithography is performed again, and reactive ion etching (RIE) is used for etching to expose the electrode sites and connection pads. After that, photolithography is carried out once more, and deep silicon etching is used to form the electrode morphology. Then, wet etching (80 °C, 30% [mass fraction] KOH) is used to etch the bottom silicon layer (350 μm Si) of the SOI to obtain individual electrodes. Finally, the residual stress of the electrode is released by back-side dry etching.

Surface modification of electrode sites: after the MEAs were fabricated, the electrodes are packaged onto printed circuit board (PCB) through wire bonding (Fig. S1). Then, the following steps were performed to modify the electrode sites with single-walled carbon nanotubes (SWCNTs)/poly(3,4-ethylenedioxythiophene):poly(4-styrenesulfonate) (PEDOT:PSS) nanocomposite materials: 113.774 mg 3,4-ethylenedioxythiophene (EDOT) (Aladdin, Shanghai, China), 824.776 mg of PSS (Herochem, Shanghai, China), and 40 mg of SWCNTs (XFNANO, Nanjing, China) were dissolved in deionized water (20 mL). The mixture was then sonicated in a cold water bath for 30 min. After that, the shanks of the MEAs were washed with deionized water. Finally, the SWCNTs/PEDOT:PSS composite material was electroplated on the electrode surface using cyclic voltammetry. The voltage of the working electrode ranged from 0 to 0.95 V, with a rate of 0.10 V/s. The reference electrode was Ag/AgCl and the counter electrode was platinum wire.

Animals and surgery: a total of three male Sprague-Dawley rats (270–330 g, 9–13 weeks, Vital River) were used for the experiment. The rats were kept in a cage with a stable and suitable temperature (about 25 °C) and humidity (about 50%), maintaining a 12-h light/dark cycle. During the feeding period, the rats had access to sufficient food and water. Afterward, food was restricted to maintain 85% of their body weight.

The process of implanting two MEAs into the rat's brain was as follows. The rat was first anesthetized with 5% (volume fraction) isoflurane. Then, it was fixed on the stereotactic apparatus, and the ear bars were adjusted to keep the rat's brain horizontal. After that, the concentration of isoflurane was reduced to about 1.5% (volume fraction). A heating pad was placed under the rat's body to maintain its body temperature. The hair on the rat's head was shaved off. After cleaning, by removing a small section of the scalp, the underlying skull was exposed. Five screws were fixed around the skull. Two windows with sizes of 4 mm×2 mm and 2 mm×2 mm were drilled on the surface of the skull in the left hemisphere of the brain. An MEA was implanted into the Str using the longest shank as the positioning point, with the coordinates of anterior–posterior (AP): 1.8 mm, medial–lateral (ML): 1.4 mm, and dorsal–ventral (DV): 6.6 mm. Then, another electrode was implanted into the CA1 (with the coordinates of AP: 3.6 mm, ML: 2.4 mm, and DV: 2.2 mm from the dura level). The implantation speed was maintained at 1 $\mu\text{m/s}$. After the implantation into the specified position, a metal wire was used to connect the ground of the MEAs to the screws. The windows opened for implantation were filled with medical petrolatum. Then, dental cement was used to cover the skull and the electrodes to isolate them from the external environment and provide sufficient mechanical strength.

Post-surgery, rats received an intraperitoneal injection of 40000 units of penicillin.

Behavioral experimental procedure: the rats were first trained in the maze (Fig. 3c), made of black polyvinyl chloride (PVC) boards. The size of the maze was 102 cm×92 cm, and the path was 10 cm wide. During the training process, every time the rat reached Goal 2, a piece of peanut was delivered as a food reward. The forward direction of the rats was fixed. When a rat returned to the red area in Fig. 3c, the right side of the red area was blocked by a PVC board to prevent the rat from moving in the reverse direction (Fig. 3d). When the rat left the red area, the baffle was removed. In the early stage of training, the rats were trained for 10 trials/d (starting from the red area and returning to the red area along the path). In the later stage, the rats underwent at least 40 trials daily for training. Error trials were defined as three scenarios: (1) the rat initiated movement from the red area along the preset forward direction (Fig. 3) but shifted to the opposite direction during locomotion; (2) the rat directly initiated movement from the red area along the preset incorrect direction; (3) the rat initiated movement from the red area along the preset forward direction but failed to return to the red area eventually. After two weeks, the rats learned this experimental paradigm, and the number of error trials (returning to the red area in the wrong direction) was less than 10%. After the training was completed, an MEA implantation surgery was performed on the rats. After the surgery, the rats were allowed to rest for at least 5 d, during which they had access to sufficient food and water. After that, the rats were trained for one more days, and then a behavioral experiment was carried out.

Electrophysiological signal recording: the two MEAs were respectively connected to 32-channel and 16-channel headstages (used for preliminary amplification and filtering of the analog signals on the electrodes nearby) and then connected to the neural signal recording instrument through a 2-m-long wire. The sampling frequency was set at 20 kHz.

Video recording and trajectory extraction: while recording the electrophysiological signals, a camera placed above the maze synchronously collected the movement trajectory of the rats at a sampling frequency of 30 Hz. After the experiment, the movement information, such as the trajectory and speed of the rats, was identified through the light-emitting diode (LED) lights fixed on the headstage (EthoVision XT 16, Noldus, China).

Movement event determination: first, the acceleration at each moment was calculated based on the rat's movement speed, and the absolute value of the acceleration was taken for analysis and display. An acceleration of 10 cm/s² was used as the threshold to extract the start time of the rat's movement. In addition, the rat should remain stationary for at least 10 s before the start of movement. The rat should maintain a moving state for at least 10 s after the start of movement.

Criteria for identifying neurons modulated by movement initiation: the neuronal activities within 20 s before and after the start of movement were divided into 100-ms bins. Then, the average firing rates of neurons across movement events were calculated to construct a peri-event time histogram (PETH). Distributions of the PETH from -10 to -5 s before movement onset were used as baseline activity. Then the neuronal firing rate during the time period from -5 to 10 s was examined. If there were at least eight consecutive bins before the start of movement that exceeded or fell below 2.58 times the standard deviation of the baseline activity (99% confidence interval) (da Silva et al., 2018), the neuron was considered to be regulated by movement initialization.

Generation of place firing rate maps: the nearest position at the time of spike firing was found by matching the firing times of spikes with the movement trajectory. To reduce the influence of rats being stationary and stopping to eat food, only the spikes and trajectories with a speed greater than 6 cm/s were analyzed. The place firing rate map was constructed using a 2 cm×2 cm bin. First, the time the rat stayed in each bin was counted. After performing

Gaussian smoothing, the distribution of the rat's positions was obtained. Similarly, the smoothed distribution of spike positions was calculated. The place firing rate map was obtained by dividing the spike distribution by the position distribution matrix.

Calculation of spatial information content: the spatial information content (SIC, S_{IC}) was calculated using the formula:

$$S_{IC} = \sum_i P_i \lambda_i \log_2 \lambda_i \quad (S1)$$

where λ_i refers to the firing rate of bin i divided by the mean firing rate and P_i represents the ratio of the time spent in bin i to the total time. The spatial information content was calculated for each stage.

Criteria for determining place fields and place-related firing: given the narrow maze passages (only 10 cm wide), using two-dimensional (2D) position information would introduce unnecessary interference. Therefore, the 2D spatial positions were converted into linear (1D) positions (Fig. S8). Then, the linear firing rate map was calculated with a 2-cm bin and used to determine the spatial firing characteristics. The spatial firing patterns were analyzed at different stages. If the spatial firing patterns in any stage met the following conditions: (1) the maximum firing rate exceeds 2 Hz and the mean firing rate exceeds 0.1 Hz; (2) at least five consecutive bins near the peak firing rate are more than 30% of it, it was considered position-related firing. Based on this, if the spatial information content was greater than 1, the consecutive bins were considered a place field, and the neuron was regarded as a place cell.

Statistical analysis: all statistical analyses were conducted using Offline Sorter (Plexon, Dallas, TX, USA), Python (Version 3.9), and ORIGIN software (OriginLab, Northampton, MA, USA). Statistical significance was assessed using one-way analysis of variance (ANOVA), with Tukey's post-hoc test applied for multiple comparisons. Unless otherwise stated, the data are presented as the mean±standard error of the mean (SEM). The significance levels are: * $P<0.05$, ** $P<0.01$, and *** $P<0.001$.

References

da Silva JA, Tecuapetla F, Paixão V, et al., 2018. Dopamine neuron activity before action initiation gates and invigorates future movements. *Nature*, 554:244-248.
<https://doi.org/10.1038/nature25457>