

# Supplementary Methods

## 1 Single neuron model

Each neuron is simulated by the leaky integrate-and-fire model. The membrane potential  $V(t)$  obeys the following equation:

$$C_m \frac{dV(t)}{dt} = -g_L(V(t) - V_L) - I_{syn}(t),$$

where  $C_m$  is the capacitance,  $g_L$  is the leak conductance,  $V_L$  is the resting potential and  $I_{syn}$  is the total synaptic current in the cell.

When the membrane potential  $V(t)$  of each neuron reaches a threshold  $V_{threshold} = -50\text{mV}$ , a spike is emitted and  $V(t)$  is set to the reset potential  $V_{reset} = -55\text{mV}$  for a refractory period  $T_r = 2\text{ms}$ . For inhibitory neurons in the superior colliculus and the cortical network models, we used the following parameters:  $C_m = 0.2\text{nF}$ ,  $g_L = 10\text{nS}$  and  $V_L = -70\text{mV}$ . For all other neurons in the model, we used  $C_m = 0.5\text{nF}$ ,  $g_L = 20\text{nS}$  and  $V_L = -70\text{mV}$ .

The synaptic current  $I_{syn}(t)$  is described by:

$$I_{syn}(t) = g_{AMPA}s_{AMPA}(t)(V(t) - V_E) + \frac{g_{NMDA}s_{NMDA}(t)(V(t) - V_E)}{1 + [Mg^{2+}]e^{-0.062V(t)}/3.57} + g_{GABA}s_{GABA}(t)(V(t) - V_E),$$

where  $V_E (=0)$  is the reversal potential,  $[Mg^{2+}] (=1.0\text{mM})$  is the extracellular magnesium concentration and  $g$  is the synaptic efficacy. The gating variable  $s$  obeys

$$\frac{ds(t)}{dt} = \sum_k \delta(t - t^k) - \frac{s}{\tau}$$

for AMPA and GABA<sub>A</sub> receptor mediated currents and

$$\frac{ds(t)}{dt} = \alpha(1 - s(t)) \sum_k \delta(t - t^k) - \frac{s}{\tau}$$

for NMDA receptor mediated current, with  $\alpha = 0.63$ . The decay constant  $\tau$  is 2ms for AMPA, 100ms for NMDA and 5ms for GABA<sub>A</sub>.  $\delta(t - t^k)$  is the delta function and  $t^k$  is the time of the  $k$ th presynaptic spike.

## 2 Short-term facilitation

We implemented short-term facilitation (STF) at the SCe-to-SCi synapses in the superior colliculus. The gating variable  $s$  is multiplied by the STF factor  $F$ , which obeys the following dynamics [1]:

$$\frac{dF}{dt} = \alpha_F(1 - F) \sum_k \delta(t - t_k) - F/\tau_F,$$

where the dimensionless factor  $\alpha_F$  equals 0.15 and the decay constant  $\tau_F$  equals 1000 ms.

### 3 Synaptic connections and background inputs

All synaptic connections between neural populations or within a neural population are all-to-all. The values of synaptic efficacy  $g$ , unless otherwise specified, are:

for SC,

$$\begin{aligned} g_{NMDA}^{SCe^R-SCe^R} &= g_{NMDA}^{SCe^L-SCe^L} = 1.5, & g_{NMDA}^{SCe^R-SCi} &= g_{NMDA}^{SCe^L-SCi} = 0.7, \\ g_{NMDA}^{SCe^R-Cxi} &= g_{NMDA}^{SCe^L-Cxi} = 0.11, & g_{NMDA}^{SCe^R-Cxe^R} &= g_{NMDA}^{SCe^L-Cxe^L} = 0.05, \\ g_{GABA}^{SCi-SCe^R} &= g_{GABA}^{SCi-SCe^L} = 2.5, \end{aligned}$$

for BG,

$$g_{GABA}^{SNr^R-SCe^R} = g_{GABA}^{SNr^L-SCe^L} = 2.5, \quad g_{GABA}^{CD^R-SNr^R} = g_{GABA}^{CD^L-SNr^L} = 0.6,$$

and for Cx,

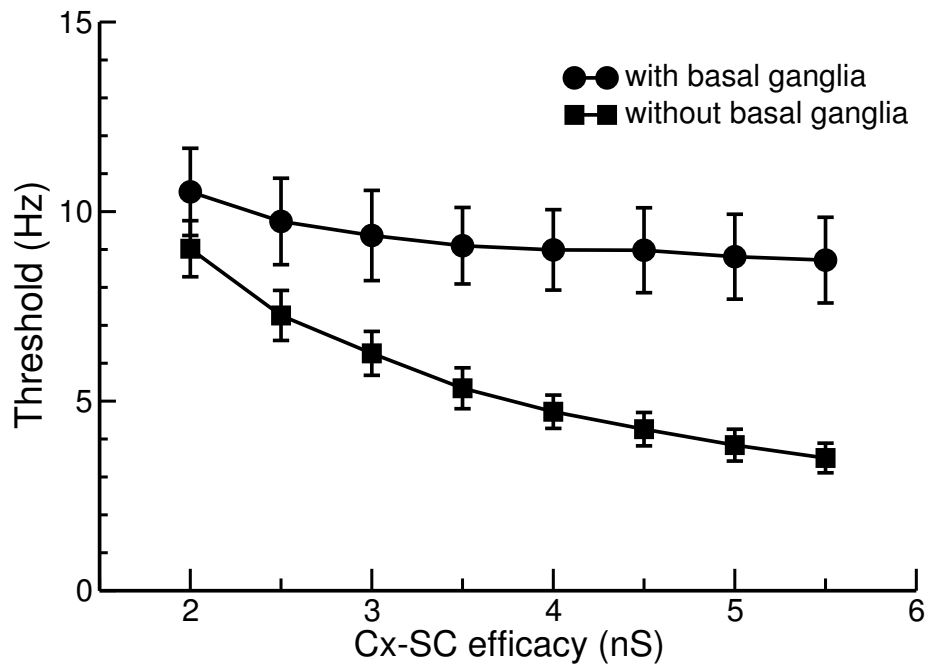
$$\begin{aligned} g_{AMPA}^{Cxe^R-SCe^R} &= g_{AMPA}^{Cxe^L-SCe^L} = 3.5, & g_{AMPA}^{Cxe^R-CD^R} &= g_{AMPA}^{Cxe^L-CD^L} = 0.8 - 4.6, \\ g_{AMPA}^{Cxe^R-Cxe^R} &= g_{AMPA}^{Cxe^L-Cxe^L} = 0.085, & g_{NMDA}^{Cxe^R-Cxe^R} &= g_{NMDA}^{Cxe^L-Cxe^L} = 0.2805, \\ g_{AMPA}^{Cxe^R-Cxe^L} &= g_{AMPA}^{Cxe^L-Cxe^R} = 0.043825, & g_{NMDA}^{Cxe^R-Cxe^L} &= g_{NMDA}^{Cxe^L-Cxe^R} = 0.14462, \\ g_{AMPA}^{Cxe^R-Cxi} &= g_{AMPA}^{Cxe^L-Cxi} = 0.04, & g_{NMDA}^{Cxe^R-Cxi} &= g_{NMDA}^{Cxe^L-Cxi} = 0.13, \\ g_{GABA}^{Cxi-Cxe^R} &= g_{GABA}^{Cxi-Cxe^L} = 1.3, & g_{GABA}^{Cxi-Cxi} &= 1.0. \end{aligned}$$

All values are in nS. Superscripts denote the presynaptic and postsynaptic populations. For example,  $SCe^R-SCi$  represents  $SCe^R$ -to- $SCi$  connection, and  $SCe^R-SCe^R$  represents the recurrent connection of  $SCe^R$ .

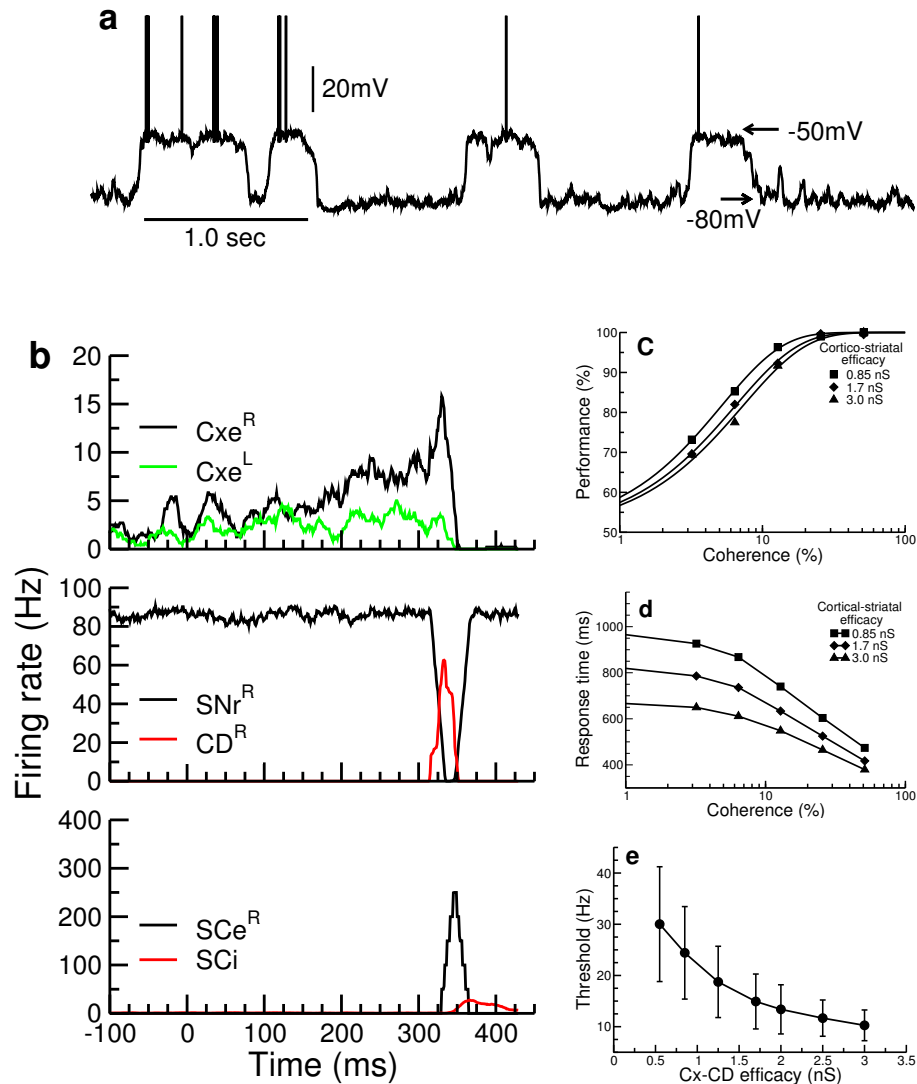
All neurons receive background Poisson inputs with mean conductance equal to 0.4864 nS for SCe neurons, 5.12 nS for SCi neurons, 13.76 nS for SNr neurons and 1.6 nS for CD neurons. We follow Ref. [2] for all the parameters, including numbers of neurons, values of synaptic strength and background Poisson inputs in the Cx network.

### References

- [1] Hempel, C. M., Hartman, K. H., Wang, X.-J., Turrigiano, G. G. & Nelson, S. B. Multiple forms of short-term plasticity at excitatory synapses in rat medial prefrontal cortex. *J. Neurophysiol.* **83**, 3031–3041 (2000).
- [2] Wang, X.-J. Probabilistic decision making by slow reverberation in cortical circuits. *Neuron* **36**, 955–968 (2002).



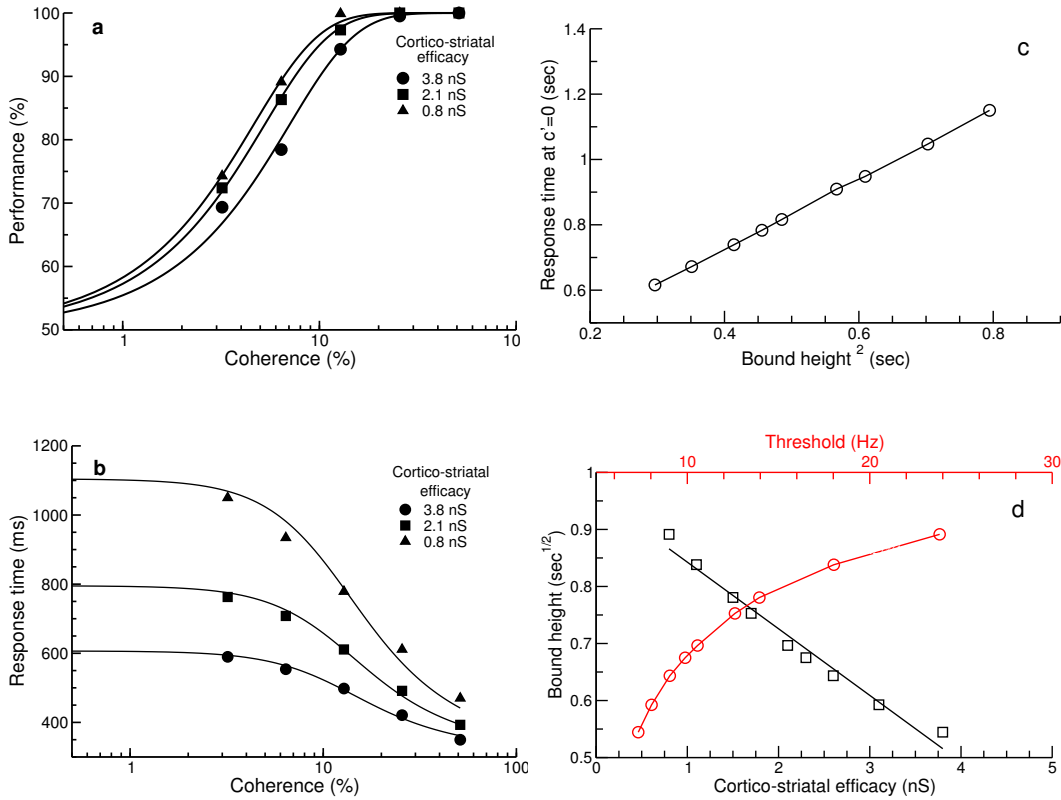
Supplementary figure 1: The sensitivity of the decision threshold to the cortico-collicular (Cx-SC) efficacy in the absence of the basal ganglia. We compare the effect of changing Cx-SC synaptic efficacy on the threshold with (circle, from Fig. 5b of the main paper) and without (square) the basal ganglia. It is evident that the basal ganglia reduces the sensitivity of the decision threshold to the Cx-SC efficacy. However, even without the basal ganglia the range of cortical firing threshold is still fairly limited.



Supplementary figure 2: Full network simulations in which single CD neurons are endowed with additional intrinsic ion channel mechanisms and exhibit Up and Down membrane states. We implement the non-inactivating potassium (KS) current and the inward rectifying potassium (IR) current [1, 2] for single CD neurons in the model. The IR current is activated at hyperpolarized membrane potentials and contributes a small outward current that stabilizes the Down state; whereas in the Up state the near-firing-threshold membrane potential is maintained with the help of the low-threshold KS current. (a) A membrane potential trace of a simulated CD neurons in its baseline activity demonstrates that the neuron switches between the Up and Down states at a frequency below 1Hz [1, 3]. The model behavior is not affected substantially by the addition of membrane bistability to CD neurons, as shown by (b) Population firing rates from a simulated trial, (c) psychometric functions, (d) chronometric functions and (e) decision threshold as a function of Cx-CD synaptic efficacy.

## References

- [1] Stern, E. A., Kincaid, A. E. & Wilson, C. J. Spontaneous subthreshold membrane potential fluctuations and action potential variability of rat corticostriatal and striatal neurons in vivo. *J. Neurophysiol.* **77**, 1697–1715 (1997).
- [2] Compte, A., Sanchez-Vives, M. V., McCormick, D. A. & Wang, X.-J. Cellular and network mechanisms of slow oscillatory activity (< 1 hz) in a cortical network model. *J. Neurophysiol.* **89**, 2707–2725 (2003).
- [3] Wilson, C. J. & Kawaguchi, Y. The origins of two-state spontaneous membrane potential fluctuations of neostriatal spiny neurons. *J. Neurosci.* **16**, 2397–2410 (1996).



Supplementary figure 3: Relationship between the bound height in the diffusion model and parameters of our model. (a) We fit the performance of our model using the function  $P(c') = 1/(1 + e^{-2Ak c'})$  predicted by the diffusion model [1], where  $c'$  is the coherence level,  $A$  is the bound height and  $k$  is the sensitivity to the stimulus. The  $Ak$  values are 10.93, 14.64 and 16.65 for the three curves from top to bottom, respectively. (b) We also fit the decision time of our model using the function  $t(c') = (A/kx) \tanh(Akx) + t_R$  predicted by the diffusion model [1], where  $t_R$  is the residual (non-decision) time. Assuming that  $t_R$  is constant for different threshold levels, we found  $(A, k) = (0.544, 20.71)$ ,  $(0.697, 16.7)$  and  $(0.891, 13.22)$  for the three curves from top to bottom, respectively. We set  $t_R = 310$  ms, which yields the best fits. We note that the ranges of shift in the psychometric and chronometric functions shown in (a) and (b) roughly correspond to those in Palmer et al. [1] for speed instructions between 0.5s and 1.0s. (c) As expected, If the model's chronometric data are fitted by the diffusion model, then the decision time at zero coherence increases quadratically with the bound height. (d) The bound height extracted from the diffusion model fit does not relate linearly with the cortical firing threshold (red). However, the efficacy of cortico-striatal synapses exhibits a linear relationship with the bound height (black). We note that the  $Ak$  values from the response time fits do not agree with those from the performance fits. A similar observation was also made in Ref [1]. Furthermore, as we vary the cortical firing threshold in the model, not only the bound height  $A$ , but also the sensitivity  $k$  change. These observations suggest that although our model can be fit approximately using the equations of the diffusion model, and there is a linear relationship between the cortico-striatal efficacy and the bound height, our neural network model is not an exact implementation of the diffusion model.

## References

- [1] Palmer, J., Huk, A. C. & Shadlen, M. H. The effect of stimulus strength on the speed and accuracy of a perceptual decision. *J. Vis.* **5**, 376–404 (2005).