Extended Thalamo–Cortical Neural Mass Model

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1 Introduction

We describe an extended conductance-based neural mass model of a thalamo–cortical circuit. Each source (cortical or thalamic) contains eight interacting neural populations spanning superficial and deep cortical layers and thalamic relay and reticular nuclei. The model incorporates multiple fast synaptic receptor types and slower voltage- or calcium-dependent intrinsic conductances, as well as a set of presynaptic and postsynaptic neuromodulatory gain mechanisms. This architecture supports rich oscillatory dynamics and permits mechanistic interpretation of pharmacological and pathological perturbations.

2 Populations, Channels, and Modulators

Neural populations

We model the following eight populations per cortical or thalamic source:

$$\mathcal{P} = \{\mathrm{SS}, \mathrm{SP}, \mathrm{SI}, \mathrm{DP}, \mathrm{DI}, \mathrm{TP}, \mathrm{RT}, \mathrm{RC}\}.$$

- **SS** – L4 spiny stellate (cortical input layer)
- **SP** – L2/3 superficial pyramidal
- **SI** – L2/3 superficial inhibitory interneurons
- **DP** – L5 deep pyramidal
- **DI** – L5 deep inhibitory interneurons
- **TP** – L6 corticothalamic projection pyramidal
- **RT** – thalamic reticular
- **RC** – thalamic relay

Receptor and channel types

Each population has conductance states for the following receptor and channel mechanisms:

$$\mathcal{R} = \{\mathrm{AMPA}, \mathrm{NMDA}, \mathrm{KA}, \mathrm{GABAA}, \mathrm{GABAB}, \mathrm{M}, \mathrm{HCN}, \mathrm{SK}, \mathrm{NaP}\}.$$

Neuromodulators

Presynaptic: {**CB1**, **MOR**} (cannabinoid and μ-opioid receptors) Postsynaptic: {**5HT2A**, **5HT1A**, **AChM1**, **DAD1**, **NE**β} (serotonergic, cholinergic, dopaminergic, noradrenergic)

Modulators act multiplicatively on synaptic drive (presynaptic) or channel gain (postsynaptic).

Table 1: Receptor and channel abbreviations.

Acronym	Description
AMPA	Fast ionotropic glutamate receptor (excitatory)
NMDA	Slow ionotropic glutamate receptor (voltage-dependent block)
KA	Kainate-type glutamate receptor (fast excitatory)
GABAA	Fast ionotropic γ-aminobutyric acid receptor (inhibitory)
GABAB	Slow metabotropic GABA receptor (inhibitory)
M	Muscarinic-sensitive M-type potassium channel (slow adaptation)
HCN	Hyperpolarization-activated cyclic nucleotide-gated channel (I_h pacemaker)
SK	Small-conductance Ca^{2+}-activated K^+ channel (afterhyperpolarization)
NaP	Persistent sodium channel (slow depolarizing drive)

3 Dynamics

3.1 Membrane equation

Each population has a membrane potential $V_i(t)$ governed by

$$C_i \dot{V}_i \;=\; \sum_{r\in\mathcal{R}} g_i^r(t)\,(E_r - V_i) \;+\; I_i^{\text{ext}}(t), \tag{1}$$

where g_i^r are conductance states and E_r their reversal potentials (e.g. $E_{\text{AMPA}} \approx +60$ mV, $E_{\text{GABAA}} \approx -90$ mV).

3.2 Presynaptic firing and delays

Presynaptic firing is a static nonlinearity of voltage:

$$r_j(t) = \frac{r_{\max,j}}{1+\exp\{-\kappa_j\,[V_j(t)-\vartheta_j]\}}, \tag{2}$$

and is subject to a pre $\to$ post axonal delay d_{ij} on each edge $j \to i$.

3.3 Synaptic edge weights and presynaptic modulation

Each anatomical edge $j \to i$ expands into receptor-specific weights w_{ij}^r, determined by presynaptic class:

$$\mathcal{E} = \{\text{SS}, \text{SP}, \text{DP}, \text{TP}, \text{RC}\}, \qquad \mathcal{I} = \{\text{SI}, \text{DI}, \text{RT}\}.$$

We parameterise log-gains for positivity:

$$g_{ij}^r = g_{ij}^{r,\text{base}}\,\exp\,(\theta_{ij}^r), \tag{3}$$

where $g_{ij}^{r,\text{base}}$ encodes receptor-mix fractions (AMPA/NMDA/KA for $\mathcal{E}$; GABAA/GABAB for $\mathcal{I}$).

Presynaptic modulators act as multiplicative scalers on presynaptic drive:

$$\mathcal{M}_{ij,r}^{\text{pre}}(P) = \prod_{m\in\{\text{CB1,MOR}\}} \exp(\alpha_{m,ij,r}^{\text{pre}}). \tag{4}$$

3.4 Postsynaptic drive and modulation

The receptor-specific drive arriving at population i is

$$\mathrm{Drive}_i^r(t) = \sum_{j \to i} g_{ij}^r \, \mathcal{M}_{ij,r}^{\mathrm{pre}}(P) \, \mathrm{STP}_{ij}^r(t) \, r_j(t - d_{ij}) + \mathrm{BE}_i^r + \mathrm{Ex}_i^r(t). \tag{5}$$

Postsynaptic modulators scale the channel gain:

$$G_{r,i}^{\mathrm{post}}(P) = \prod_{m \in \{5\mathrm{HT2A},5\mathrm{HT1A},\mathrm{AChM1},\mathrm{DAD1},\mathrm{NE}\beta\}} \exp(\alpha_{m,r,i}^{\mathrm{post}}). \tag{6}$$

3.5 Channel (gate) dynamics

Each receptor/channel $r \in \mathcal{R}$ has a conductance gate $g_i^r(t)$.

Synaptic receptors

$$\tau_r \, \dot{g}_i^r(t) = -\, g_i^r(t) + G_{r,i}^{\mathrm{post}}(P) \times \begin{cases} \mathrm{Drive}_i^{\mathrm{NMDA}}(t) \, B\big(V_i(t)\big), & r = \mathrm{NMDA}, \\ \mathrm{Drive}_i^r(t), & \text{otherwise}, \end{cases} \tag{7}$$

where $B(V) = \frac{1}{1+\eta e^{-\gamma V}}$ is the NMDA Mg^{2+} block.

Intrinsic channels

$$\tau_r \, \dot{g}_i^r(t) = -\, g_i^r(t) + G_{r,i}^{\mathrm{post}}(P) \; T_r\big(V_i(t), \, \zeta_i(t)\big), \tag{8}$$

with

$$T_{\mathrm{M}}(V) = \sigma\!\left(\frac{V + 35}{8}\right), \qquad T_{\mathrm{HCN}}(V) = \sigma\!\left(\frac{-V - 80}{6}\right), \tag{9}$$

$$T_{\mathrm{NaP}}(V) = \sigma\!\left(\frac{V + 55}{7}\right), \qquad T_{\mathrm{SK}}(\zeta) = \frac{\alpha \, \zeta}{1 + \alpha \, \zeta}, \tag{10}$$

and $\zeta_i(t) = g_i^{\mathrm{NMDA}}(t)$ as a Ca^{2+} proxy for SK.

3.6 Observation model

We observe a weighted sum of state variables with additive noise:

$$y(t) = C \, x(t) + \varepsilon(t), \qquad x(t) = \big[\, V_i(t), \; \{g_i^r(t)\}_{r \in \mathcal{R}} \,\big]_{i \in \mathcal{P}}^{\top}. \tag{11}$$

4 Parameters and Defaults

Free parameters typically include:

- Edge log-gains θ_{ij}^r for each $(j \to i, r)$
- Time constants $\{\tau_r\}_{r \in \mathcal{R}}$
- Modulator sensitivities $\{\alpha_{m,ij,r}^{\mathrm{pre}}\}$ and $\{\alpha_{m,r,i}^{\mathrm{post}}\}$
- Observation gains C and noise hyperparameters

Default values are chosen for stable operation and realistic gain functions, e.g. $r_{\max} = 1$, STP= 1 if disabled, and $I^{\mathrm{ext}} = 0$ unless explicitly driven. Setting $g_{ij}^{r,\mathrm{base}} = 0$ removes a receptor from an edge.