Recurrent Neuronal Models

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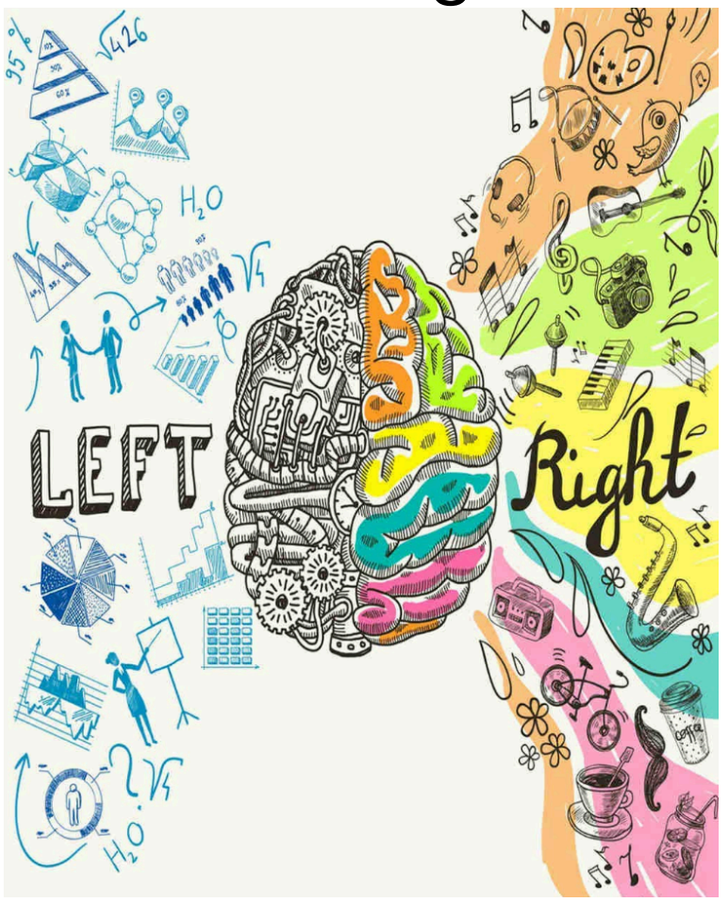
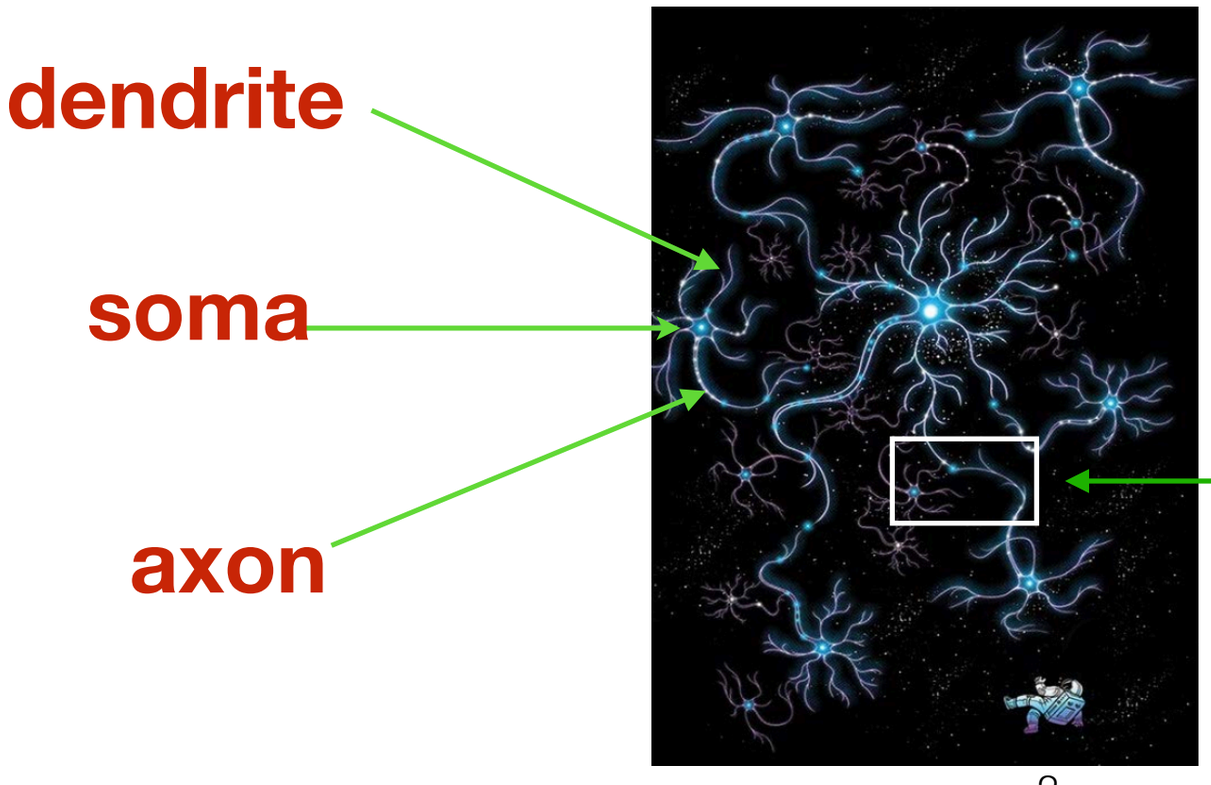
A Brain-Mind Odyssey:

- A **brain** is a complex organ controls over a body.
- Each region codes specific features of an environment. All together form our experience of the world.
- A brain has billions of neurons.
- **Neurons** are specialized cells in the nervous system that transmit information to other nerve cells through excitation.

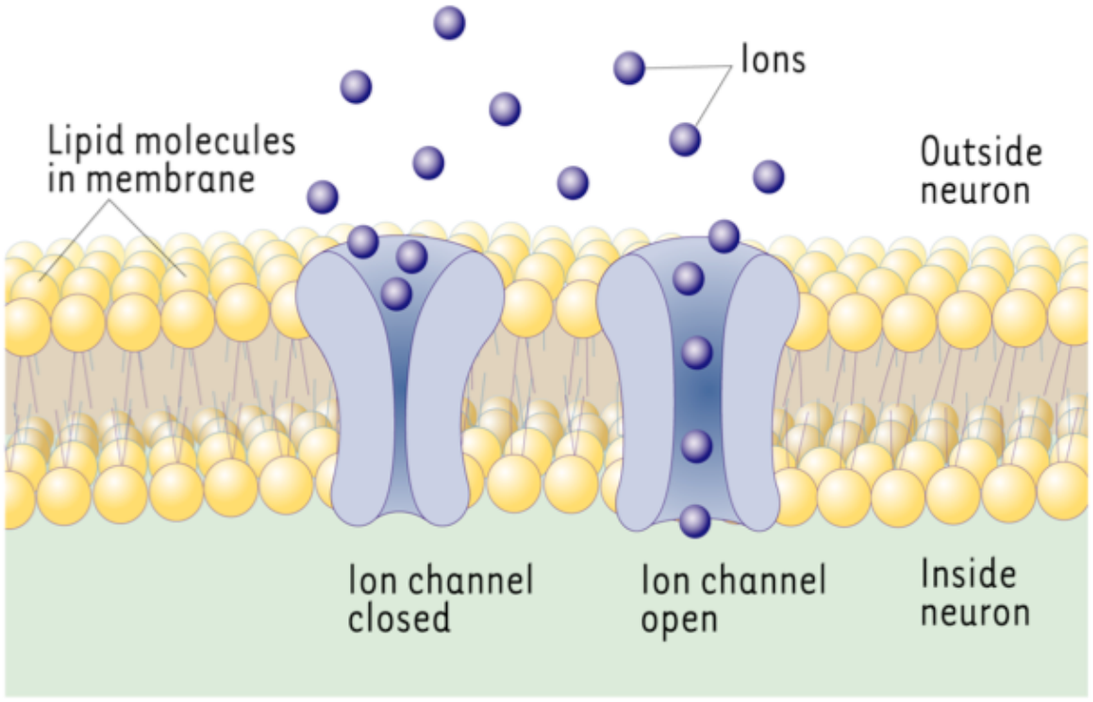


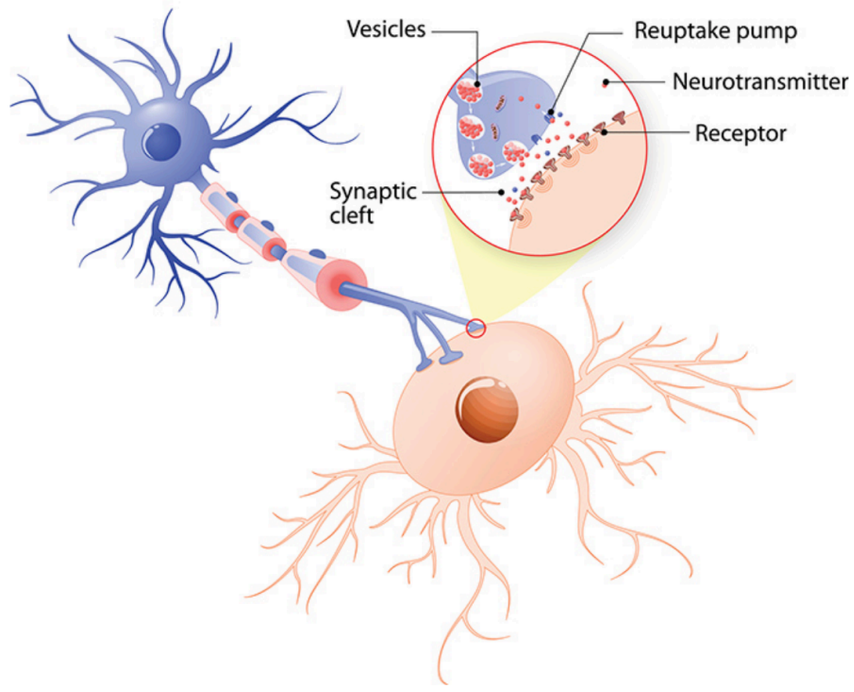


Synapse is the connection between two neurons.

A Brain-Mind Odyssey:

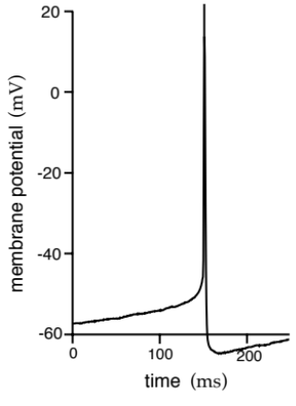
- A neuron has a **membrane** that is made of a bilayer of lipid molecules with many types of protein embedded in it.



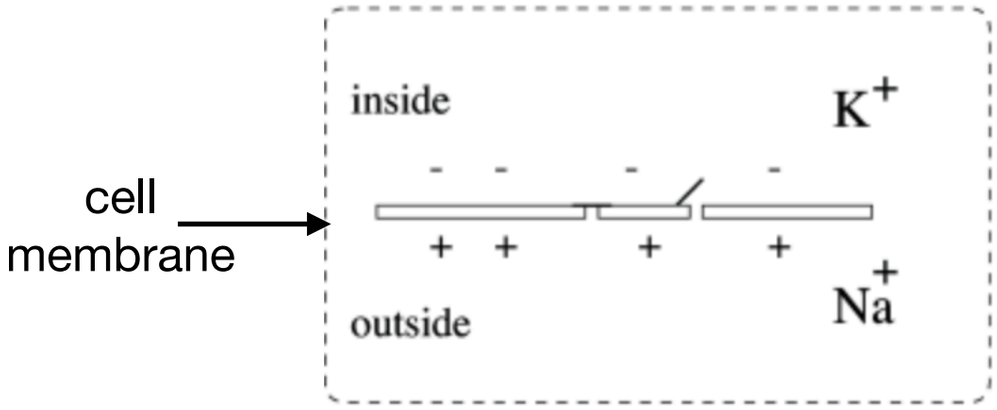


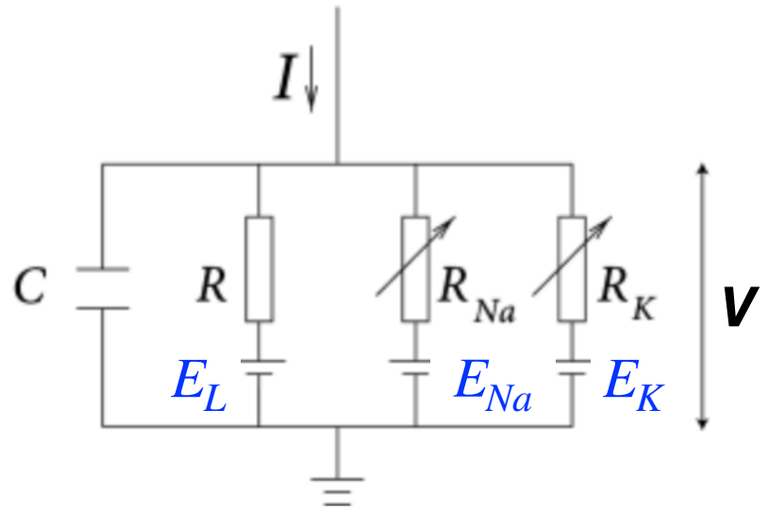
- Neurons send and receive signals via **neurotransmitters**, a type of chemical messenger controls the ion channels to change the neuron's membrane potential (voltage differential).

- When a neuron has large voltage differential (depolarized), an action potential/**spike** happens.



Hodgkin-Huxley Model





C: capacitance, measures its lipid bilayer allowance for the voltage differential.

R: resistor, measures the allowance for the ions to pass through.

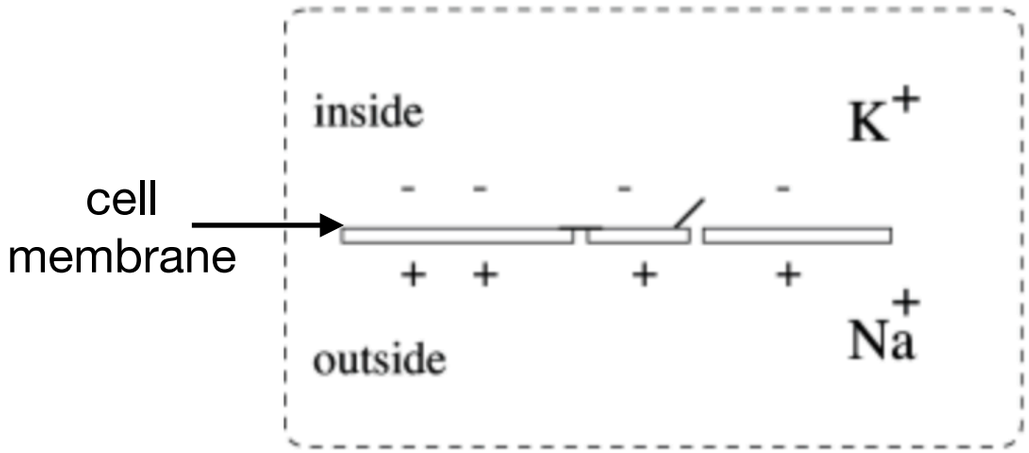
E, reverse potentials: a measure of zero net flow.

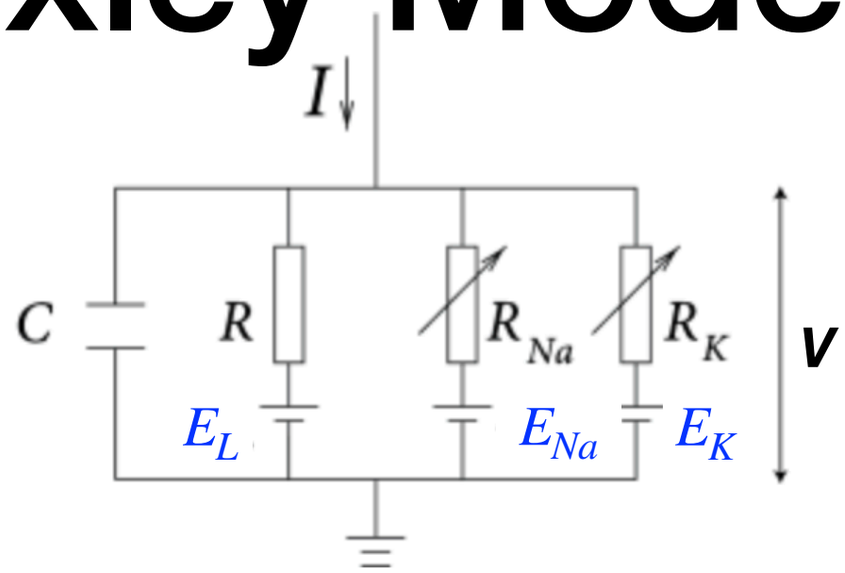
- **A biological model:** want to measure current(I) across the cell membrane(C) through the changes of membrane potentials(V), $I_c = C\frac{dV}{dt}$
- By **conservation** of electric charge, $I_{inj.} = I_C + I_{pass}$

  The charging current $I_C = C\frac{dV}{dt} = -I_{pass} + I_{inj.}$
- Apply **Ohm's law,** $I_{pass} = gV$, so $I_{total} = \sum_{ion} g_{ion}(V - E_{ion})$
  - g: conductance: measures the resistant level of each ion channel.

(Gerstmer *et al.*, *Neuronal Dynamics*, 2014)

Hodgkin-Huxley Model





$$I = C\frac{dV}{dt} = -\bar{g}_L(V-E_L) - \bar{g}_K n^4(V-E_K) - \bar{g}_{Na} m^3 h(V-E_{Na}) + I_{inj.}$$

$$\frac{dn}{dt} = (1-n)\alpha_n(V) - n\beta_n(V)$$

$$\frac{dm}{dt} = (1-m)\alpha_m(V) - m\beta_m(V)$$

$$\frac{dh}{dt} = (1-h)\alpha_h(V) - h\beta_h(V)$$

Na: 2 types of gates
m is fast, h is slow

gating variables(n, m, h): probability controls ion channels.

(i.e. the effective conductance of Na is modelled as $\frac{1}{R_{Na}} = g_{Na} m^3 h$)

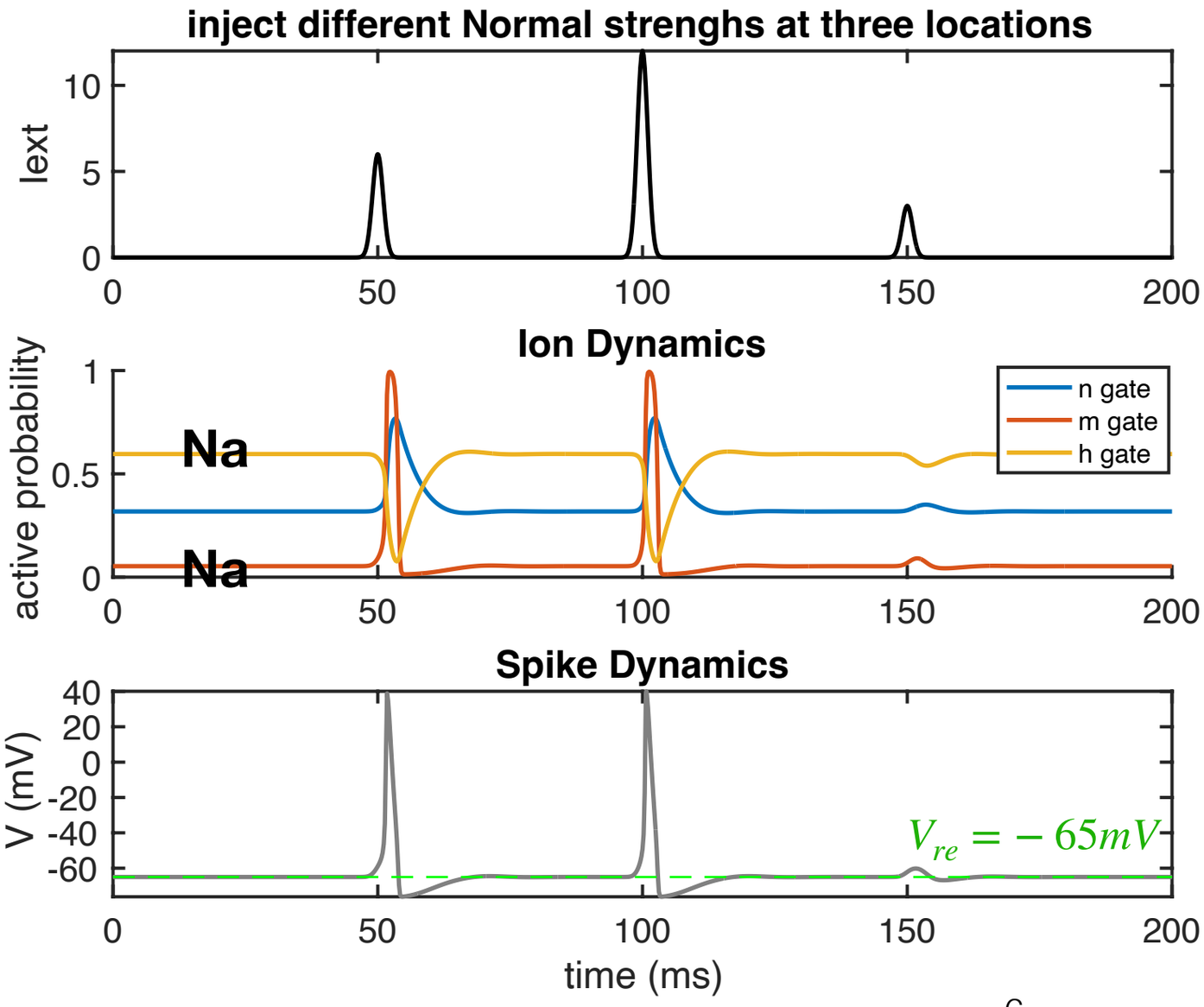
—α: the rate of closed gates open; β: the rate of open gates close

(Gerstmer *et al.*, *Neuronal Dynamics*, 2014)

Hodgkin-Huxley Model

HH model describes the **spiking** dynamics:

- Spike(voltage difference=100mV) appears when given strong input;
- Spike is short(width=2.5ms);
- After spike, V fails below resting potential, $V_{re} = -65mV$.



Ion dynamics:

- m causes V to increase,
- h stops V from increasing,
- n causes V to decrease

Drawback

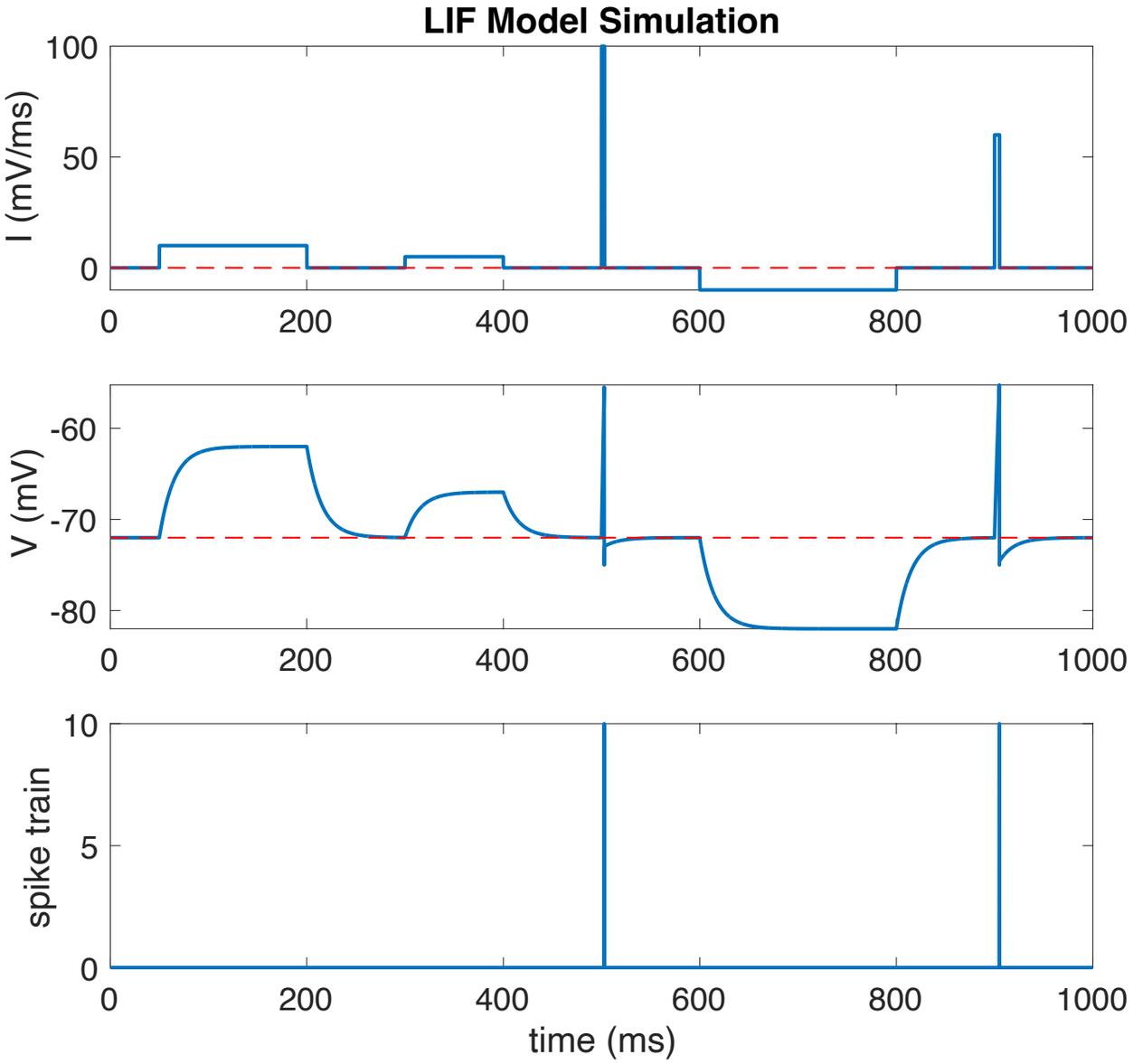
computational complex

Integrate-and-fire Models

Leaky IF model : $C\frac{dV}{dt} = -g_L(V(t) - E_L) + I(t)$

when $V(t) > V_{th} \approx 10mV$: record a spike,

reset $V(t)$ to $V_{re} \approx -75mV$



- Reset is to capture K channel effect. (*i.e.* spikes have the same height)
- This model captures neurons decay back to the E_L w/o input. *(i.e.* solve the ode, exponential decay)

Drawback

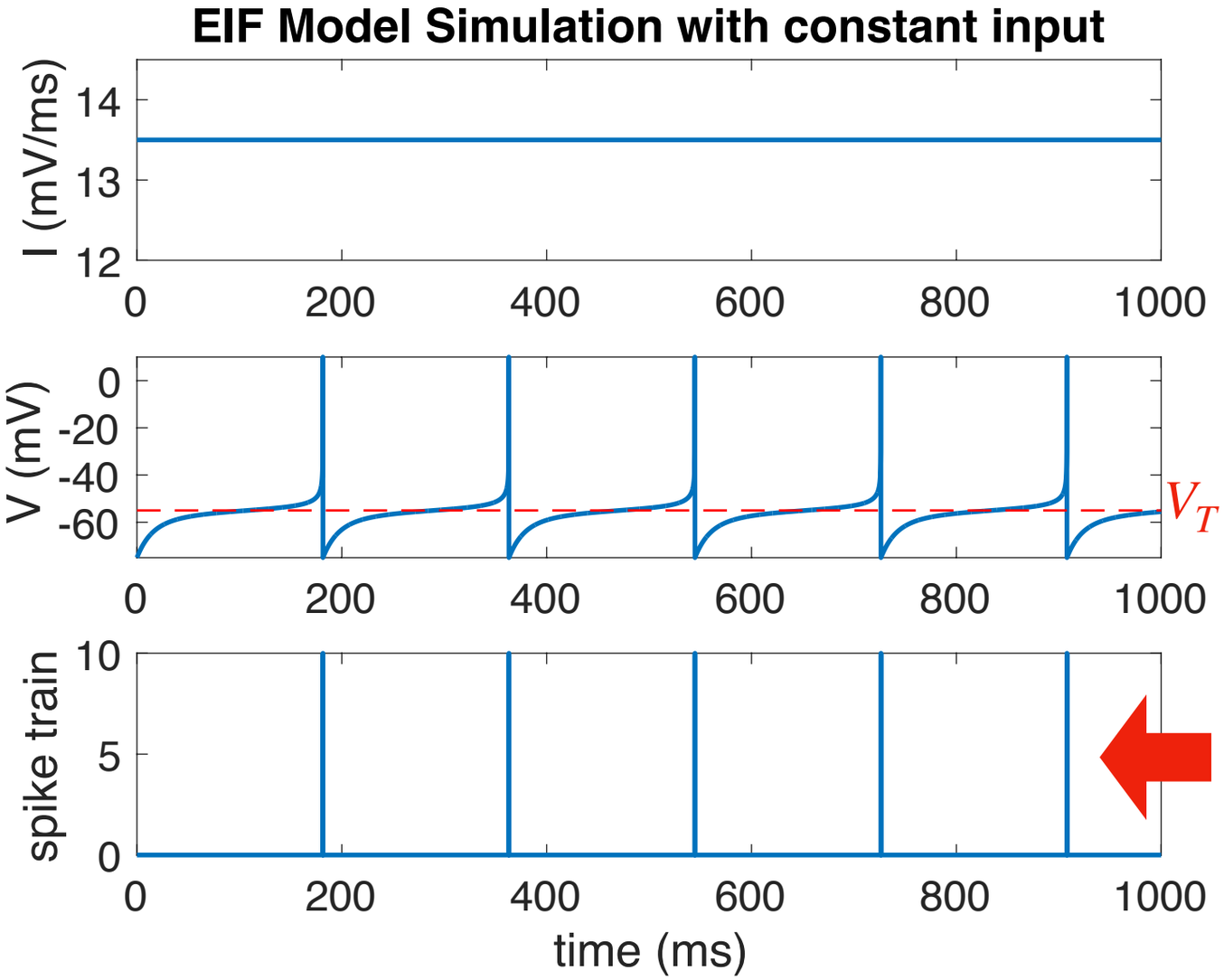
Na channels are ignored (less realistic!)

Integrate-and-fire Models

Exponential IF model: $C\frac{dV}{dt} = -g_L(V(t) - E_L) + g_L\Delta_T exp(\frac{V(t) - V_T}{\Delta_T}) + I(t)$

$V_T \approx -55mV$, is a **soft threshold**, Δ_T is a scaling factor.

when $V(t) > V_{th}$: record a spike, reset $V(t)$ to V_{re}



- The exp. term approximates the effects of Na channel in HH model.
- When $\Delta_T \to 0$, EIF = LIF

Spike train: a point process,

$S(t) = \sum_n \delta(t - t_i)$, can be modelled as $Pois(r)$

Spike rate: $r(t) = \lim_{\Delta \to 0} \frac{\int_t^{t+\Delta} S(t)dt}{\Delta} = E[S(t)]$

Synapses on IF models

So far, neuron models driven by an applied current, $C\frac{dV}{dt} = f(V, I) + I_{app}(t)$

other neurons

Synaptic current : in post-syn neurons' membrane, $I_{syn}(t) = g_{syn}(t) \cdot (V(t) - E_{syn})$

after pre-syn neuron spikes, the **conductance:** $g_{syn}(t) = J \cdot \sum_n \alpha(t - t_{spike})$

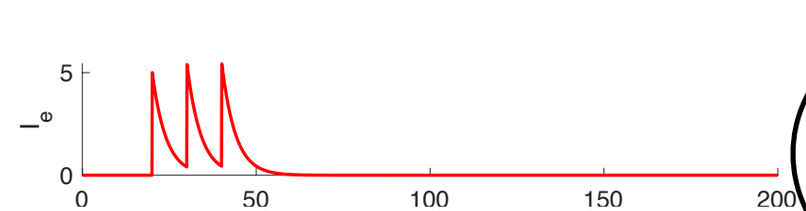
Synaptic strength: J with Je>0 and Ji<0

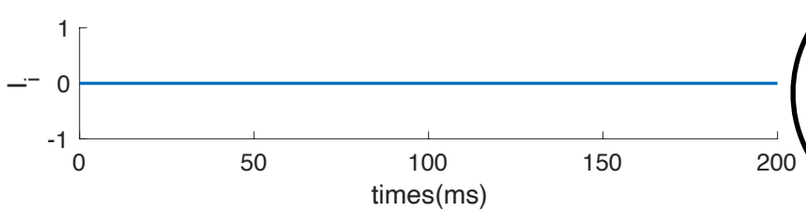
Q: What does a post-syn neuron look like after a pre-syn neuron spikes?

neurotransmitter: smooth out synaptic activities, $\alpha(t) = \frac{1}{\tau_{syn}} exp^{-\frac{t}{\tau_{syn}}} H(t)$

Post-synaptic conductance waveform, $\alpha(t)$

s.t. $\int \alpha(t) = 1$, i.e. $\alpha(t) = \delta$

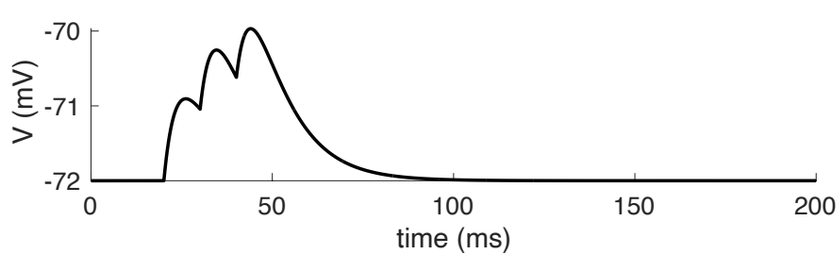




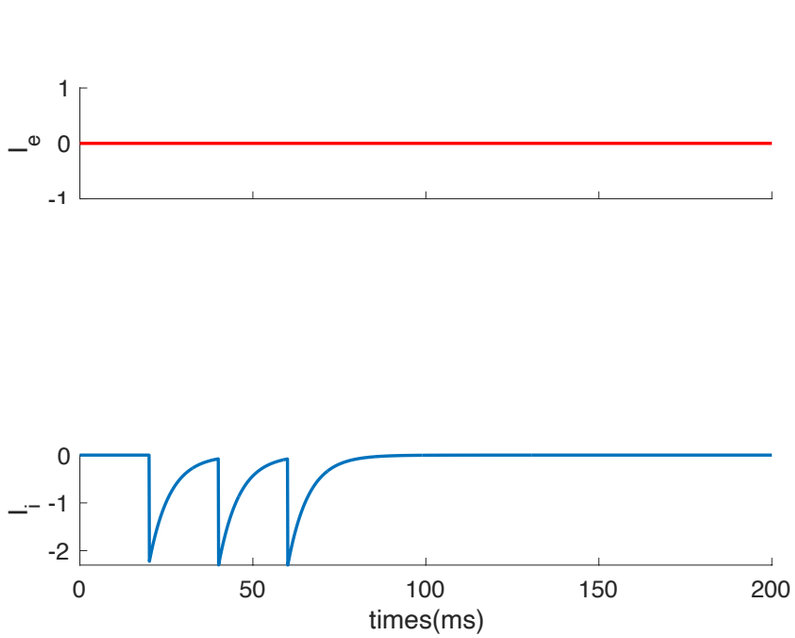
1 exc. neuron

1 inh. neuron

post-syn neuron



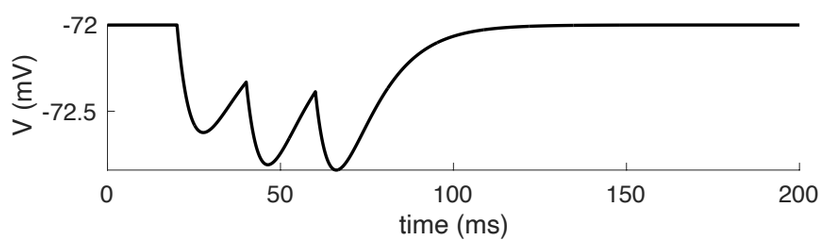
Synapses on IF models



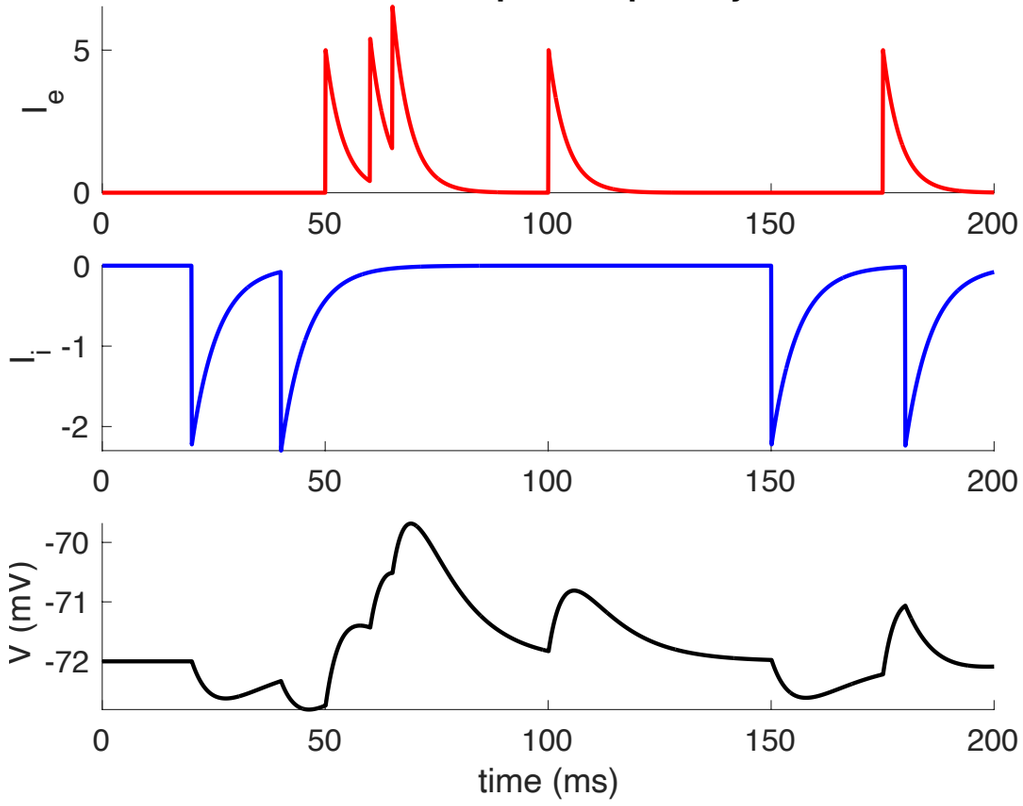
1 exc. neuron

1 inh. neuron

post-syn neuron

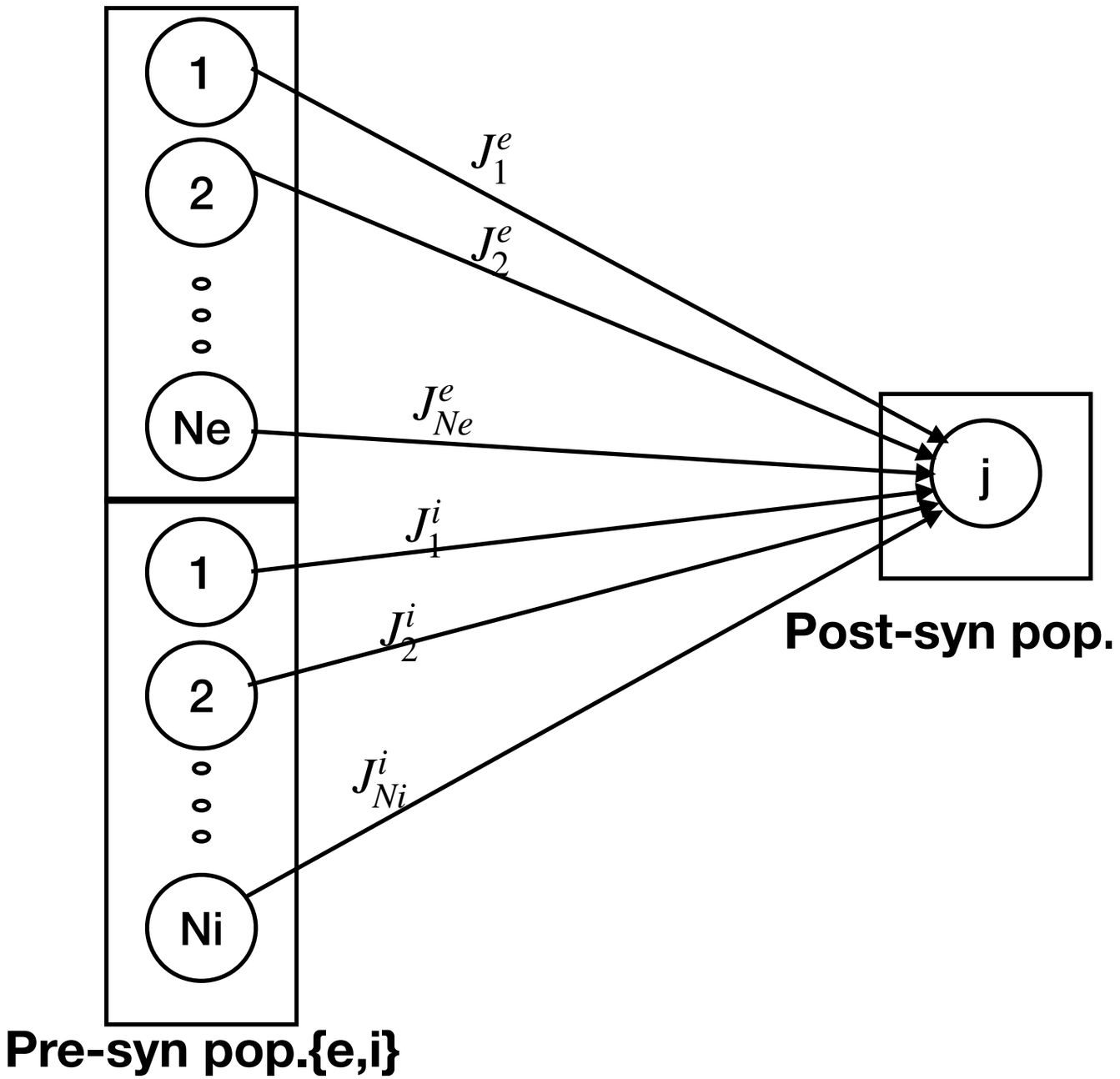


1 exc. and 1 inh. input to 1 post-syn. neuron



Feedforward Input

More realistic, a post-syn neuron j receives inputs from many presynaptic neurons.

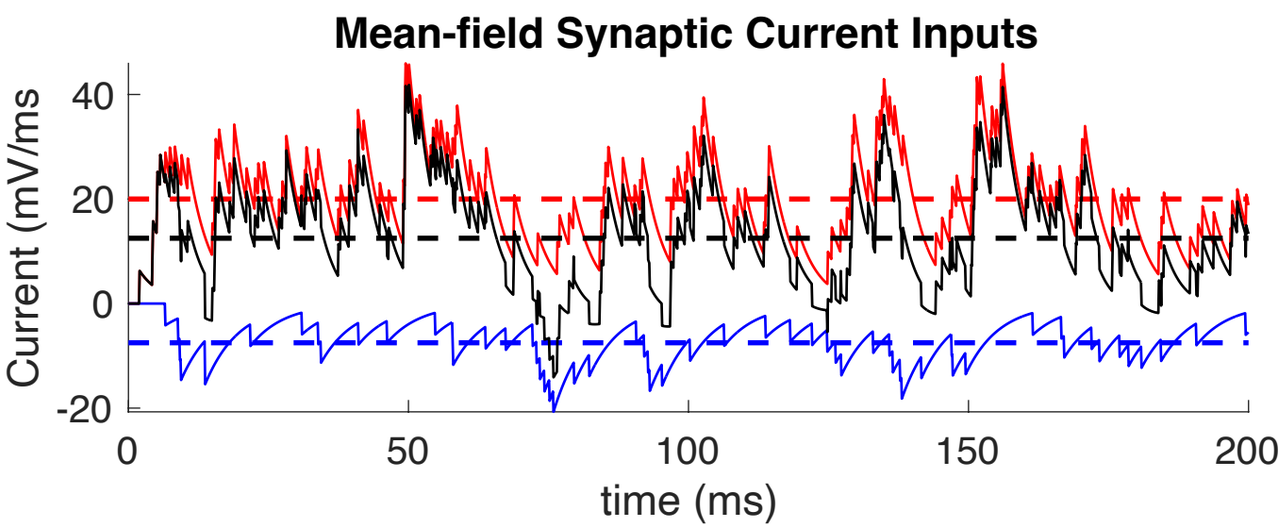


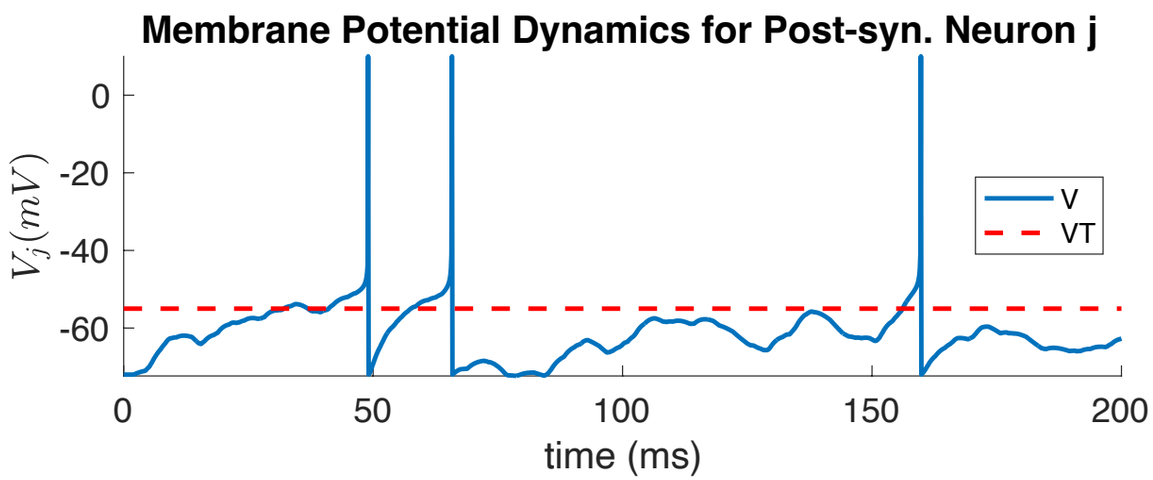
Solve the ode for $\bar{I}_{syn}$

Mean-field Input

$$\begin{cases} \tau \frac{dV_j}{dt} = f(V_j) + I_{syn} \\ \tau_b \frac{dI_b}{dt} = -I_b + J^b S^b \end{cases}$$ with b = {e, i} ; and $S^b(t) \sim Pois(r^b)$, so $E[S^b] = r^b$

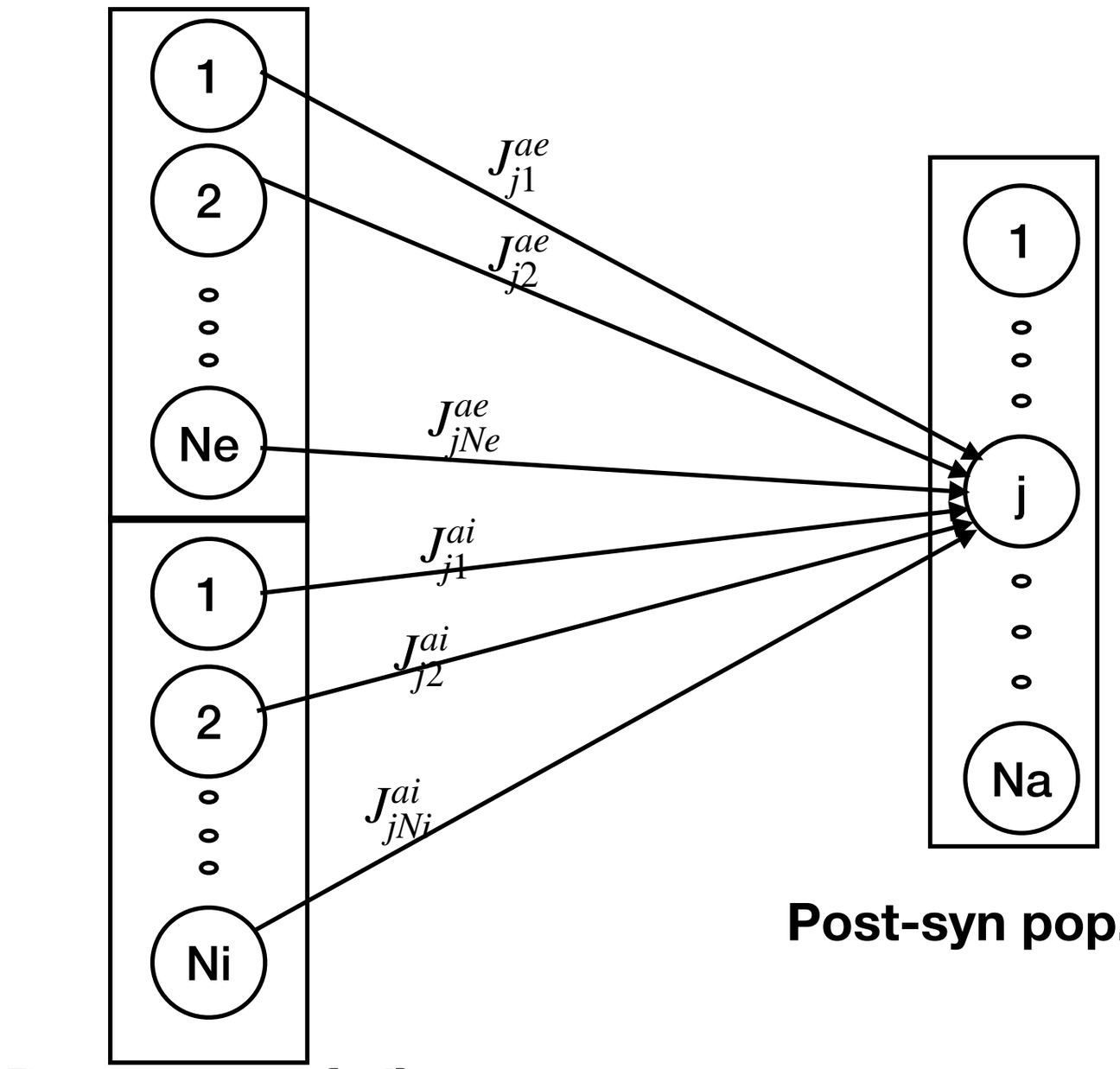
- Take expectation(time-average): $\tau_b \frac{d\bar{I}_b}{dt} = -\bar{I}_b + J^b E[S^b]$
- Solve for the ODE: $\bar{I}_b$ converges exponentially to the fixed point, $J^b r^b = \sum_k^{N_b} j_k^b r^b$.
- **Mean-field Input**: $\bar{I}_b = N_b j^b r^b$ is the time-average of $I_b(t)$.
- Without $I_{app}(t)$, Mean Total input, $\bar{I} = \bar{I}_e + \bar{I}_i$





Feedforward Network Models

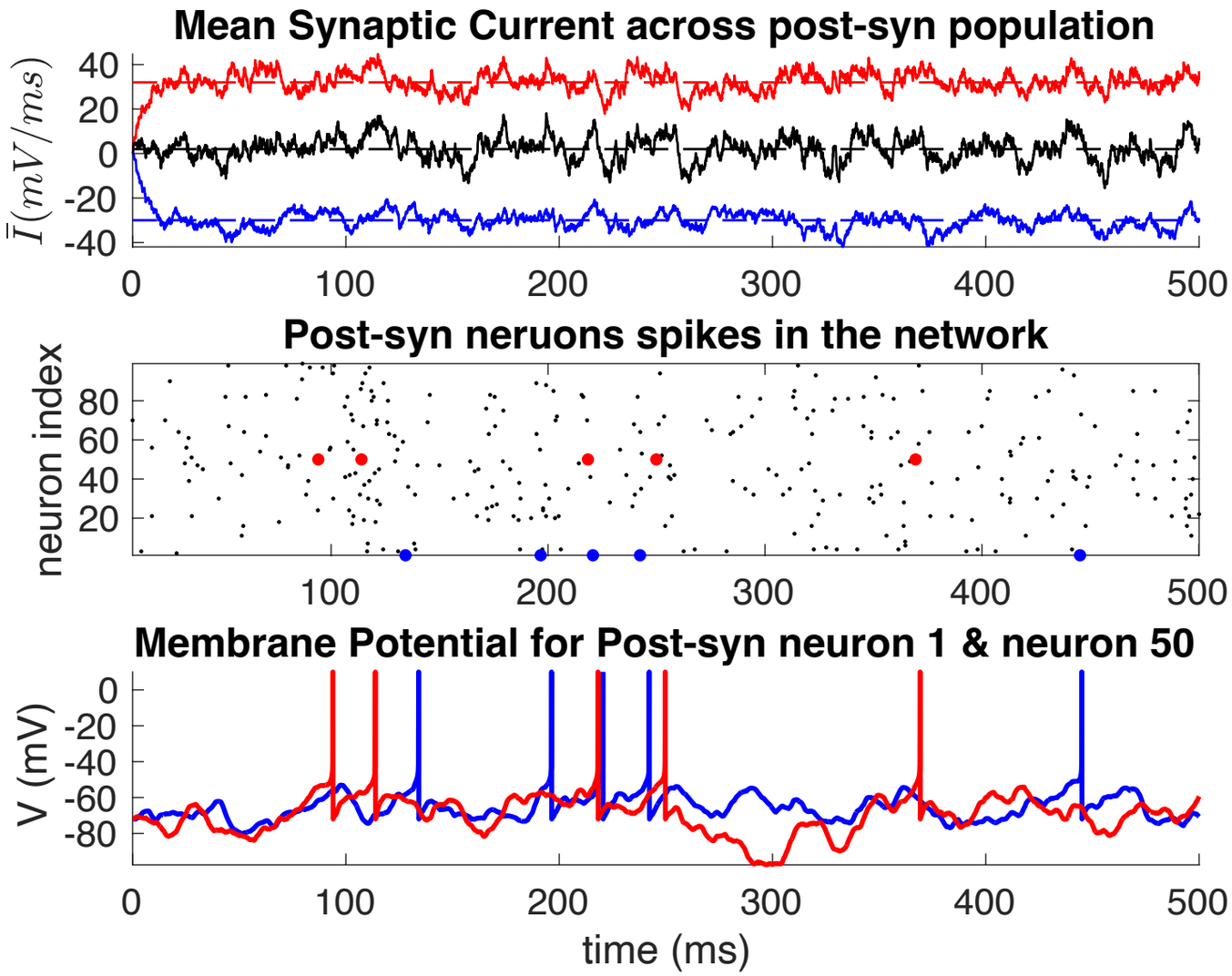
More realistic: we have a post-syn neuron population instead of a single neuron.



Solve the ode for $\bar{I}_{syn}$

From mean-field theory: $\bar{I}_{syn} = \frac{1}{N_a} \sum_{j}^{N_a} \left(N_e p_{ae} j_{ae} r_e + N_i p_{ai} j_{ai} r_e \right)$

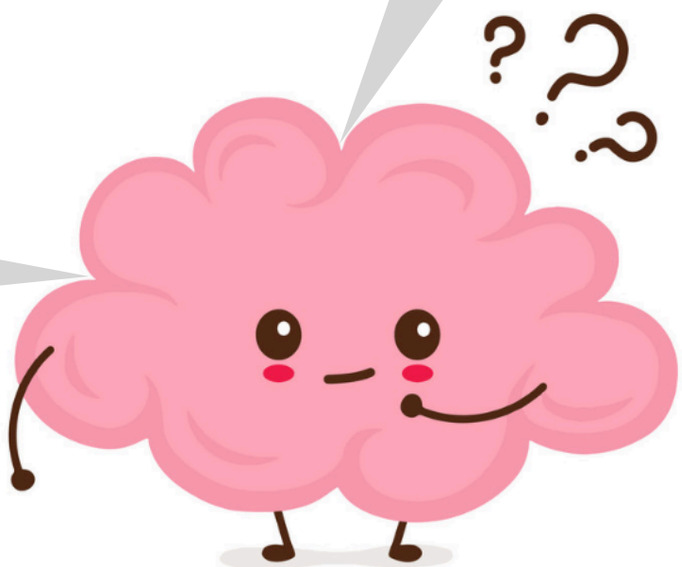
Feedforward Network Models



- **fluctuation around mean;**
- **Individual neuron receives large exc. & inh. input;**
- **cancellation, $\bar{I}_{total}$ is small.**

Balanced Network?

Firing rate, r^{post} ?



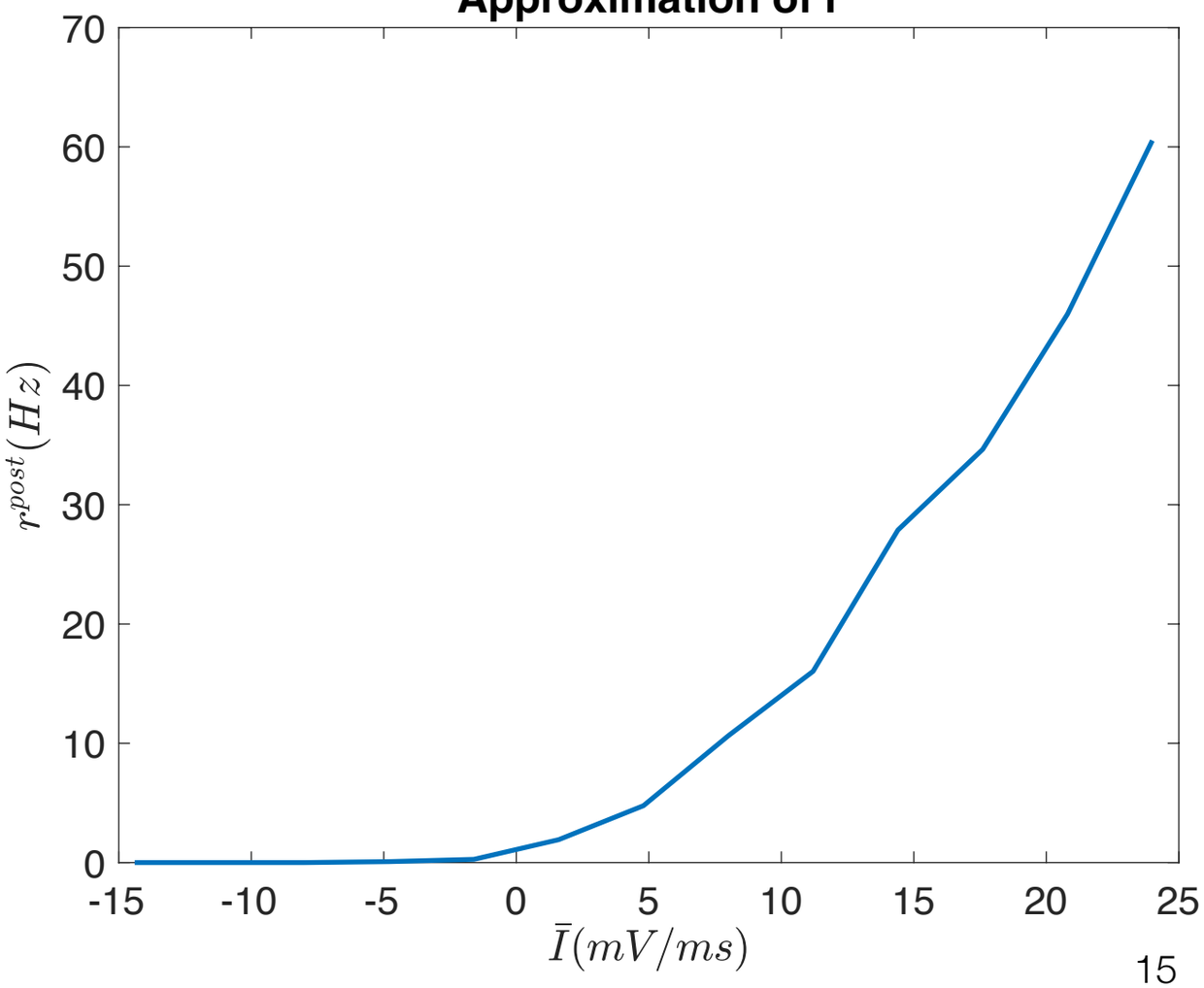
Claim: $\bar{r}^{post} \approx f(\bar{I})$

Dales Law: a neuron connects to all its post-syn targets with the same "type" of synapse.

Recall, $\bar{I}_{syn} = \frac{1}{N_a} \sum_{j}^{N_a} \left(N_e p_e j_e r_e + N_i p_i j_i r_i \right)$

If defined a weighted connectivity $W = \begin{bmatrix} w_{ee} & w_{ei} \\ w_{ie} & w_{ii} \end{bmatrix} = \begin{bmatrix} + & - \\ + & - \end{bmatrix}$ s.t. $w_{ab} = N_b p_{ab} j_{ab}$

In matrix notation, $\bar{I} = W r^{pre}$; with $r^{pre} = \begin{bmatrix} r_e \\ r_i \end{bmatrix}$



- approximate $\bar{r}^{post} \approx f(W r^{pre})$
- $f(\cdot)$ is non-decreasing
  (*i.e.* $f(\cdot) = [\cdot]^+$, $f(\cdot)$ = sigmoid…)

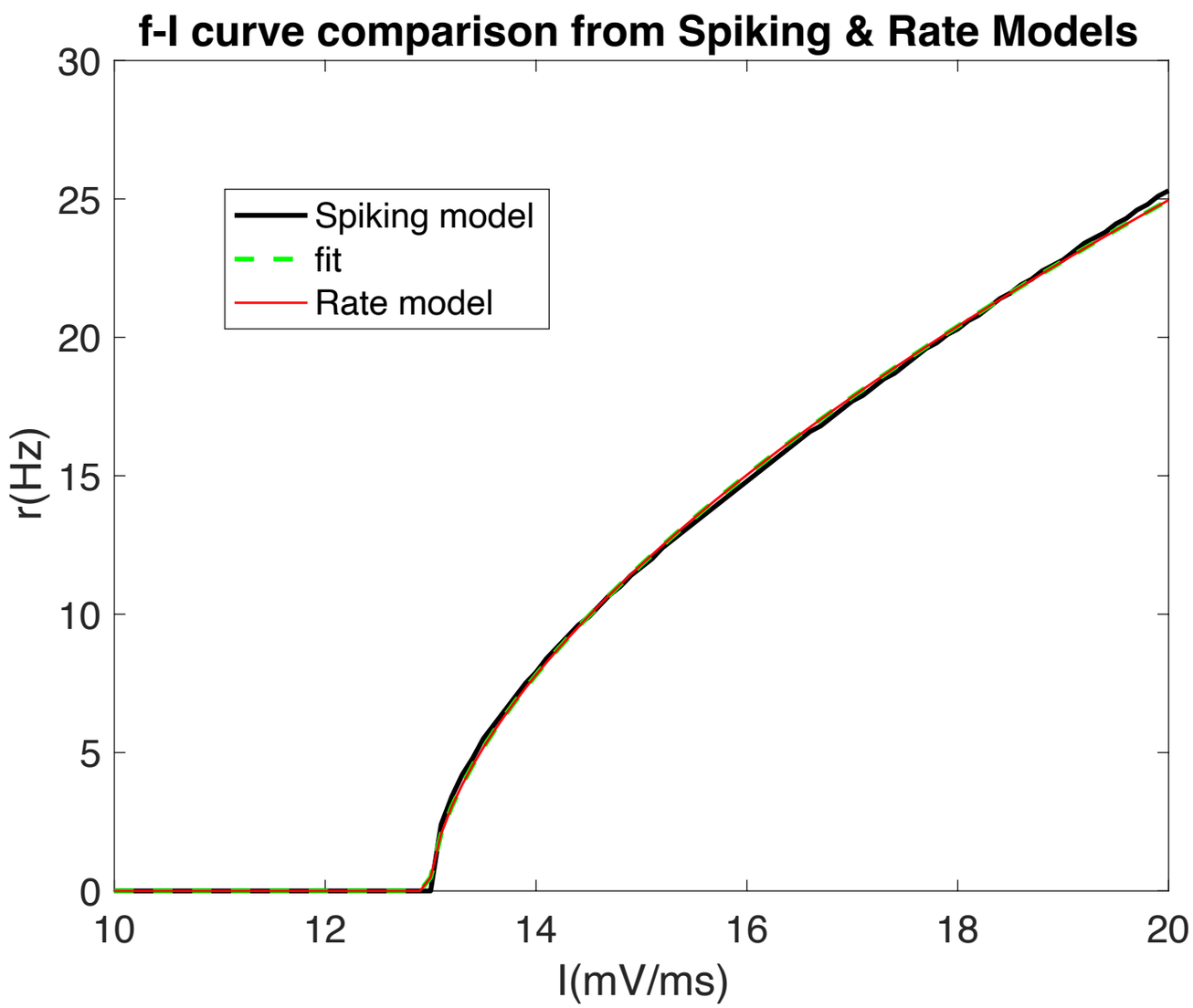
Rate Models

So far, we discussed spiking models that track the membrane potential,V

Rate Model : focus on the frequency of spikes in large network

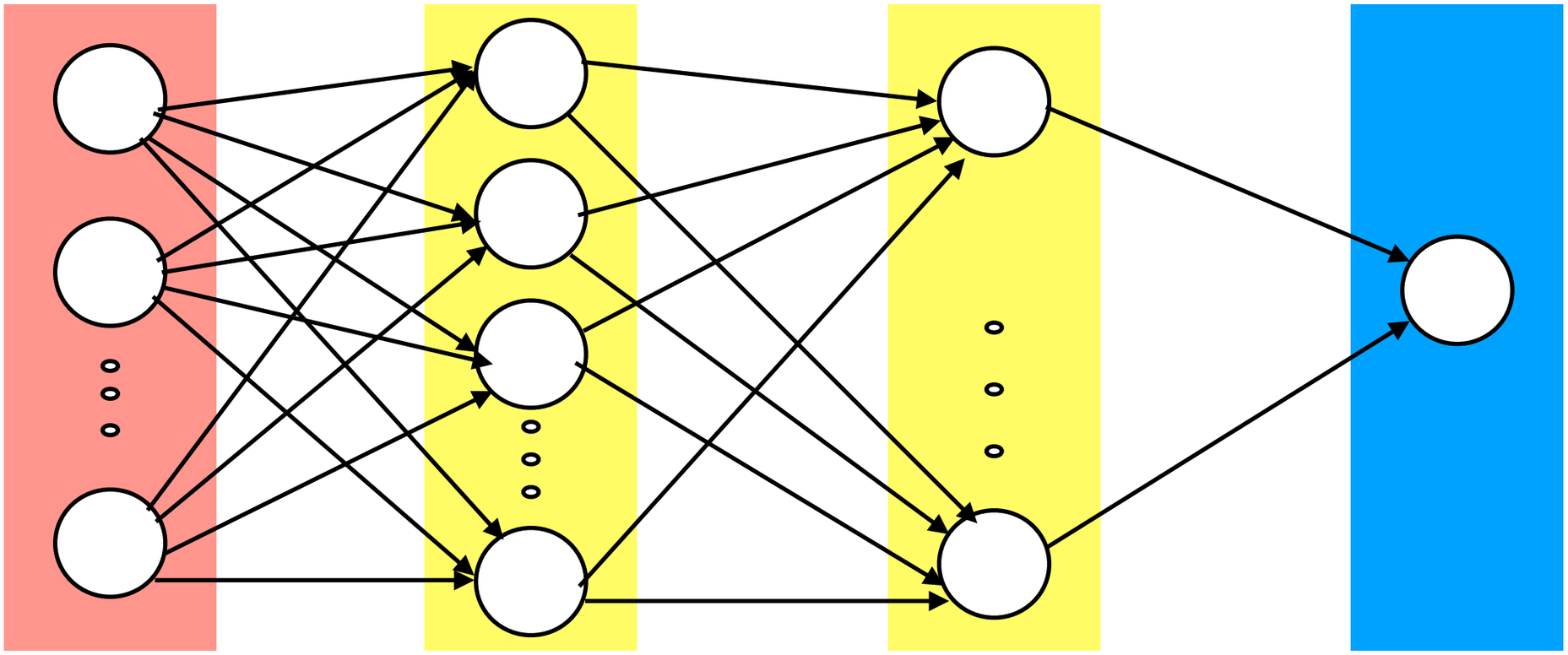
$$\tau_r \frac{d\vec{r}}{dt} = -\vec{r} + f(W\vec{r} + X)$$

Drawback



This is one neuron spiking rate and it ignores biophysical details!!!

Feedforward Network Models



Input

Hidden layers

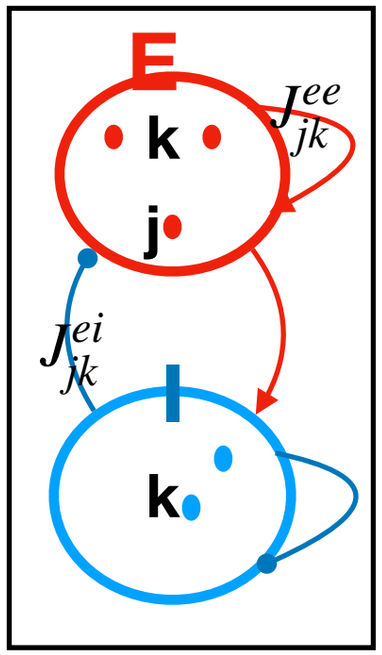
Output

One advantage of rate model is to connect with Artificial Neural Networks

$$\begin{cases} r^1 = f(W^1 r^0) \\ r^2 = f(W^2 r^1) \\ \cdots \\ r^m = f(W^m r^{m-1}) \end{cases}$$

Recurrent Network Models

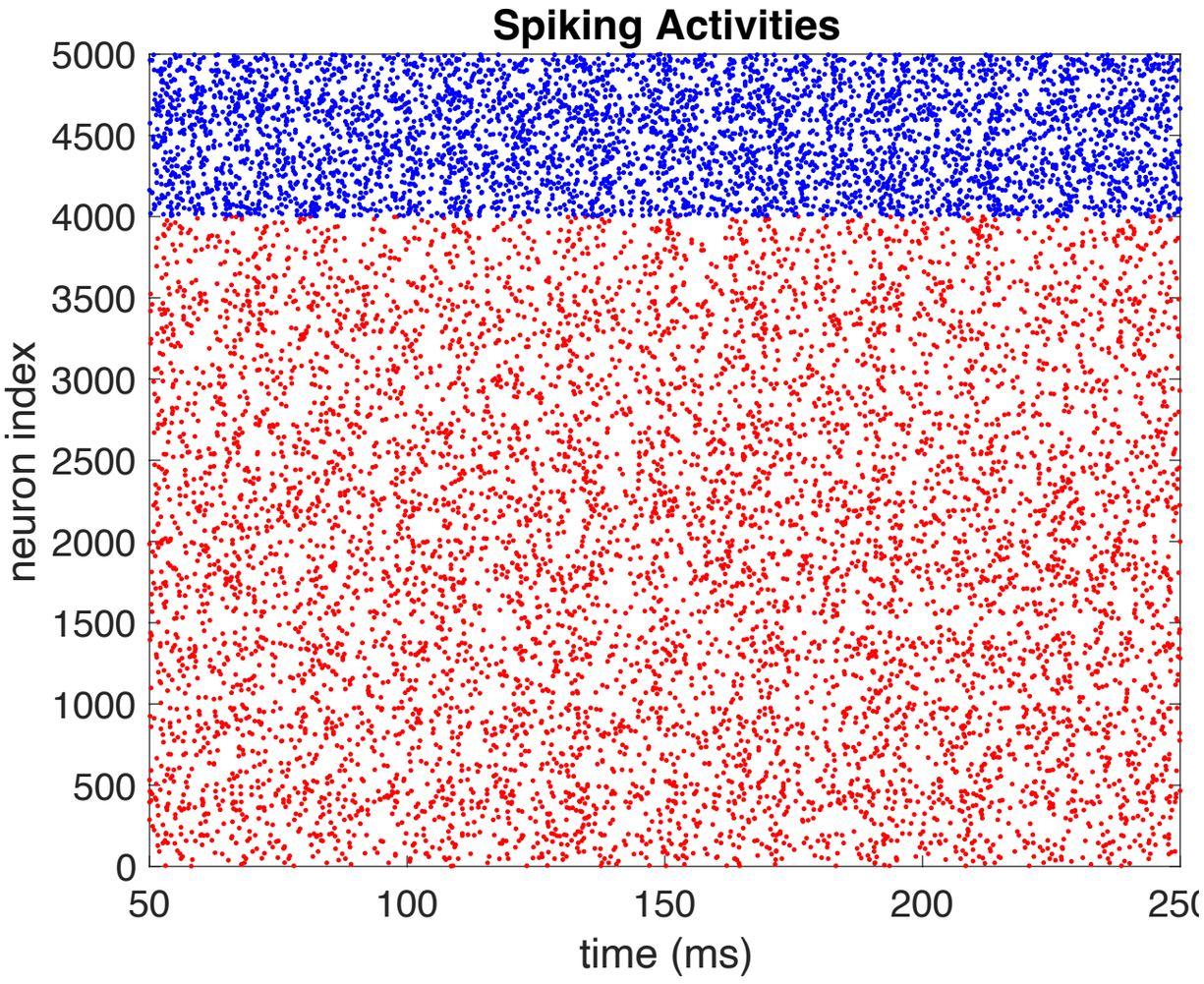
—Reality: Neurons in the same layer are interconnected
—Recurrent Network: there is a path from a neuron back to itself.

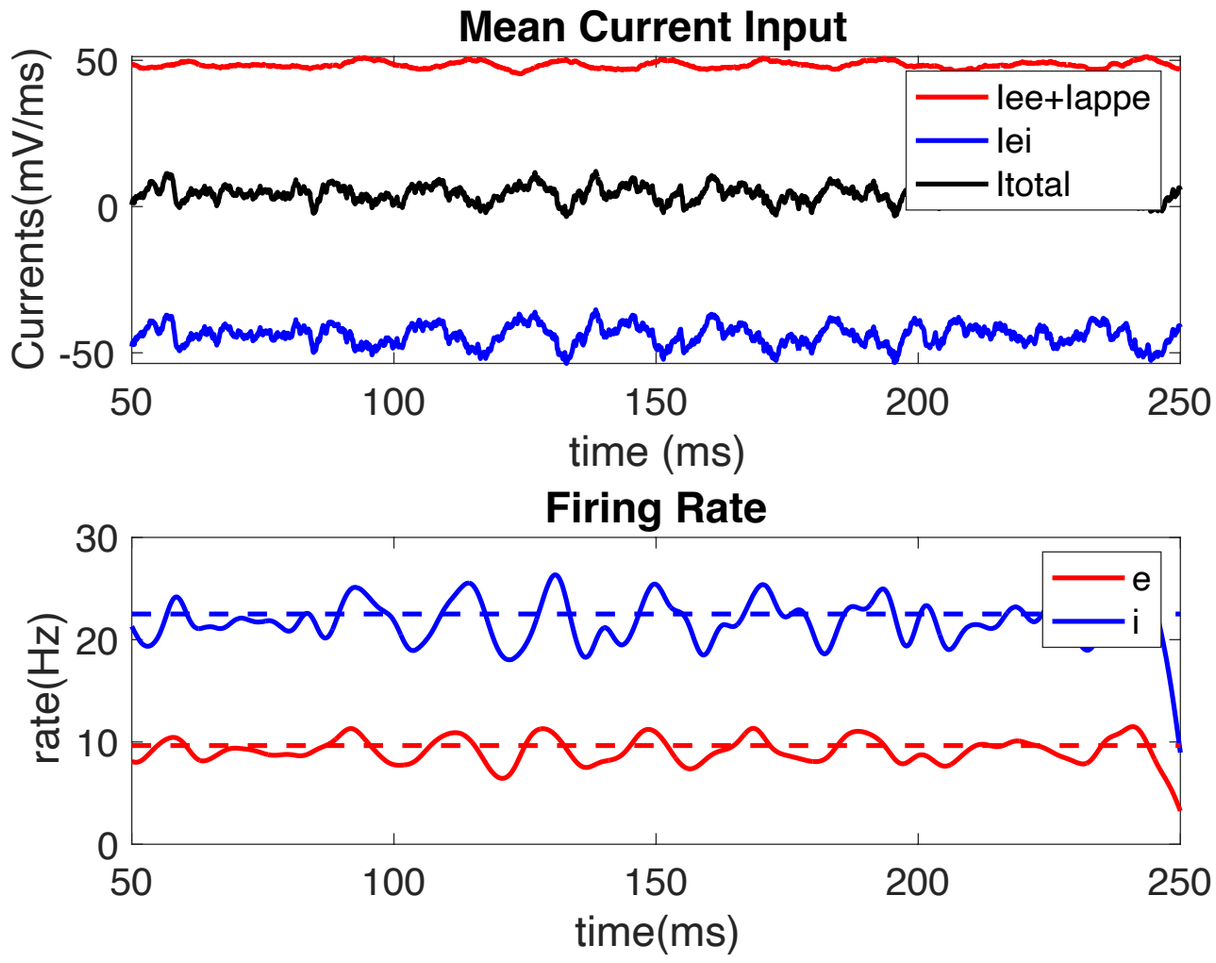


Recurrent Network
(i.e.Cortex)

$$
\begin{cases}
\tau \frac{d\vec{V_e}}{dt} = f(\vec{V_e}) + \vec{\bar{I}}_{ee}(t) + \vec{\bar{I}}_{ei}(t) + \vec{I}_{app,e}(t) \\
\tau_e \frac{d\vec{\bar{I}}_{ee}}{dt} = \vec{\bar{I}}_{ee} + J_{ee}\vec{S}_e(t) \\
\tau_e \frac{d\vec{\bar{I}}_{ei}}{dt} = \vec{\bar{I}}_{ei} + J_{ei}\vec{S}_e(t) \\
\tau \frac{d\vec{V}_i}{dt} = f(\vec{V}_i) + \vec{\bar{I}}_{ie}(t) + \vec{\bar{I}}_{ii}(t) + \vec{I}_{app,i}(t) \\
\tau_i \frac{d\vec{\bar{I}}_{ii}}{dt} = \vec{\bar{I}}_{ii} + J_{ii}\vec{S}_i(t) \\
\tau_i \frac{d\vec{\bar{I}}_{ei}}{dt} = \vec{\bar{I}}_{ie} + J_{ie}\vec{S}_i(t)
\end{cases}
$$

Recurrent Network Models





- **Irregular, asynchronous**
- **Inh. rate > exc. rate**

So far our models are with fixed N, what if N gets large?

Balanced Network Models

"Mean total input stays $\bar{I} \sim O(1)$ via cancellation"

Assumption on the network: p_{ab} and the connectivity strength $j_{ab} \sim O(1)$;

So $J^{ab} = \frac{1}{\sqrt{N}} \begin{cases} j_{ab} & \text{with } p_{ab} \\ 0 \end{cases} \sim O(\frac{1}{\sqrt{N}})$

1. This implies weighted connectivity matrix, $W = \begin{bmatrix} w_{ee} & w_{ei} \\ w_{ie} & w_{ii} \end{bmatrix} = \begin{bmatrix} + & - \\ + & - \end{bmatrix}$

with $w_{ab} = j_{ab} p_{ab} q_b \sim O(1)$, where $q_b = \frac{N_b}{N}$

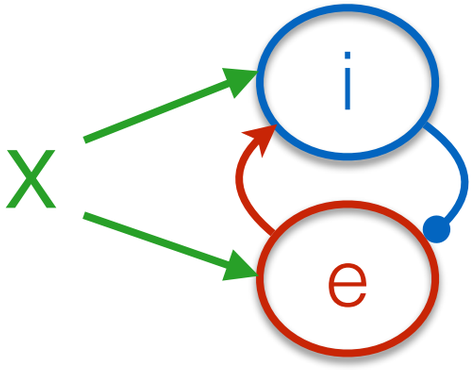
2. Input current, $I_j^a(t) = \sum_{b=e,i,x} [\sum_{k=1}^{N_b} J_{jk}^{ab} (\alpha_b * S_k^b)(t)] = \sum_{b=e,i,x} [\sum_{k=1}^{N_b} \frac{j_{ab}}{\sqrt{N}} (\alpha_b * S_k^b(t))]$

3. After taking time-average, $\bar{I} = \sqrt{N}[w_{ae} r_e + w_{ai} r_i + w_{ax} r_x]$
$= \sqrt{N}[Wr + X]$

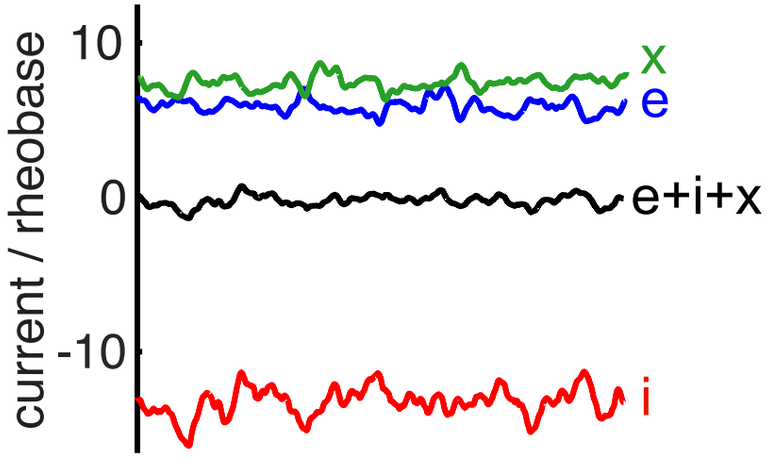
4. In the balanced context, $\bar{I} \sim O(1)$ implies that $[Wr + X] \sim O\left(\frac{1}{\sqrt{N}}\right)$ from cancellation.

Taking the limit as $N \to \infty$, solve for r : $r \approx -W^{-1}X \sim O(1)$

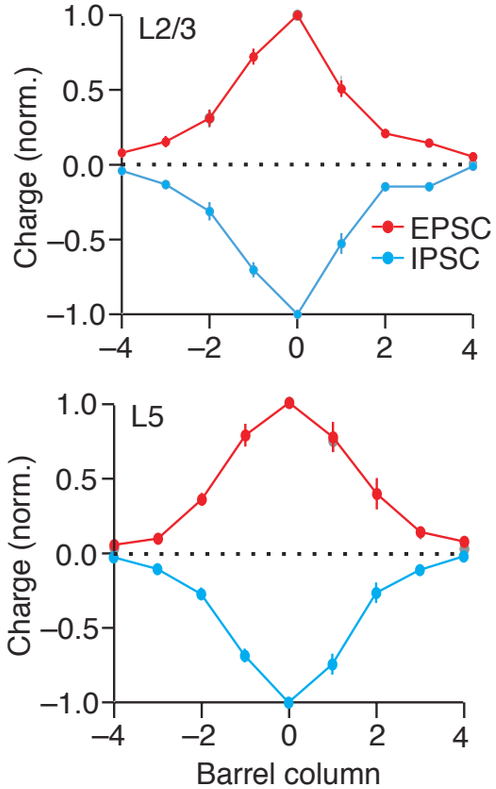
Balanced Network Models



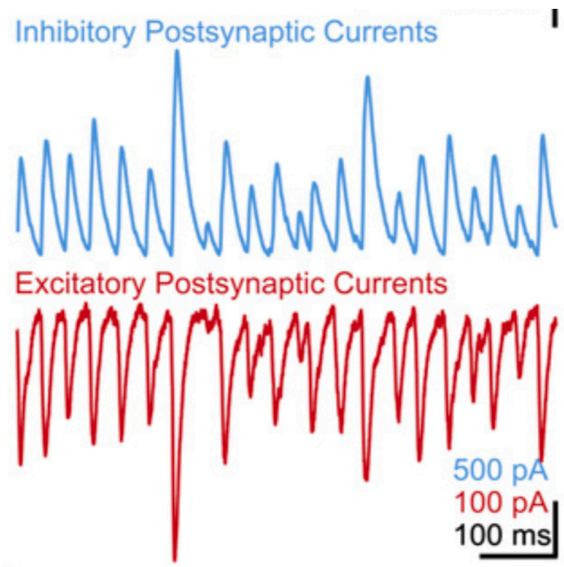
$$\mathbf{r} \approx -W^{-1}\mathbf{X}$$



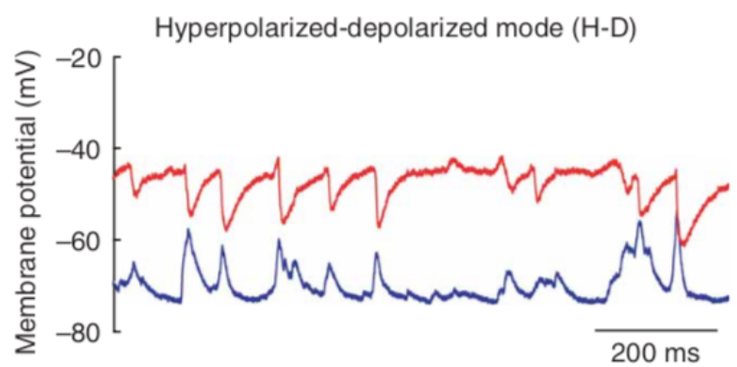
- Approximate E-I balance is widely observed in cortex



(Adesnik and Scanziani, *Nature*, 2010)



(Atallah and Scanziani, *Neuron*, 2009)

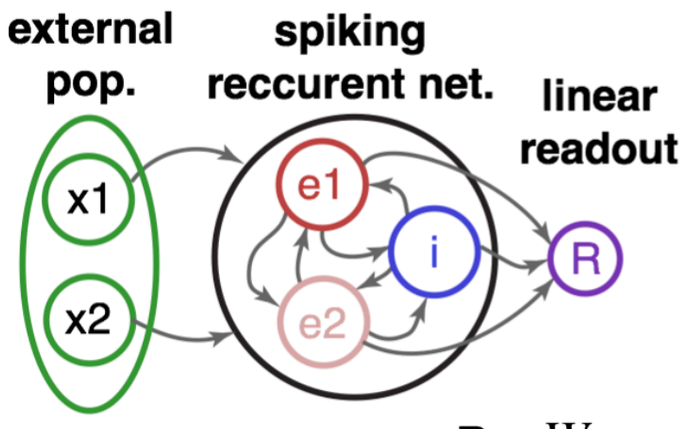


(Okun and Lampl, *Nature Neurosci.*, 2008)

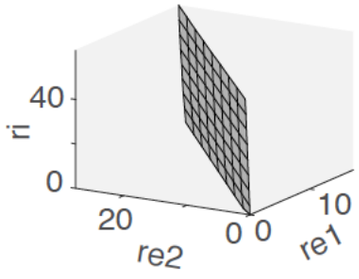
Balanced Network Models

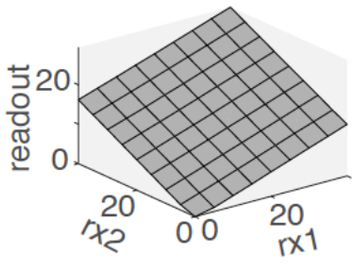
$$\mathbf{r} \approx -W^{-1}\mathbf{X}$$

- **Problem 1:** Rates are a linear function of stimulus:



$\mathbf{R} = W \cdot r$





- **Problem 2:** Parameters must produce non-negative predicted rates:

$$-W^{-1}\mathbf{X} \geq 0$$

Neuron manifold: is an $n_x - dim$ hyperplane in $n - dim$ space

if $n = |\{e_1, e_2 \ldots i\}| \rightarrow \infty$, then **hard** to find $n_x = \{r_{x1}, r_{x2} \ldots\}$satisfying $-w^{-1}X \geq 0$

but cortex performs nonlinear computations!!!

(Baker, Zhu, & Rosenbaum. *bioRxiv,* 2019)

My Work so far : Semi-balanced state

Theorem:
for every W satisfies Dale's law, there exists an $X > 0$ s.t. $r = -W^{-1}X$ has negative entries.

- When balance is broken: $[-W^{-1}X]_k < 0$
    - Excess inhibition to some populations, say e1
    - Remaining populations(e2 &i) form a balanced sub-network

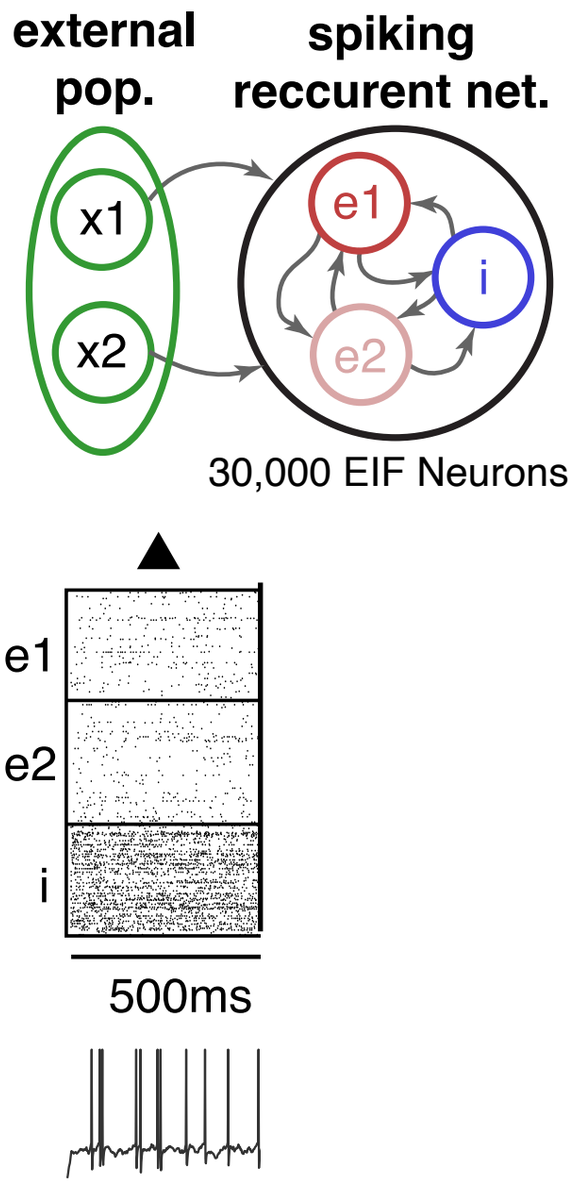
Semi-balanced state

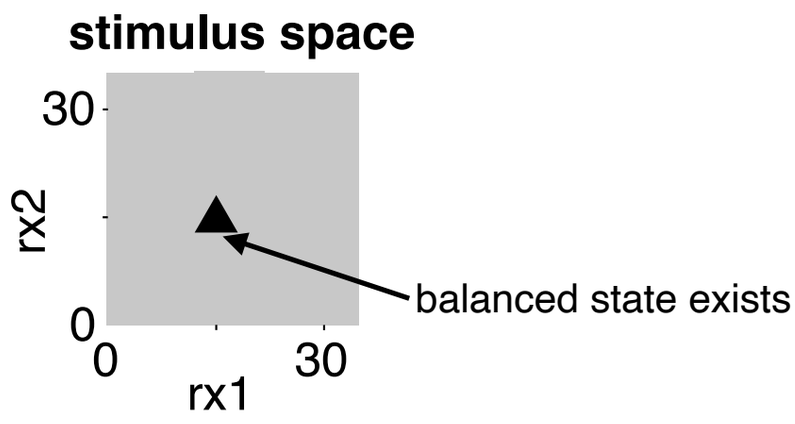
(Baker, Zhu, & Rosenbaum. *bioRxiv,* 2019)

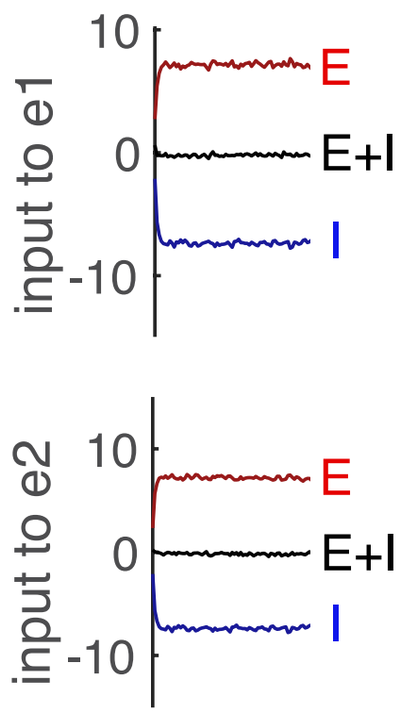
Semi-balanced state

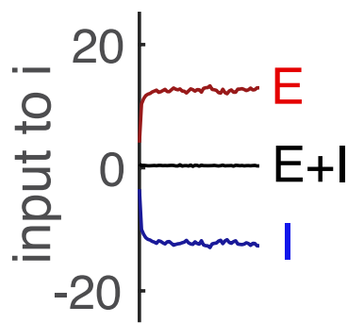
$$\mathbf{r} = [W\mathbf{r} + \mathbf{X} + \mathbf{r}]^+$$

- Non-linear mapping from stimulus to rates!
- More biologically realistic!









(Baker, Zhu, & Rosenbaum. *bioRxiv,* 2019)

Semi-balanced state

$$\mathbf{r} = [W\mathbf{r} + \mathbf{X} + \mathbf{r}]^+$$

- Non-linear mapping from stimulus to rates!
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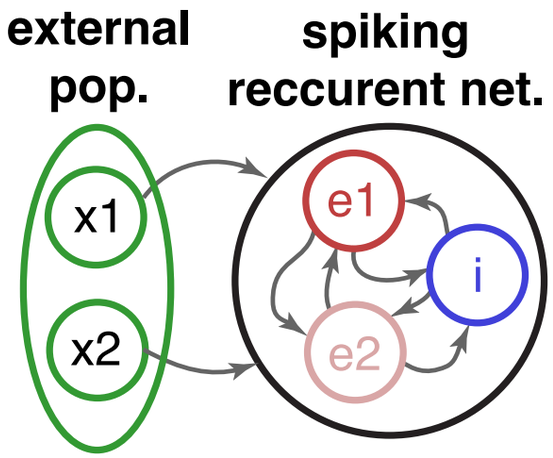
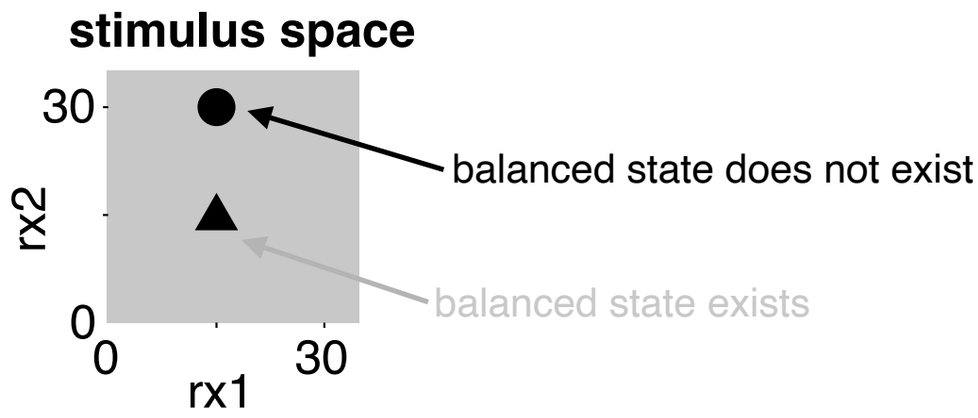
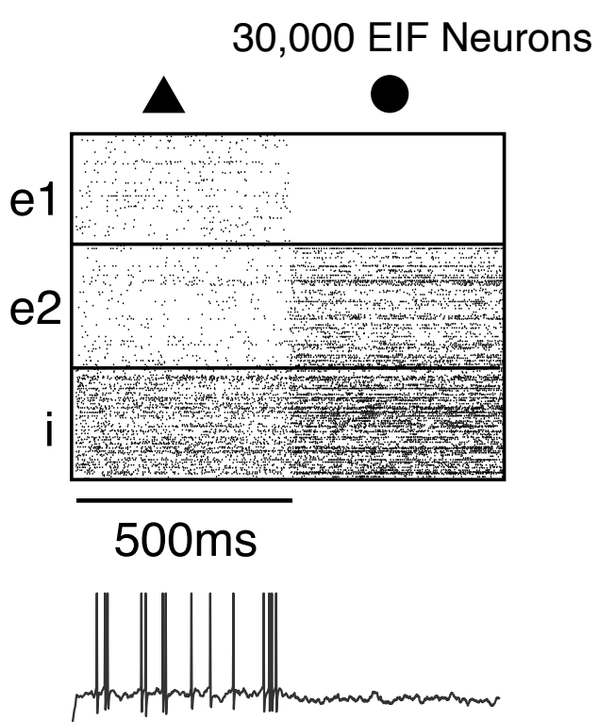
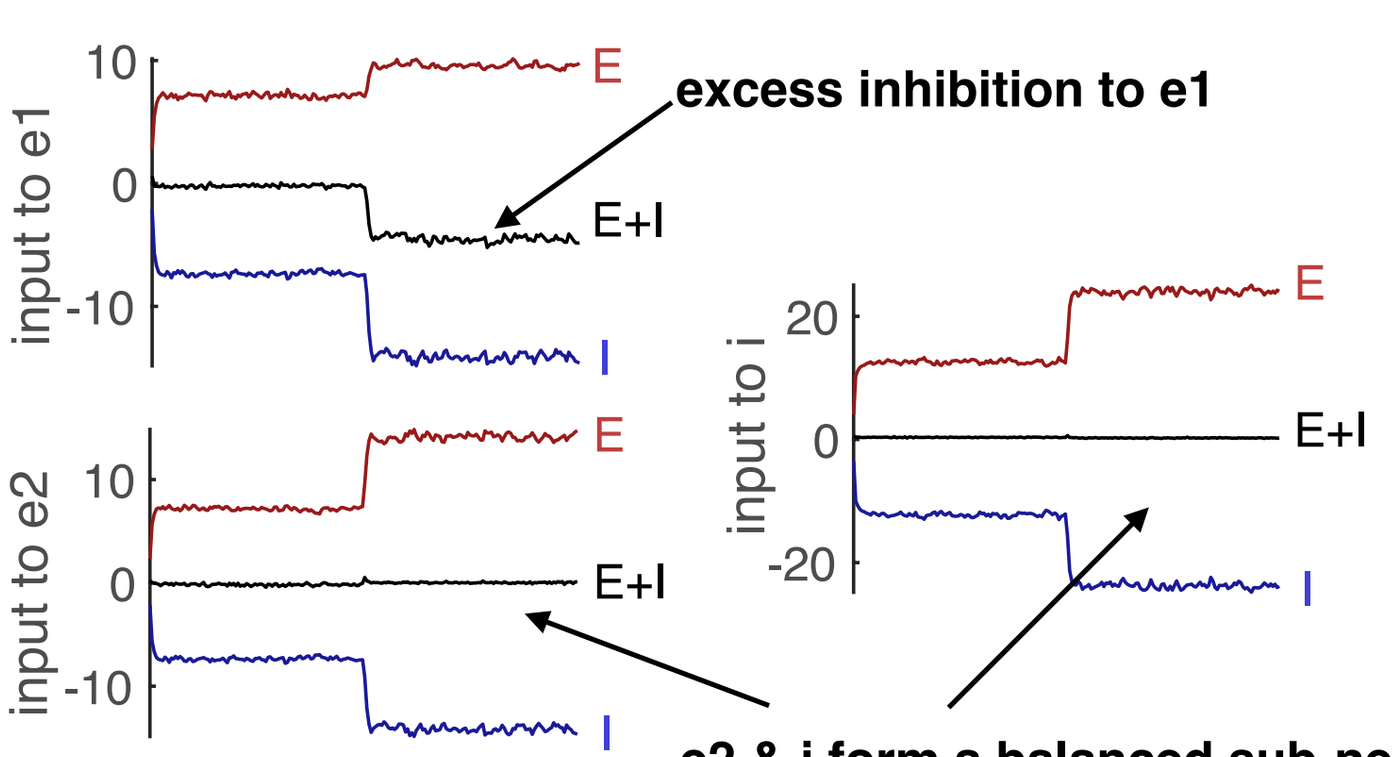




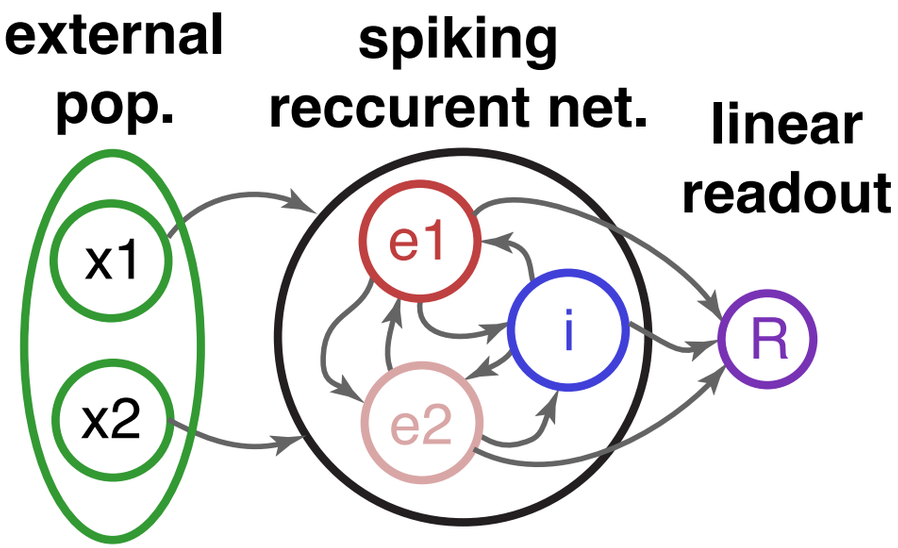




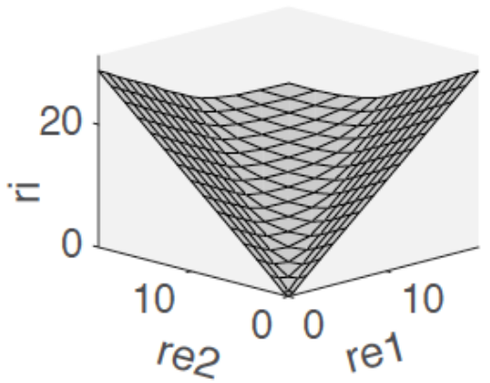
(Baker, Zhu, & Rosenbaum. *bioRxiv,* 2019)

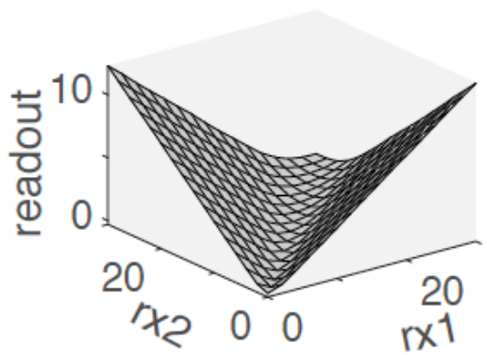
Semi-balanced states

$$\mathbf{r} = [W\mathbf{r} + \mathbf{X} + \mathbf{r}]^+$$



R = $W \cdot r$





It implements nonlinear stimulus representation !!!

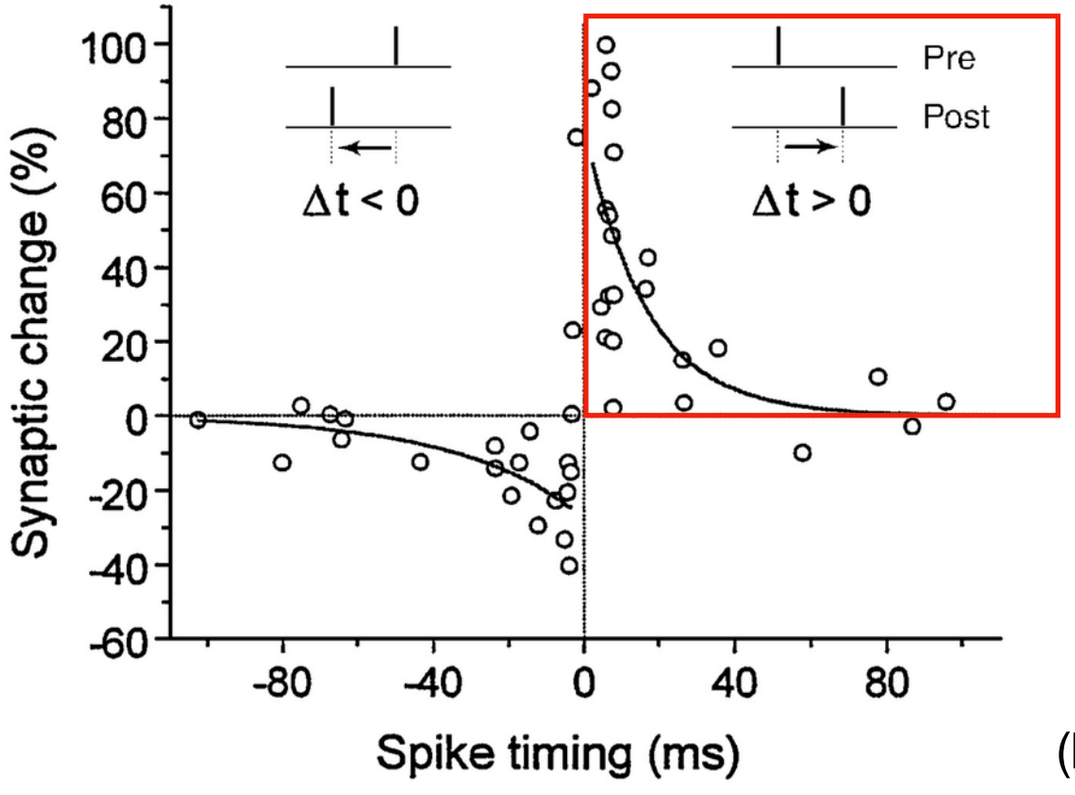
(Baker, Zhu, & Rosenbaum. *bioRxiv,* 2019)

Plasticity

Plasticity: the changes of synaptic weight, ΔJ_{jk}.

(*i.e.* Hebbian rule: “neurons fire together wire together”,)

Spike-timing-depedent Plasticity(STDP): ΔJ_{jk} is related to $\Delta t = |t_j^n - t_k^n|$ with pre-syn neuron(k) and post-syn neuron(j) at n-th spike.



Potentiation: $\Delta J_{jk} > 0$

Depression: $\Delta J_{jk} < 0$

(Experiment, Bi & Poo, 1998)

Our focus:

Implement the STDP rule in **inhibitory** synapses connections: $\begin{cases} \Delta J^{ae} = 0 \\ \Delta J^{ai} \neq 0 \end{cases}$

(Vogels *et al.*, *Science*, 2011)

Plasticity

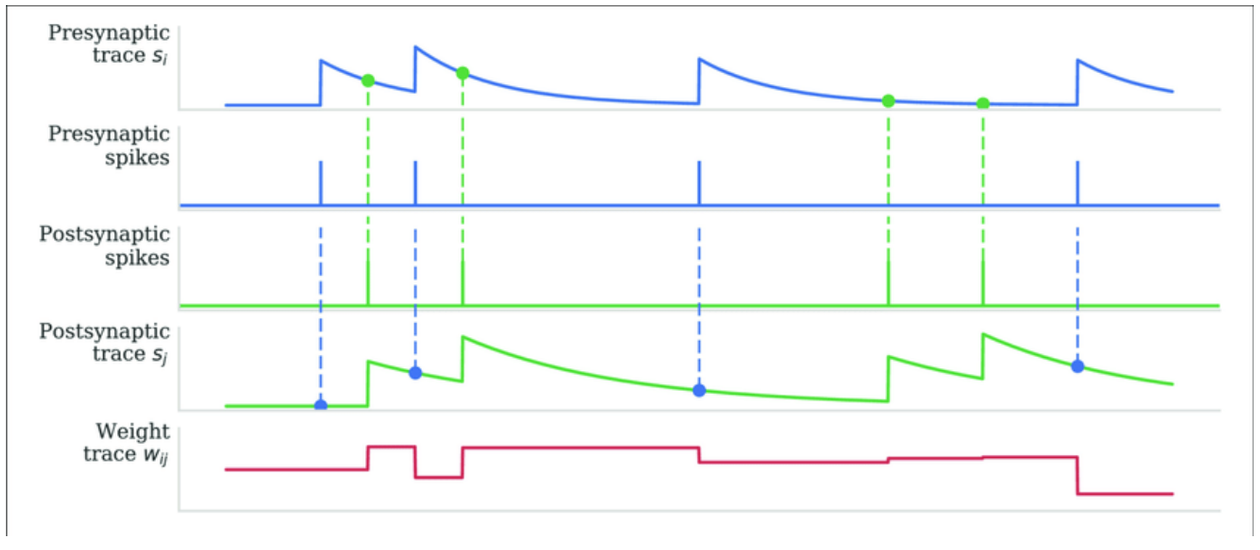
Inhibitory Synaptic Plasticity(ISP): it updates with learning(η_a) and target rate(r_a^0).

$$J_{jk}^{ai} \leftarrow J_{jk}^{ai} + \eta_a J_{jk}^{ai}(x_j^a - 2r_a^0) \quad \text{after each pre-syn spike at time } t_k^n$$

$$J_{jk}^{ak} \leftarrow J_{jk}^{ai} + \eta_a J_{jk}^{ai}(x_k^i) \qquad \text{after post-syn spikes at time } t_j^n$$

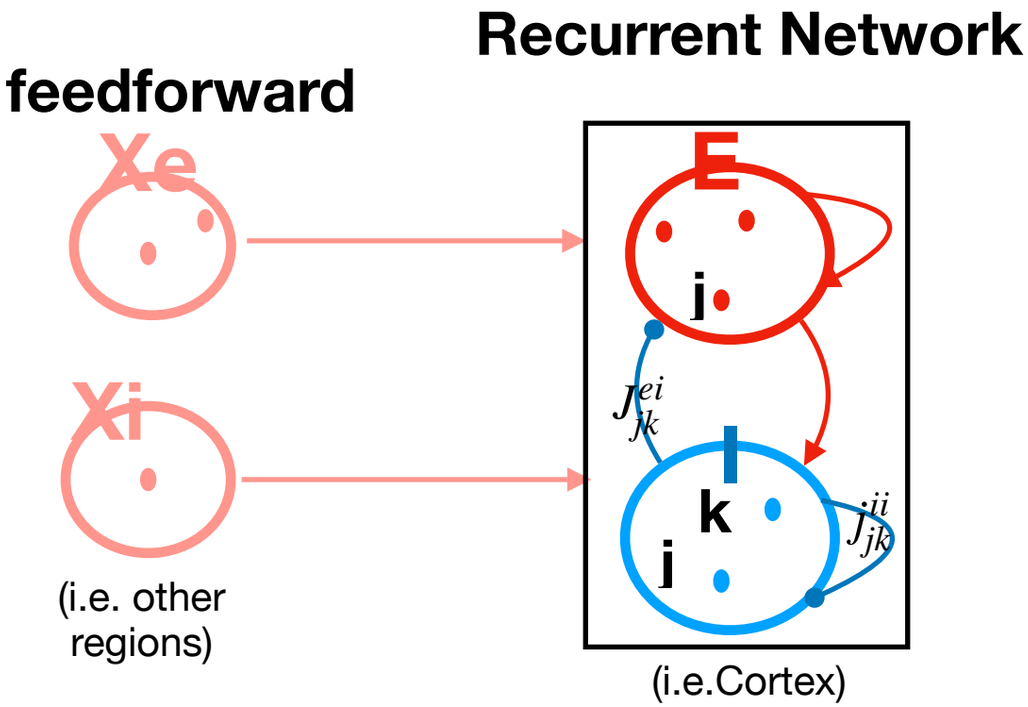
Synaptic trace: to measure the running-average of the firing rate

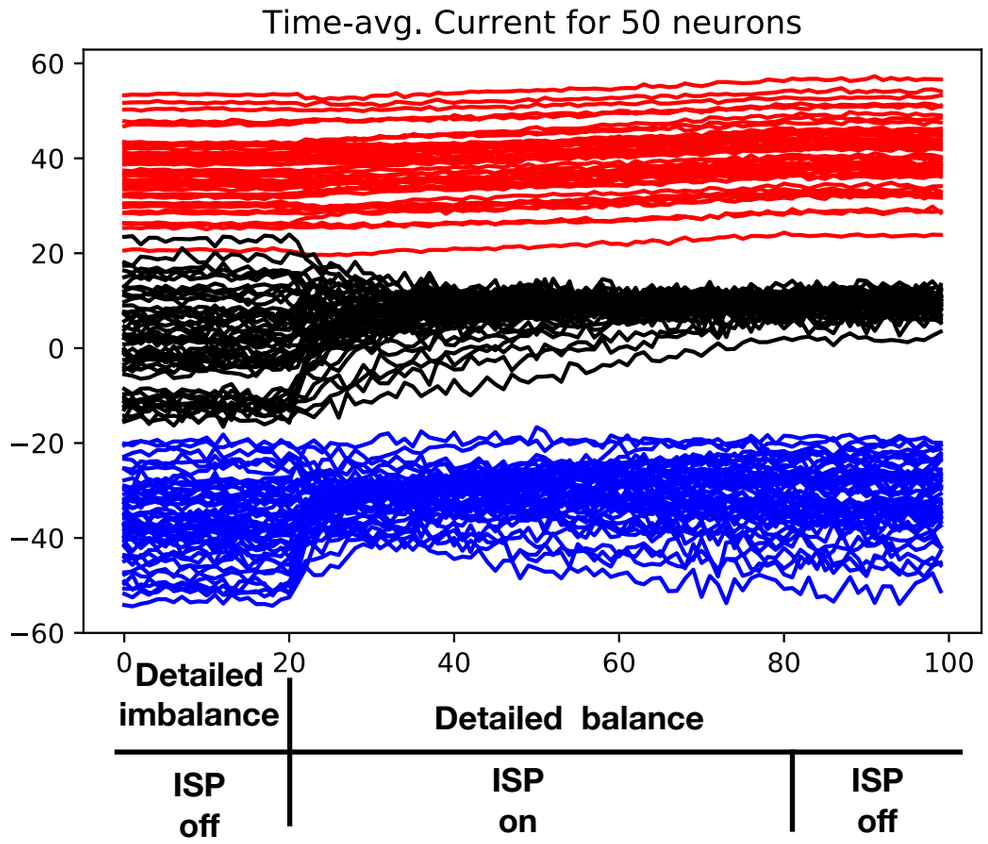
over previous τ_{STDP} window, $x_j^a(t) \leftarrow x_j^a(t) + \frac{1}{\tau_{STDP}}$

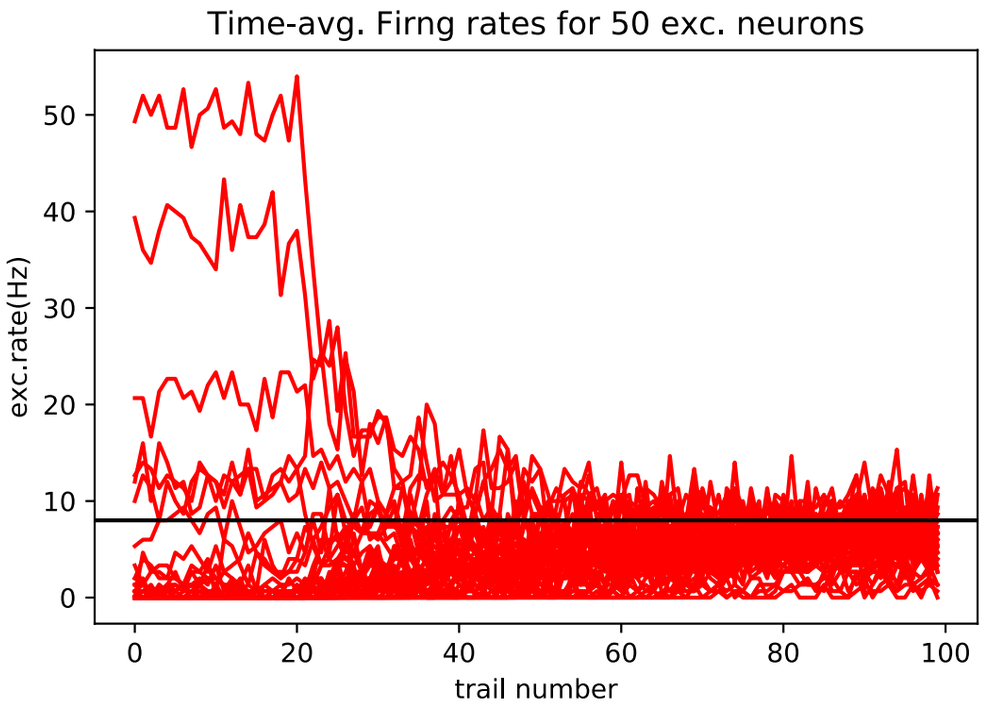


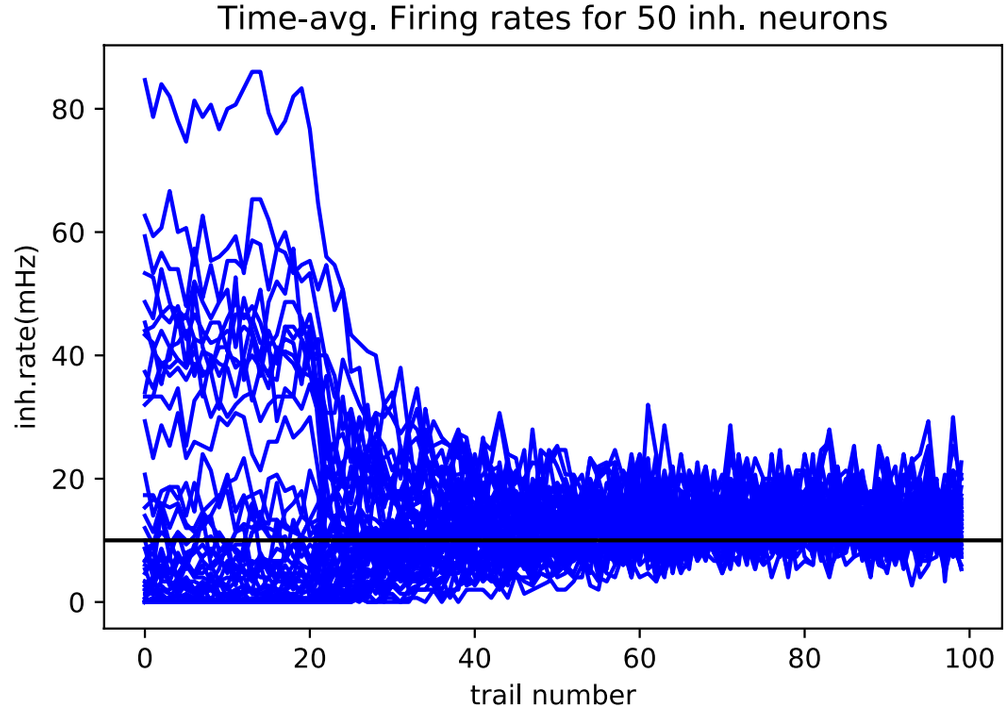
(Morrison et al. 2008)

ISP in spiking EIF models



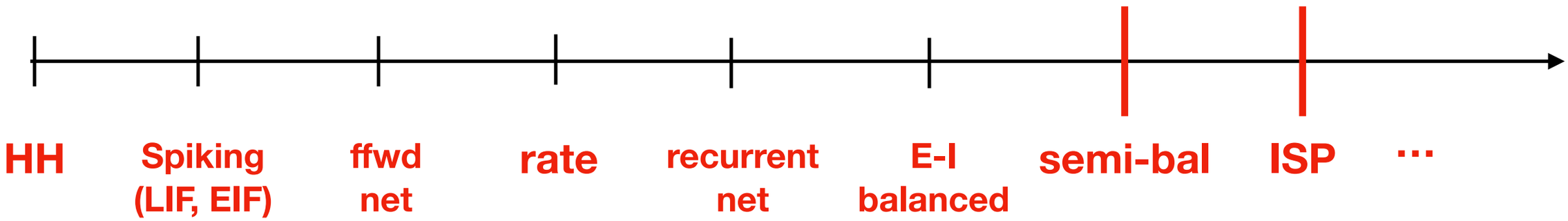






Summary & Discussion :

A brief history of neuronal models:



Some difficulties that I encountered:

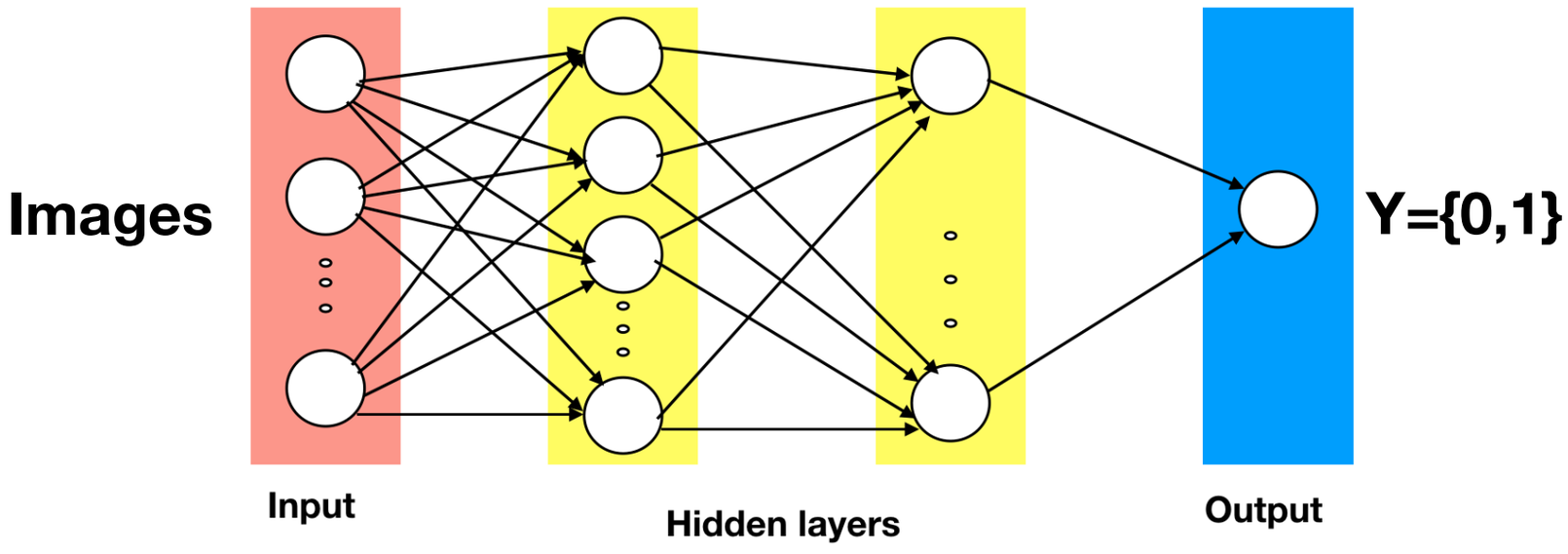
1. Computational cost: use GPU, CuPy lib.
2. Stability analysis of rate model: after dynamics course…
3. Parameter tuning: learning rate is too fast/slow!
4. …

In the future…

1. Understand ISP in rate models :

	Before	During	After
Task 1	X1, X2, X1, X2, …	X1, X1, X1, X1…	X1, X2, X1, X2…
Task 2	X1, X2, X1, X2, …	X1, X2, X1, X2…	X1, X2, X3, X1…
Task 3	X1+Y1, X2+Y2, …	X1+Y1, X2+Y2, …	Xi+Yi, Xi+Y-i, …
…			

2. Connect to Artificial Neural Network & machine learning :



3. Apply Bayesian Models in brain dynamics: predictive coding

…

References

[1] Cody Baker, Vicky Zhu, and Robert Rosenbaum. Nonlinear stimulus representations in neural circuits with approximate excitatory-inhibitory balance. bioRxiv.(2019).

[2] Ian Goodfellow, Joshua Bengio, and Aaron Courville. *Deep learning.* MIT Press.(2016).

[3] Peter Dayan and L.F. Abbott. Theoretical Neuroscience: Computational and mathematical modelling of Neural Systems.MIT Press. (2001).

[4] Vogels, T.P., Sprekeler, H., Zenke, F., Clopath, C., & Gerstner, W. Inhibitory plasticity balances excitation and inhibition in sensory pathways and memory networks. Science 334, 1569-73 (2011).

[5] Wulfram Gerstner, Werner M. Kistler, Richard Naud and Liam Paninski. Neuronal Dynamics: From Single Neurons to Networks and Models of Cognition. Cambridge University Press. (2014).

Thank you!

All the code I use for plotting is available on my website:
https://vickcul.github.io/