Dual Homeostasis: Preserving Information

D.S. Magruder, P. Miller
Volen Center for Complex Systems, Brandeis University, Waltham MA

Requisite for Informative Spikes:

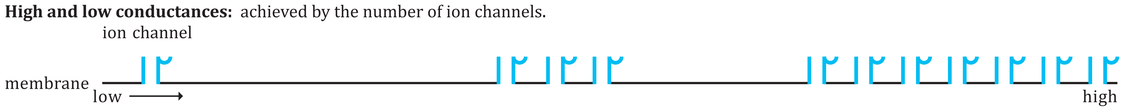
Spiking is a channel for communicating information between neurons. Maintaining a non-zero mean firing rate on a large timescale grants neurons full gamut of this spiking language.

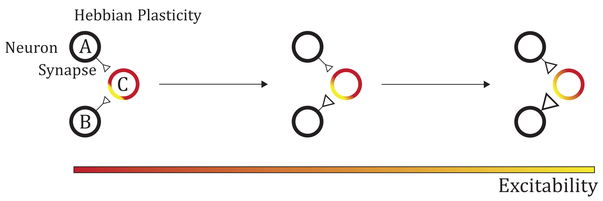
Information is conveyed through fluctuations on a short timescale from either a(n) increase/decrease in firing rate from the mean or in the temporal spacing of the spikes.

Deviation from the Mean

Experiencing continual change, neurons undergo plasticity to adapt. Plasticity alters the excitability of a neuron by affecting conductance, how well current can flow, thus altering its firing rate. Faced with destabilizing forces, like Hebbian plasticity, how can an average rate be maintained on a large timescale?

High and low conductances: achieved by the number of ion channels.



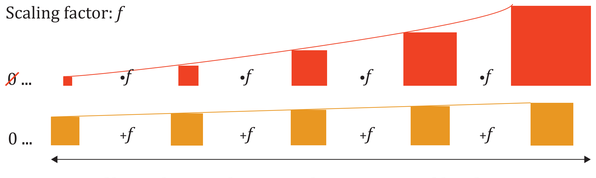


Hebbian plasticity induced instability:
Continual excitation of neuron C by B increases their synaptic efficiency and C becomes more excitable. Eventually this allows A to excite C as well further increasing C's excitability. With no opposing forces, this cycle can continue and destabilize C.

Staying Average, Stabilizing Change:

Understanding the dynamics of how a neuron maintains a mean firing rate when constantly subjected to change is paramount for stabilizing neuronal networks from otherwise epileptic active. Homeostatic plasticity preserves the mean firing rate by opposing destabilizing forces. Where Hebbian plasticity acts on the strength of conductances at specific synapses to achieve apical efficiency, homeostatic synaptic plasticity (HSP) scales the strength of conductance across all synapses to maintain stability. Scaling can occur either additively or multiplicatively.

Additive and Multiplicative Scaling: depending on current size and scaling factor, *f*, either scaling scheme can vary at a faster rate than the other. However, with a $f > 0$ multiplicative scaling will only approach zero, whereas additive scaling can scale the object to zero. Additive scaling therefore includes competition, for small objects may eventually be extinguished. Extinguished objects can recover with additive scaling, whereas with multiplicative an object with zero value will always be zero.



Homeostatic plasticity has also been observed intrinsically, where the conductance of leak currents over the membrane of the neuron are scaled. In this way synaptic and intrinsic homeostatsis can act cooperatively and opposingly i.e. greater synaptic conductance polarizes the neuron towards excitation, whereas greater leak conductance hyperpolarizes the neuron. Homeostatic synaptic plasticity is most likely scaled in a multiplicative manner (Kim et al., 2012, Turrigiano, 2008, van Rossum et al., 2000). Less is known about the nature of intrinsic homeostatic plasticity.

Most theoretical models currently establish stability via a single homeostatic sensor. It is likely that homeostatic plasticity is effectuated via two calcium sensors (Lee et al., 2014, Watt and Desai, 2010 Achard & De Schutter, 2008). Here we investigate the stability requirements of a model with dual homeostatic functions independently modulating intrinsic and synaptic conductances. We aim to establish the benefits and shortcomings of a dual sensor model.

Adaptive Exponential Integrate and Fire Model:

$$C\frac{dV}{dt} = -g_L(V - E_L) + g_L\Delta_T e^{\frac{V-V_T}{\Delta_T}} + I - \omega$$
$$\tau_\omega \frac{d\omega}{dt} = a(V - E_L) - \omega$$

If spike $(V > V_{max})$:

$$\begin{Bmatrix} V \to V_r \\ \omega \to \omega_r = \omega + b \\ Ca \to Ca + 1 \end{Bmatrix}$$

Calcium Equation:

$$\frac{dCa}{dt} = Ca * e^{\frac{-t}{\tau Ca}}$$

Conductance Equations:

$$g_L = g_L + g_L\epsilon_\theta p(f(r_\theta) - f(r_\theta^G))$$
$$g_s = g_s * (1 + \epsilon_s p(f(r_s^G) - f(r_s))$$

Activation Functions:

$$f(r_\theta) = \frac{1}{1+e^{\frac{Ca-C_\theta}{C_{\theta dev}}}} \quad f(r_s) = \frac{1}{1+e^{\frac{Ca-C_s}{C_{sdev}}}}$$

Rate Equations:

$$\tau_r \frac{dr}{dt} = -r + [g_s I(t) - \theta]_+$$
$$\tau_s \frac{dg_s}{dt} = g_s[f_s(r_s^G) - f_s(r)]$$
$$\tau_\theta \frac{d\theta}{dt} = -[f_\theta(r_\theta^G) - f_\theta(r)]$$

Adaptive Exponential Leaky Integrate and Fire model (AdEx-LIF) with Dual Homeostasis:

1.i) Equations governing the AdEx-LIF model as described by Naud et al. 2008 with an exponentially decaying calcium step function.

1.ii) Equations governing activation functions and approximation of conductance differential equation

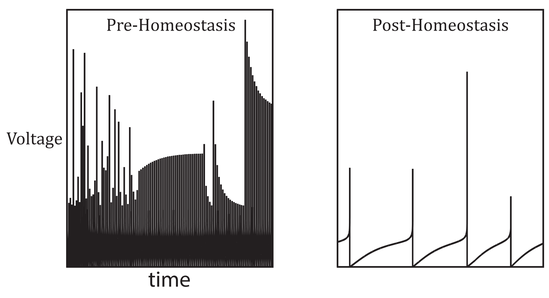
1.iii) Firing Rate equations

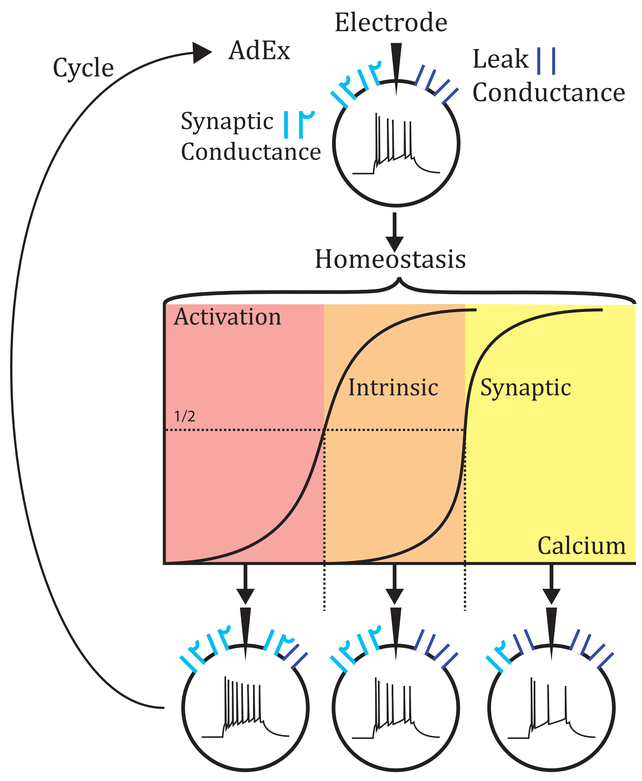
2.i) Graphical representation of AdEx model with homeostasis.

2.ii) Sigmoids depicting homeostasis derived from a firing rate model.

2.iii) Colored ion channels and spike trains represent the changes that occur to the graphic model through homeostasis in response to activation

1.d) The effect of homeostasis on voltage over many cycles.





Summary and Future direction

A first look indicates that dual homeostasis is capable of establishing and maintaining an average firing rate on a large timescale. Our aim is to continue assessing the stability requirements of a single neuron with dual homeostasis and its advantages over a single sensor. Future work may integrate dual homeostasis into network models, as well as include spike-timing-dependent plasticity.

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