580.439/639 Final Exam 2008, Solutions

Problem 1

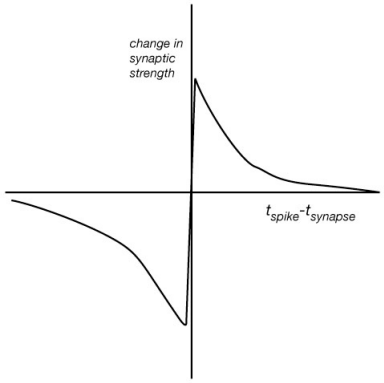
Part a) Ionotropic receptors contain a channel that is directly gated open by activating the receptor. Metabotropic receptors are coupled to a second-messenger system, like a G-protein, that indirectly affects other cellular processes, which can include opening or closing ion channels.

NMDA receptors are ionotropic. They differ from most other ionotropic channels by being conditionally gated through a Mg-block mechanism so to conduct ions, there must be neurotransmitter (glutamate) present and depolarization of the postsynaptic membrane.

Part b) Inhibitory synapses have their reversal potential at E_{Cl} which is in the range of the resting potential. While a -65 mV rest potential is likely to be positive to E_{Cl} allowing hyperpolarizing currents to flow, a -80 mV rest potential may not be. In the latter case the main effect of the synapse could be shunting or the synapse could even give a depolarizing current.

Part c) 1) Cable properties which cause the effect of a synapse to decrease roughly exponentially as the synapse is located further from the soma. 2) The strength of the synapse itself, meaning the conductance of the postsynaptic channels activated. 3) Active properties in the dendritic tree can amplify the depolarization produced by the synapse.

Part d) In STDP, the synapse is strengthened or weakened depending on when it is activated relative to invasion of the postsynaptic space by a backpropagating action potential. The plot at right shows an example. When the synapse is activated before the spike (positive abscissa), the synapse is strengthened and vice-versa. This is Hebbian plasticity in that synapses that are activated before the cell fires a spike are strengthened. The opposite behavior, essentially a reversal of the abscissa, is anti-Hebbian. Both varieties are observed.



Part e) The anti-Hebbian plasticity weakens synapses that are activated before the cell spikes. Presumably those are synapses that are effective in causing spiking, weakening the strongest synapses. The homeostatic plasticity maintains the overall synaptic input, by adding synaptic strength globally when the STDP has weakened the synaptic input too much. The net effect is that synapses over the whole cell are made to be roughly equal in effectiveness.

Part f) A voltage-gated potassium channel is added in parallel with the capacitor and resting conductance. This channel may or may not have a battery in series, depending on what is being modeled. Now the differential equation is

$$C\frac{dV}{dt} = \mathbf{w}^T\mathbf{x} - G_{rest}V - G_K n^p (V - E_K)$$

$$\frac{dV}{dt} = \frac{1}{C}\mathbf{w}^T\mathbf{x} - \frac{1}{\tau}V - \frac{1}{\tau_K}n^p (V - E_K)$$

where the $1/C$ term on the r.h.s. is usually absorbed in the weights. Notice there is now a HH variable n^p, so a second differential equation for n is required. This can be of the usual HH type.

$$\frac{dn}{dt} = \frac{n_\infty(V) - n}{\tau_n(V)}$$

Although it was not part of the question, the threshold device usually operates by producing a pulse at its output Y whenever V exceeds a preset threshold and then resetting V to the resting potential. Refractoriness can be incorporated by resetting V to a negative potential and allowing it to decay exponentially toward threshold or by incorporating an appropriate potassium channel whose n value is increased when a spike occurs.

Problem 2

Part a) K_{ij} computes V_j from the current I_i injected at point i. Given V_j one can compute V_k from the voltage gain A_{jk} as $V_k = A_{jk}V_j = A_{jk}K_{ij}I_i$.

Part b) The voltage gain extends only across a continuous single piece of cylinder. Thus there needs to be separate voltage gains computed between the branch points in the problem cell.

$$K_{ES} = K_{E1}A_{12}A_{2S}$$

In each case, the branch points are handled because they contribute to the load admittance on the cylinder across which the voltage gain is computed.

Part c) Using the result of part B with an additional break at the inhibitory synapse gives

$$K_{ES} = K_{E1}A_{1I}A_{I2}A_{2S} \qquad (*)$$

Looking at the terms one by one:

$$K_{E1} = \frac{1}{(Y_{1S} + Y_{1E})\cosh qL_{E1} + \left(\frac{Y_{1S}Y_{1E}}{G_\infty q} + G_\infty q\right)\sinh qL_{E1}}$$

The load admittance Y_{IS} is the load on the cylinder from E to 1 looking toward the soma; Y_{1E} is the load admittance on the same cylinder looking toward the end of the dendritic tree at E. When synapse I is activated Y_{IS} will increase (because of the synapse) so that K_{EI} will decrease. For the next term

$$A_{1I} = \frac{1}{\cosh qL_{1I} + \frac{Y_{IS}}{G_\infty q}\sinh qL_{1I}}$$

Y_{IS} is the load admittance on the cylinder from 1 to I looking toward the soma. When the synapse is activated, G_{syn} appears in parallel with Y_{IS} so that the denominator of A_{1I} increases, decreasing A_{1I}. The remainder of the voltage gains are not changed, so the net effect is to decrease K_{ES}. Note that the

argument above is for the D.C. steady state, where all terms are real. The same argument applies for complex gains, but has to be argued more carefully.

Part d) The same gain function, equation (*), applies here. However, because the inhibitory synapse is distal to *E* the only factor that is modified when the synapse is activated is Y_{IE} which will be increased when the synapse is activated. There will be no effect on any of the voltage gains. Thus K_{EI}will be smaller when the synapse is activated, giving a reduced gain. The effect should be smaller than in the on-path case, however, because the voltage gains don't change.

Problem 3

Part a) The dot product $\mathbf{w^T x}$ can be written $\mathbf{w^T x} = |w||x| \cos\phi_{wx} = |w|\cos\phi_{wx}$, where ϕ_{wx} is the angle between **w** and **x**. Given that **w** is fixed so that $|w|$ is a constant, the output is a maximum for ϕ_{wx}=0, when **w** and **x** are parallel.

Part b) If *S* is monotone increasing, it will be maximized when its argument is maximized. To find the maximum, differentiate *V*

$$\frac{dV}{d\phi} = -|w|\sin\phi\, S'\left[|w|\cos\phi\right]$$

$dV/d\phi = 0$ only if $\sin\phi$=0, because of the assumption that $S' > 0$. sin ϕ=0 at ϕ=0. Thus the answer doesn't change. Evaluating the second derivative at ϕ=0 verifies that this is a maximum.

Part c) For the two layer network

$$V_1 = \mathbf{w}_1^T\mathbf{x} \qquad V_2 = \mathbf{w}_2^T\mathbf{x}$$
$$V_3 = \mathbf{w}_3^T\begin{bmatrix}\mathbf{w}_1^T\mathbf{x}\\ \mathbf{w}_2^T\mathbf{x}\end{bmatrix}$$
$$V_3 = \mathbf{w}_3^T\begin{bmatrix}\mathbf{w}_1^T\\ \mathbf{w}_2^T\end{bmatrix}\mathbf{x}$$

Thus the two layer linear network is equivalent to a single perceptron with weight vector $\mathbf{w_4}$

$$\mathbf{w}_4^T = \mathbf{w}_3^T\begin{bmatrix}\mathbf{w}_1^T\\ \mathbf{w}_2^T\end{bmatrix}$$

Part d) Given the unit amplitude constraint on the input vector, the output of the nonlinear network can be written as a function of the single variable ϕ which is the angle between the weight vector $\mathbf{w_1}$ and the input **x**. Assuming that there is an angle ϕ_{12} between $\mathbf{w_1}$ and $\mathbf{w_2}$ then the angle between $\mathbf{w_2}$ and **x** is ϕ - ϕ_{12}. Then

$$V_1 = S\left[|\mathbf{w}_1|\cos\phi\right] \qquad V_2 = S\left[|\mathbf{w}_2|\cos\left(\phi-\phi_{12}\right)\right]$$
$$V_3 = S\left[\mathbf{w}_3\cdot\left(V_1,V_2\right)\right]$$

Differentiating V_3

$$\frac{dV_3}{d\phi} = \frac{\partial V_3}{\partial V_1}\frac{\partial V_1}{\partial \phi} + \frac{\partial V_3}{\partial V_2}\frac{\partial V_2}{\partial \phi}$$
$$= -S'\left[\mathbf{w}_3^T\mathbf{v}\right] w_{31} |\mathbf{w_1}| \sin\phi \; S'\left[|\mathbf{w_1}|\cos\phi\right] - S'\left[\mathbf{w}_3^T\mathbf{v}\right] w_{32} |\mathbf{w}_2| \sin(\phi - \phi_{12}) \; S'\left[|\mathbf{w}_2|\cos(\phi - \phi_{12})\right]$$

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In order for an extremum to occur there must be a value of ϕ at which $dV_3/d\phi$=0.