Gatsby Computational Neuroscience Unit
Theoretical Neuroscience

Final Examination
30-31 Jan 2012

Part I

There are five sections with four questions each. Please answer three out of each four, starting the answers for each new section on a new page. Don't forget to write your name at the top of each block of answers.

You have a maximum of 3 hours for this exam.

Good luck!

1 Biophysics

1. Consider a synapse that can both facilitate and depress. The release probability, p, behaves as follows:

   - Every time there is a spike, the probability, p, increases by $f_F(1-p)$ and, with probability p, it also decreases by $(1-f_D)p$. Both factors are relative to the probability immediately before the spike.
   - Between spikes, p decays back to p_0, its equilibrium value in the absense of spikes, with time constant τ.

   Write down a differential equation describing the time-evolution of p. Note that you will need both Dirac delta-functions and random variables.

2. Explain, via biophysical mechanisms, how the weight change of a synapse during learning can depend on *both* pre-synaptic and post-synaptic activity.

3. If you doubled the axial resistance, r_L, in a passive dendrite (assumed to be infinitely long and uniform), what would happen to the electrotonic length?

4. Name one major excitatory and one major inhibitory neurotransmitter in the brain.

2 Networks

1. Assuming the standard Hodgkin Huxley model, show, **by sketching steady-state I-V (current-voltage) curves**, that if the steady state m current, $m_\infty(V)$, activates at too high a voltage, or the steady state h current, $h_\infty(V)$, inactivates at too low a voltage, slowly increasing the voltage will not result in a spike. Note: The steady-state I-V curve is a plot of CdV/dt versus V, with the channels replaced by their equilibrium values (e.g., with m replaced by $m_\infty(V)$).

2. You perform simulations of a large, randomly connected network of excitatory and inhibitory neurons in which the synapses are current-based. The network operates at low firing rates in the asynchronous regime. You notice that a small perturbation to the firing rate decays as

$$\delta\nu \propto e^{-\lambda t}\cos(\omega t)\,.$$

   A friend of yours claims, based on years of experience with the standard Wilson and Cowan equations, that if you were to increase the connection strengths of all the neurons by 10%, both λ and ω would increase by almost exactly 10%, with the increase getting closer and closer to 10% as the network gets larger (assuming you scaled the weights appropriately with network size). Explain why your friend is wrong.

3. To model a 1-memory Hopfield network within the context of the Wilson and Cowan model, you use following equations,

$$\begin{aligned}\nu_{E,i} &= \phi_E\left(K^{1/2}\left[W_{EE}\nu_E - W_{EI}\nu_I\right] + \frac{\beta}{K}\sum_j c_{ij}\xi_i\xi_j\nu_{E,j}\right)\\ \nu_I &= \phi_I\left(K^{1/2}\left[W_{IE}\nu_E - W_{II}\nu_I\right]\right)\,.\end{aligned}$$

   Here ϕ_E and ϕ_I are sigmoidal gain functions (they approach 0 for large negative arguments and approximately 100 Hz for large positive arguments), the Ws are $\mathcal{O}(1)$ weights, $\nu_{E,i}$ is the firing rate of the i^{th} excitatory neuron, ν_E and ν_I are the population averaged excitatory and inhibitory firing rares, $c_{ij} = 1$ with probability K/N and 0 with probability $1 - K/N$, and $\xi_i = \pm 1$, both with probability 1/2.

   Explain why the factor of $1/K$ in front of the sum over j is needed to ensure that the network equilibria are independent of K in the large K limit.

4. Consider a 1-memory Hopfield network with analog rates,

$$\begin{aligned}x_i(t+1) &= \tanh\left(\frac{\beta}{f(1-f)}\frac{1}{N}\sum_{j=1}^{N}(\eta_i - f)(\eta_j - f)x_j(t)\right)\\ \eta_i &= \begin{cases}1 & \text{with probability } f\\ 0 & \text{with probability } 1-f\,.\end{cases}\end{aligned}$$

   Show that in the large N limit, the steady state solution to this equation is $x_i = a + b\eta_i$. Write down explicit equations for a and b. Derive a condition on β such that b can be nonzero.

3 Coding

1. Sketch the form of spike-response current that would lead a GLM model to exhibit bursting, with slow adaptation in the burst rate given constant input. Take care to ensure that the spike trains generated will look biologically plausible.

2. Consider a neuron whose spiking is described renewal process with the ISI distribution

$$p(\tau) = \begin{cases} 0 & \tau < \tau_{\text{ref}} \\ \lambda e^{-\lambda(\tau - \tau_{\text{ref}})} & \tau \geq \tau_{\text{ref}} \end{cases}$$

   Show a typical spike train $\{t_i\}$ from this cell as points along a line, and sketch the corresponding function $u(t)$ which would rescale time so that the rescaled spike events $\{u(t_i)\}$ would be Poisson distributed.

3. Give an example of a neural population that uses a "Cartesian" code. Why do you think that this coding strategy might have evolved in the case you chose—why not a joint distributed code?

4. Consider a neuron that responds to a stimulus s with a spike count n that is Poisson distributed about a Gaussian tuning curve:

$$n \sim \mathsf{Poisson}(f(s)) \qquad f(s) = e^{-(s-\xi)^2/2\sigma^2}$$

   At what value(s) of s would a downstream population monitoring the spike count of this neuron be most accurately able (in principle) to register a small change in s.

4 Systems

1. What happens to connections into the brain if you endow a frog with an extra eye? What would you expect to observe in barrel cortex of a mutant mouse which has an extra whisker?

2. Give at least three reasons why neuromodulators might be well suited to functions such as reward or uncertainty processing.

3. What are the roles of the spinal cord.

4. What sort of linear receptive field would a neuron need to have in order to be direction selective for visual motion patterns?

5 Learning

1. Write down Oja's rule for synaptic plasticity. How would you alter the rule to enforce normalization throughout the evolution of the weights rather than just asymptotically?
2. How might the place fields of hippocampal place cells appear to have their fields grow backwards in space as a function of experience in a linear maze? Justify your answer.
3. What is the difference between direct and indirect actors? Could one tell them apart behaviourally?
4. Use arguments based on probability theory to justify the existence of three factors that could control the rate of learning in a conditioning experiment.