Gatsby Computational Neuroscience Unit
Neuroscience Candidacy 2013

Written Examination
30 Jan 2013

Part I

This part has 20 short questions. Answer all of them, to the best of your ability. Each is worth 4 marks. No reference materials are allowed.

It should take no more than 3 hours.

1. Why is the potassium current in the Hodgkin Huxley model often called a delayed rectifier?

2. What is the 3/2 power law for dendrites? If dendrites obey that law, why does that make it easy to determine the voltage at the tips of the dendrites given the voltage at the soma, but not the other way around?

3. Sketch steady-state I-V curve for NMDA channels with and without magnesium present.

4. A typical EPSP associated with an AMPA synapse is 0.5 mV. Approximately what conductance change does this correspond to? You should make a back-of-the-envelope calculation; within a factor of about 2 is fine. Use reasonable parameters for the synaptic and membrane time constants, membrane resistance, reversal potentials, etc.

5. Consider a bump-attractor network with spike frequency adaptation,

$$\tau \frac{d\nu_i}{dt} = \phi\left(\sum_j W_{i-j}\nu_j - a_i\right)$$
$$\tau_a \frac{da_i}{dt} = c\nu_i - a_i$$

   where ϕ is a sigmoidal function of its parameters and W_{i-j} is bell-shaped and symmetric with respect to i and j; e.g., $W_{i-j} \propto \exp(-(i-j)^2/2\sigma^2)$. Assume parameters are such that when $c = 0$ the equations admit a stable hill of activity. Argue qualitatively that for large enough c the equations admit a moving bump.

6. Low dimensional dynamics. Suppose a network evolves according to the equations

$$\tau \frac{d\nu_i}{dt} = \phi\left(\frac{1}{N}\sum_{j=1}^{N}\sum_{\mu=1}^{p}\xi_i^\mu \xi_j^\mu \nu_j\right) - \nu_i$$

   The ξ_i^μ are random vectors but, unlike with the Hopfield network, they are correlated. Moreover, also unlike with the Hopfield network, p is small (although you won't need that fact). Let

$$x_\mu = \frac{1}{N}\sum_i \xi_i^\mu \nu_i \,.$$

   Show that x_μ evolves according to

$$\tau \frac{dx_\mu}{dt} = \psi_\mu(x_1, x_2, ..., x_p) - x_\mu \,.$$

   Write down explicit expressions for the ψ_μ.

7. You do an experiment in which you inject a small amount of current into most of the inhibitory neurons in an isolated network of spiking neurons (something that is entirely feasible these days). You find that the inhibitory firing rate goes up and the excitatory firing rate goes down. Is the network operating in the balanced regime? Explain your reasoning.

8. Consider a network of neurons in which the steady state firing rates of the individual neurons (ν_{Ei} and ν_{Ii}) are given by

$$\nu_{Ei} = \phi_E\left(W_{EE}\nu_E + I_i^E, W_{EI}\nu_I\right)$$
$$\nu_{Ei} = \phi_I\left(W_{IE}\nu_E + I_i^I, W_{II}\nu_I\right)$$

   where ν_E and ν_I are the population averaged excitatory and inhibitory firing rates and I_i^E and I_i^I are external currents. As usual, all the W's are positive and both gain functions, ϕ_E and ϕ_E, are non-negative, increasing functions of their first argument, and decreasing functions of their second.

   You don't know the ϕ's or the W's, but you have complete control over the external currents (I_i^E and I_i^I), and you can measure the firing rates of all the neurons in the network. Explain how to use this to empirically determine $\phi_E\left(W_{EE}\nu_E, W_{EI}\nu_I\right)$ and $\phi_I\left(W_{IE}\nu_E, W_{II}\nu_I\right)$.

9. A scalar stimulus that is uniformly distributed in a fixed range (say $[0, 1]$) is encoded in the firing rate of a neuron. Sketch a (pathological) tuning curve for which the average Fisher information conveyed by the firing rate may be very large, but the average mutual information very low. Is the reverse possible: can the mutual information be high, whilst the Fisher information is very low?

10. For what types of population code is the "population vector" an optimal decoding strategy? Try to be as comprehensive in your characterisation as you can.

11. Object-tuned cells in IT are often said to have "position-invariant tuning". However, their firing rates do change as a visual stimulus is moved to different parts of visual space. So what does "position-invariant tuning" mean? Describe briefly two different theories for how it might arise.

12. A scalar stimulus that is uniformly distributed in a fixed range (say $[0, 1]$) is encoded in the firing rate of a neuron. Sketch a (pathological) tuning curve for which the average Fisher information conveyed by the firing rate is very large, but the average mutual information is low. Can you give an example of the opposite: a high average mutual information but low Fisher information? Justify both your answers.

13. Compare the phenomena of adaptation and after-effects in motor control and perception. Do you think similar neural adaptive mechanisms might act within both systems?

14. What is a "grid cell"? Where in the brain are they found? Sketch the sort of response property that characterises this class of cell.

15. Describe the anatomical connections between the various regions of the striatum and the dopamine system.

16. Define at least four computationally distinct forms of *attention*.

17. How might associative plasticity work in synapses that lack NMDA receptors?

18. Describe empirical evidence in favour of synaptic tagging.

19. Briefly describe some similarities and differences between developmental and adult plasticity.

20. Describe the phenomenon of reconsolidation. What challenge does it pose to standard models of consolidation?