Gatsby Computational Neuroscience Unit
Theoretical Neuroscience

Final Examination
30-31 Jan 2013

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. In the Hodgkin-Huxley equations, the time constant for the m-channel is much smaller than that of the h-channel. Why is that?

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

3. In voltage clamp recordings in which voltage is clamped at rest (around -65 mV), EPSPs show up as a downward deflection in current and IPSPs show up as an upward deflection. Why is that?

4. Approximately how much charge flows into a cell during an action potential? Consider only the time when the neuron goes from rest to its peak voltage. Assume that the neuron has a membrane resistance of 100 MΩ and its membrane time constant is 10 ms, and it is compact enough that the voltage is about the same everwhere inside the neuron. (Hint: remember, $Q = CV$.)

2 Networks

1. Explain why oscillations are ubiquitous in the brain, and why they tend to be relatively broadband.

2. The total conductance, $G_i(t)$, associated with the input from presynaptic neurons is

$$G_i(t) = \sum_j W_{ij} g_j(t)\,.$$

   Here $g_j(t)$ is the individual conductance associated with presynaptic neuron j; assume it evolves according to.

$$\frac{dg_j}{dt} = -\frac{g_j}{\tau_s} + g_0 \sum_l \xi_{ij}^l \delta(t - t_j^l)$$

   where t_j^l is the time of the l^{th} spike on neuron j, ξ_{ij}^l is 1 with probability p_{ij} (indicating release) and 0 with probability $1 - p_{ij}$ (indicating failure). Write down an expression for the time average of $G_i(t)$.

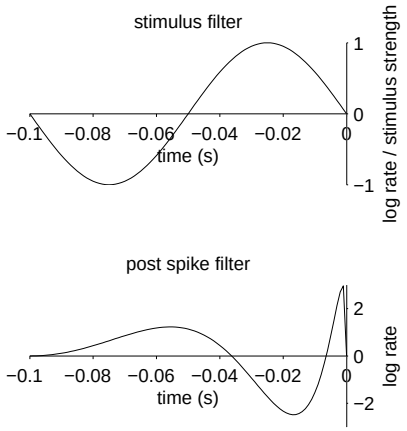
3. You do an experiment in which you inject a small amount of current into most of the inhibitory neurons in an isolated network of spiking neurons (something that is entirely feasible these days). You find that the inhibitory firing rate goes up and the excitatory firing rate goes down. Is the network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

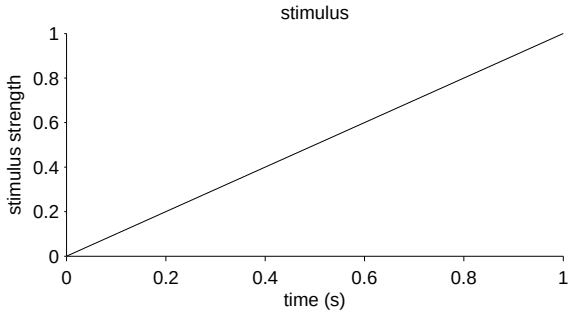
4. A large, isolated network of randomly connected spiking neurons is firing steadily at low rate – well below the maximum firing rate of the individual neurons in the network. You do an experiment in which you first measure the firing rate of all the inhibitory neurons, and then dynamically clamp every one of them so that they fire at the rate you just measured (independent of whatever is happening in the network). What happens to the firing rates of the excitatory neurons? (Hint: there are two things that could happen.)

3 Coding

1. The simplest model of neural encoding has each neuron's spikes drawn from an inhomogeneous Poisson process whose rate is determined by the stimulus and the cell's response function. Give three biophysical or experimental observations that this model **cannot** account for.

2. Consider a GLM neural model with an exponential nonlinearity, conditionally-Poisson spiking, and stimulus- and post-spike-filters as drawn on the left below.





Draw an example of the sort of spike train it might generate given the ramp stimulus shown on the right. Also show the estimate of the log-intensity from which your spike train was derived. You may ignore the transient at the onset of the stimulus.

3. 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. Is the reverse possible: can the mutual information be high, whilst the Fisher information is very low? Justify both your answers.

4. 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.

4 Systems

1. What is the difference between "cell-attached" and "whole-cell" recording? What sort of information does each provide?

2. What is the "complex logarithmic" retinotopic mapping? Where is it found? What purpose might it serve?

3. Explain, with reference to specific neural mechanisms, why text on a projected slide may look black even though the screen looks white and the projector is unable to take away light incident from ambient sources.

4. Object-tuned cells in IT are often said to have "position-invariant tuning". However, their firing rates do change as the visual stimulus is moved to different parts of visual space. So what does "position-invariant tuning" mean?

5 Learning

1. What is subtractive normalization? What consequence does it have for Hebbian development/learning?
2. If the effect of taking an addictive drug was a phasic release of dopamine in target structures, what effect on predictions and actions would we expect based on standard reinforcement learning?
3. What is the difference between Q-learning and SARSA? What effect does this have on the way they work? How could one tell them apart physiologically?
4. How distinct are supervised, unsupervised and reinforcement learning? Justify your answer.