COGS 180

HW 2

1. For the LGN, which structures supply (a) feedforward, (b) feedback, and (c) recurrent input? Why is it categorized this way?

   LGN neurons get feedforward inputs from retinal ganglion cells which are driven by signals that are caused by external stimuli, and interneurons which are inhibitory neurons activated by retinal cells. Depends on the retinal input, these cells supply feedforward inhibition to LGN, in which the inhibition can either generate temporal diversity or serve as “a form of lateral inhibition” (p. 138). LGN receives feedback information from multiple downstream structures, including visual cortex and brainstem, where the former acts to implement “graceful degradation” and the latter has the function of gating state and arousal (p. 136). Both inhibitory interneurons and thalamic reticular nucleus (TRN) are contributing to a “local recurrent inhibitor loop,” where LGN relay is recurrently inhibited by the activation of projection neurons (i.e. neurons driven by the retina) (p. 136). Recurrent input like this “allows for sign reversal” and add intrigue to the circuits. (p. 136). It is categorized by the directions of information flow (i.e. bottom-up and top-down).

2. How is the efficacy of the retinogeniculate drive defined? How does it depend on the animal's sleep/arousal state? Why does it almost never reach 100% in awake behaving animals?

   The efficacy is defined as “the ratio of LGN spikes/retinal spike” (p. 143) or can be understood as the extent to which the LGN is responding or ignoring the retinal retina (p. 136). It is suggested that in a sleep cycle, efficacy increases as the animal move from
slow-wave, REM and to wakefulness (p. 143). When the animal is alert, factors such as arousal, as well as attention, can further increase efficacy. However, even so, the efficacy never reaches 100%, because the LGN is doing computations or filtering with the information coming from the retina and such computations always come with information lost. If the efficacy is 100%, essentially there will be no need for LGN when we have retina.

3. Can LGN provide more visual information than the retina? Why or why not?

   LGN does not provide more bottom-up “visual information” than the retina, in the sense that retina is the only place in the brain that directly obtains visual information from the external world. In other words, information from the retina is the upper bound of visual information that LGN can get. LGN does get contextual information from higher structures, such as cortexes and the brainstem. However, these information does not necessarily has to be within the visual modality. Most of the time the top-down information only facilitates/interferes the processing of visual information, with the exceptions of cases like visual hallucinations.

4. Why is it useful for the LGN to provide "efficient access... to selective populations of retinal groups" (P. 2)? Give one example.

   Because LGN can get contextual information from top-down, information can be functionally segregated as early as LGN. This contextual modulation allows a faster behavioral response and the attentionally selected information is easier for the downstream structures to encode than the raw information. This is useful especially when we have a particular behavior goal, such as locating a particular feature in the visual scene. For example, if we are looking for a green object in the room, this contextual information will
help us allocate a green object faster than it would have if we do not have this contextual information.

5. (Extra credit) What does it mean that LGN cells have higher gain at low contrast, and lower gain at higher contrast? What function does it serve and why?

   It means that as high sensitivity compensates for low contrast, and low sensitivity is compensated by high contrast, the resulted outputs are normalized (as sensitivity becomes more evenly distributed). It functions this way so that LGN cells can better response to local conditions (i.e. texture and structure) instead of constantly adapting to absolute conditions (i.e. brightness) (p. 142). In some sense, contrast gain control is trading resolutions for reduced noise, as the effect of noise is also scaled with the gain.

![Diagram](image)

6. (Extra credit) How do lagged and non-lagged cells differ in their anatomical connections from the retina? What differential function might they serve (see slide 10)?

   Non-lagged cells receive inputs directly from the retina while lagged cells are connected to the inhibitory interneurons that are driven by the same retinal input. It is also suggested that the axons that lagged cells project to cortex have slow conducting rate (Mastronarde,
Given the activities profiles (slide 10), it seems that non-lagged cells are more sensitive to an immediate change in the environment (which elicits the observed burst of activities), and lagged cells are better at registering the on-going activities happening in the background.

7. (Extra credit) Why might there be inter-species differences in how much multiplexing or integration there is in the LGN (as opposed to downstream in the visual pathway)?

Multiplexing is common in cats, but minimal in monkeys. It may have something to do with the brain size of the animals. The monkeys have bigger brain size that can afford more downstream computation. This is preferable in the sense that there is less information lost from integration, and there is less loss of independency among signals (which can also be seen as information). In contrast, it may be more evolutionarily beneficial for cats to have more information computed early in the visual process, as their heavily rely on their vision for detecting preys and their activities in dim light.
Reference

II. Retinal inputs and the generation of receptive-field properties. *Journal of Neurophysiology, 57*(2), 381-413.