Questions for Van Versendaal et al., 2012

- Van Versendaal et al. (2012), along with another study published in the same issue of *Neuron,* were the first to image inhibitory synapses in live animals, as opposed to fixed tissue or cultured neurons. (see questions below)
    - How were inhibitory synapses labeled in Van Versendaal et al. (2012)?
    - What control experiment did the authors do to confirm that the labeled objects were inhibitory synapses?
    - Did the authors do any additional control experiments to check whether their labeling approach perturbed the cells/synapses?
- List one central claim in Van Versendaal et al. (2012).
    - Which figure provides evidence for this claim?
    - Are you convinced by the evidence provided?

Questions for Van Tan et al., 2012

1. What do we refer to as homeostatic scaling? What type of studies have contributed to our understanding of homeostatic regulation of neuronal activity?

2. What is the significance of the new method presented in the paper to monitor AMPA receptors in live mice? How are they accomplishing this task? Please be specific.

3. Why do you think AMPA receptors are more dynamic in primary visual cortex (V1) Layer 2/3 neurons compared to Layer 5? What is the data supporting this claim? How do you explain this phenomenon?

4. In Figure 3, the authors claim that binocular enucleation induces an initial decrease in AMPA levels followed by a recovery only after prolonged deprivation? What do you think there is a delay in recovery? Name additional experiments that would further validate this claim.

5. The authors claimed that GRIP1 is in part responsible for the increase in AMPA levels following deprivation despite showing opposite results in the GRIP1 knockout mice. Do you agree with their conclusion? What is an alternative interpretation of their results that could explain a decrease in AMPA levels in the GRIP1 knockout?

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Fall 2022