

During the course of the discussion, the main topic for discussion was the separation of analogue and digital signalling. It was understood by the majority of the class that analogue signalling was represented (by the paper) as a sub-threshold stimulation of membrane potential, whereas digital signalling was represented by the paper as a stimulated action potential; however, the issue raised was regarding distinguishing the two from one another, and under what circumstances could signalling within the brain be represented by either one or the other (exclusively).

It was stated that, even though an analogue signal was represented by a sub-threshold stimulation, at the microscopic level, the analogue signal could be seen as an infinitesimal sum of digital signals over the entire course of the stimulation. This raised the question regarding what size/dimensional constraints could this analogue-digital facilitation be analyzed under. Personally, it was initially believed that the goal of the paper was to base its hypothesis/argument within a localized region in space wherein the analogue signal and digital signal could be distinguished from one another (and/or ultimately complement each other); however, under extremely small sizes, analogue-digital facilitation is ultimately grounded within the digital scope of things.

Due to the outcome of this discussion, it was generally understood post-discussion that one of the key arguments to the existence of analogue-digital facilitation was the ability of the hippocampal region within the brain to distinguish whether to use analogue signalling (in the form of neurotransmitter release/diffusion mechanism under short distances) or digital signalling (over long range information transmission). This idea was the key foundation in regards to the overall function of the hippocampus: inhibition (alternating states of distraction and attention), memory (encoding, storage and retrieval of collective and individual information), and space (analysis and decoding of information from the immediate spatial environment).

Another key idea brought forth during the discussion was the comparison between the Hodgkin-Huxley model and the mechanisms of hippocampal signalling. Initially, it was stated that regardless of how different the Hodgkin-Huxley model was (example – potassium channels within the HH model are the opposite to that hypothesized to be inactivated within the hippocampal region, ie. slow-activating and fast-inactivating in HH versus fast-activating and slowly-inactivating in hippocampus), the same fundamental ideas could be used to analyze how the overall circuitry of the system functioned. One idea that was proposed during the discussion was a modification to the Hodgkin-Huxley model, in which the potassium gate channels would be represented by multiple channels with varying properties as opposed to four subunits with similar properties (the “n” gates).

Overall, it was in agreement with the general audience that every possible explanation brought forth by the paper in regards to increased neurotransmitter release/increased excitatory postsynaptic response merely gave a brief overview of the reasoning without any actual analysis regarding the finer details. It was understood though that, regardless of how the paper was written or who it was written for (aimed at a scientific audience), it was primarily written as a piece of secondary literature, which, by nature, is characterized as being a well-concise summary of many published primary papers.