Playing tennis with the cerebellum

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A functional imaging study in which subjects tracked different targets with eye movements and a joystick provides evidence that the cerebellum is involved in eye–hand coordination. The data suggest that internal models used for motor control may also be involved in cognition.
The diversity of synaptic plasticity

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In contrast to the hippocampus, low-frequency stimulation in the amygdala produces synaptic enhancement via kainate receptors that spreads to inactive synapses on the same cell.

It’s official; the amygdala is not the hippocampus. It is tempting to focus on the similarities between the two structures. They are both found in the medial temporal lobes, they are heavily interconnected and share connections with many other structures, and perhaps most importantly, they have both been strongly implicated in learning and memory. Although neuroscientists focusing on the biological substrates of learning and memory have recognized the anatomical differences between the amygdala and hippocampus, they have also demonstrated a tendency to assume that the synaptic physiology of these two limbic system structures is very similar, if not identical. A growing body of evidence, exemplified by the new report by Rogawski and colleagues, suggests that this is far from the case. Rogawski and colleagues find that low-frequency stimulation of synaptic inputs to the basolateral amygdala produces a long-lasting enhancement of synaptic transmission. In contrast to typical hippocampus-based synaptic plasticity, this enhancement is dependent upon kainate-type glutamate receptors, and spreads to adjacent, inactivated synapses. These unique physiological characteristics of amygdala-based plasticity may have important functional implications in behavior.

The canonical synapse for studying use-dependent changes in synaptic function is between the CA3 and CA1 pyramidal neurons of the hippocampus. At this synapse and in the perforant pathway synapses onto granule neurons of the dentate gyrus, long-term potentiation (LTP) can be induced by high-frequency stimulation through the activation of the N-methyl-D-aspartate (NMDA)-type glutamate receptor. This has become the de facto model for learning and memory in the mammalian brain; brief bursts of activity at much higher than normal frequency lead to NMDA receptor-dependent increases in synaptic efficacy, which alters network function to code for new memories. Pharmacological or genetic manipulations that block NMDA receptor function should (and generally do) prevent learning of behavioral tasks that depend on the hippocampus.

But the story cannot be that simple. High-frequency stimulation of mossy fiber inputs to CA3 pyramidal neurons produces LTP that does not depend on NMDA receptors, and some patterns of stimulation can produce NMDA receptor-independent LTP even at CA3-CA1 synapses. Moreover, prolonged stimulation at a relatively low frequency (1–5 Hz, compared to the 100–400 Hz typically used for LTP induction) causes long-lasting decreases in synaptic efficacy in neurons of the hippocampus and neocortex, and this long-term depression (LTD) also depends on NMDA receptor activation. So, blocking NMDA receptors in vivo could alter behavior by preventing either LTP or LTD; conversely, NMDA receptor-independent plasticity may or may not contribute to behavioral changes under these conditions.

Into this mix, we now must add the amygdala. Most LTP experiments in the amygdala have focused on the lateral and/or basolateral nuclei (LA/BLA), which receive inputs from adjacent polymodal neocortical regions, the thalamus, and other subnuclei within the amygdala. Although the stimulus parameters used to induce LTP have varied from study to study, the amygdala exhibits a similar degree of plasticity as the hippocampus. The new study by Rogawski and colleagues highlights the importance of understanding the unique physiological characteristics of the amygdala, and suggests that future research should take into account the specific role of kainate receptors in synaptic plasticity in this brain region.