LTP induction in the vertical lobe (VL) of octopus and cuttlefish is mediated by nitric oxide (NO)

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The VL system, involved in learning and memory in modern cephalopods, comprises three neuron types. Superior frontal lobe neurons (SFLn) send their axons to small amacrine interneurons (AM) *en passant*, that then converge onto large efferent neurons (LN). The synaptic inputs to the AM are glutamatergic, while the synaptic connections from AM to the LN are cholinergic. While the neuronal connectivity is similar in both *Octopus vulgaris* and *Sepia officinalis*, the short- and long-term synaptic plasticities are different. The activity-dependent LTP in octopus is in the glutamatergic SFLn-AM synapses, while in cuttlefish is in the cholinergic AM-LN synapses.

In the current study we tested the involvement of NO in VL plasticity because NO may mediate LTP irrespectively of the nature of the neurotransmitter and because NO synthase inhibitors impair memory formation in cephalopods and other molluscs. To test this we examined the effect of NO synthase inhibitors on LTP induction in VL slices of octopus and cuttlefish. We observed an inhibitory effect on neural activity and specific blockage of LTP induction. Short-term facilitation was unaffected. These results suggest that NO may mediate LTP in cephalopods, suggesting that NO plays a general role in complex forms of learning and memory in molluscs.

Support: Smith Lab HUJI, Israel Science Foundation, Binational Israel-US Science Foundation, EP7 projects STIFF-FLOP and OCTOPUS, Golda Meir and Lady Davis Postdoctoral Fellowships for T-MAL.