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Project Title: The Effect of Secretin on Extinction of Eyeblink Conditioning

Abstract

Eyeblink classical conditioning (EBC) is a well studied model of cerebellum-dependent learning. In EBC, each trial consists of a tone that precedes a mild eyelid stimulation by about half a second. After many trials of the tone followed by the eyelid stimulation, an eyeblink is made to the tone in anticipation of the eyelid stimulation. The eyeblink to the tone is the learned, or conditioned, response (CR) and is dependent upon the cerebellum. If, after conditioning, the tone is no longer followed by the eyelid stimulation, eyeblink CRs to the tone gradually diminish; this is termed extinction. While there is support for plasticity in the cerebellar cortex and in the deep cerebellar nuclei (interpositus nucleus) that supports conditioning, brain mechanisms of extinction in EBC remain elusive and disputed. Secretin is an endogenous neuropeptide found in cerebellar cortex. . Our laboratory has shown that infusions of secretin into cerebellar cortex during conditioning increase the number of trials with an eyeblink CR to the tone. We therefore hypothesized that infusions of secretin prior to extinction would disrupt extinction, by also increasing the number of eyeblink CRs to the tone. Preliminary evidence suggests that rats that receive secretin infusions prior to the first (but not second) session of extinction show a higher percentage of trials with an eyeblink CR to the tone, compared to rats that receive saline infusions on days 1 and 2 of extinction. Our working model of the cellular mechanism underlying this effect involves secretin-produced suppression of a voltage-gated potassium channel, Kv1.2, in cerebellar cortex. This represents a very novel mechanism underlying mammalian learning.