

THE ROLE OF GUSTATORY NEOCORTEX (GNC) IN THE EXTINCTION AND SPONTANEOUS RECOVERY OF A CONDITIONED TASTE AVERSION (CTA).
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While substantial advances have been made in discovering how the brain learns and remembers, less is known about how the brain mediates extinction of conditioned behaviors or changes during spontaneous recovery (SR). These topics are not only relevant to normal brain functioning but also speak to pathologies in which painful memories do not wane but are evoked time and again (e.g., Post-traumatic stress disorder; PTSD). CTAs may be acquired when an animal consumes a novel taste (conditioned stimulus; CS) and then experiences the symptoms of poisoning (unconditioned stimulus; US). When later given a choice between the CS and some more-familiar gustatory stimulus (typically water), the animal will avoid the taste that it previously associated with malaise. Extinction of a CTA is observed following repeated, nonreinforced exposures to the CS and represents itself as a resumption of eating/drinking the once-avoided tastant. SR of a CTA (a revival of the taste avoidance) occurs after a latency period in which the CS is not presented. The neuronal pathway that subserves CTA acquisition is well known and the GNC plays a primary role in the mediation of both the acquisition and extinction of this memory. This study investigated changes in GNC functioning during acquisition, extinction and spontaneous recovery of a CTA. Brain *c-Fos* protein expression was analyzed in fluid-deprived rats that had acquired a CTA [3 pairings of 0.3% oral saccharin (SAC) and 81mg/kg i.p. Lithium Chloride (LiCl)] followed by extinction training (i.e., subsequent non-reinforced SAC exposures) resulting in 90% reacceptance of SAC. Other animals were extinguished but spontaneously recovered the CTA upon exposure to SAC following a 15-, 30-, 45- or 60-day recovery period of water drinking. Rats were sacrificed on the final day of SAC exposure and GNC *c-Fos* protein expression was evaluated. SR of the CTA depended on both the time to meet the extinction criterion and the length of the recovery period. Animals allowed 30- or 60-day recovery periods exhibited a significant SR. The numbers of *c-Fos*-labeled neurons in GNC was low following CTA acquisition but increased dramatically as rats fully extinguished the aversion. However, a significant decline in *c-Fos* expression accompanied SR of the CTA. These data elucidate the behavioral parameters required to observe the SR of a CTA. Further, the immunohistochemical measurements suggest the dynamic nature of GNC activity during acquisition, extinction and SR of a CTA and further reinforce an important role for these cortical neurons in the reorganization of learned information. *Supported by NIMH.*