**Account Hacker V3.9.9 Activation Code 1109 ((FREE))**

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Methods: We recorded neurons in awake, head-restrained mice while they performed a rat-in-a-maze task. We trained the mice to associate a visual cue with reward delivery in a hole-board. The task was subsequently modified to test the animals' ability to associate cues with effort. After training, we removed cues and introduced false-cues in an effort task. We found that NAcc neurons in the mice accurately predicted the nature of the learned cue–reward association, and expressed this information both in the time-course of their activation and in the absence of cues. NAcc neurons showed less directional selectivity in the effort task but did show evidence of a ‘skill’ to learn two opposing signals, representing effort and the cue for reward. This skill-like behavior was not seen in the dmPFC and amygdala. Neurons recorded in the perirhinal cortex showed no evidence of encoding for effort in either the naïve or the effort tasks. Additionally, a linear SVM was able to decode cue identity from population activity in both the NAcc and dmPFC, but only in the effort task. Furthermore, overall population activity in these areas decoded from trial number, but only in the effort task. Age-related vascular dementia (VaD) is a common form of dementia in the elderly population. Patients with this disease has slow progression of cognitive and functional decline associated with recurrent V-type intracerebral hemorrhage (VHT-ICH). Clinical evidence suggests that activation of glial cells and microglia play important roles in pathogenesis of cognitive deficits after VHT-ICH. However, how the cognitive deficits are associated with the activation of glial cells and microglia remains elusive. Here, we investigated the role of microglial activation in cognitive deficits of aging mice after VHT-ICH using FKN, a functional activation marker of microglia. To this end, we assessed the cognitive impairments of a novel vascular dementia model induced by intravenous injection of anti-VEGF antibody (NAB) into the jugular vein to the animals. FKN+ microglia significantly increased in the ipsilateral cortex at 2 weeks after NAB injection, and the FKN+ microglia also increased in the ipsilateral hippocampus at the onset phase of cognitive deficits. Furthermore, the FKN+ microglia was observed in cholinergic neurons in the hippocampus.

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