Activation of phenotypically distinct neurons in the amygdala of rats following exposure to ferret odor

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Exposure of rats to an odor of a predator can elicit an innate fear response. In addition, such exposure has been shown to activate limbic brain regions such as the amygdala. However, there is a paucity of data on the phenotypic characteristics of the activated amygdalar neurons following predator odor exposure. In the current experiments, rats were exposed to cloth which contained either ferret odor, low or high amounts of butyric acid (a noxious odor), or no odor for a duration of 30 minutes. Two hours following exposure, rats were sacrificed, perfused with PBS and paraformaldehyde (4%), and the brains were isolated. Sections of the brains were prepared for single- (cFOS) and dual-labeled immunohistochemistry and the number of single and dual-labeled neurons of the left and right basolateral (BLA), central (CeA), and medial amygdala were counted. To analyze some of the major cell populations of the amygdala, co-localization of cFOS and Ca2+/calmodulin-dependent protein kinase (CAMK) II, parvalbumin, and calbindin was assessed. Data were analyzed with 1- or 2-way ANOVA followed by Student-Newmann-Keul’s post-hoc test. Behavior analysis showed an increase in defensive burying in ferret odor-exposed versus control rats during the behavior trial. Dual-labeled immunohistochemistry showed a significant increase in the percentage of CAMKII neurons co-localized with cFOS in both the left and right BLA and CeA of ferret odor-exposed compared to control and/or butyric acid-exposed rats. Further results suggested a decrease in the percentage of calbindin neurons that were also labeled with cFOS in the MeA of ferret-exposed versus low butyric acid-exposed rats. The results suggest that exposure to predator odor activates the glutamatergic projection neurons of the BLA, while potentially decreasing the activation of some inhibitory neuronal populations. These results enhance our understanding of the functioning of the amygdala following exposure to predator threats by showing phenotypic characteristics of activated amygdalar neurons. With this knowledge, specific neuronal populations could be targeted to further elucidate the fundamental underpinnings of anxiety or possibly for developing novel therapeutics.

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