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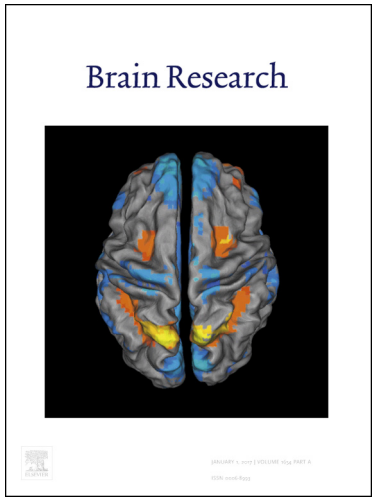

Research report

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ACCEPTED MANUSCRIPT

Odor preference and olfactory memory are impaired in Olfaxin-deficient mice

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List of abbreviations: PRUNE2, prune homolog 2; BNIP, Bcl-2/adenovirus E1B 19 kDa-interacting protein; BNIPXL, BNIP2 extra long; BMCC1, BCH-motif-containing molecule at the C-terminal region 1; BCH, BNIP2, and Cdc42GAP homology

Abstract

Olfaxin, which is a BNIP2 and Cdc42GAP homology (BCH) domain-containing protein, is predominantly expressed in mitral and tufted (M/T) cells in the olfactory bulb (OB). Olfaxin and Caytaxin, which share 56.3% amino acid identity, are similar in their glutamatergic terminal localization, kidney-type glutaminase (KGA) interaction, and caspase-3 substrate. Although the deletion of Caytaxin protein causes human Cayman ataxia and ataxia in the mutant mouse, the function of Olfaxin is largely unknown. In this study, we generated *Prune2* gene mutant mice (*Prune2*$^{Ex16-/-}$; knock out [KO] mice) using the CRISPR/Cas9 system, during which the exon 16 containing start codon of *Olfaxin* mRNA was deleted. Exon 16 has 80 nucleotides and is contained in four of five *Prune2* isoforms, including PRUNE2, BMCC1, BNIPXL, and Olfaxin/BMCC1s. The levels of *Olfaxin* mRNA and Olfaxin protein in the OB and piriform cortex of *KO* mice significantly decreased. Although *Prune2* mRNA also significantly decreased in the spinal cord, the

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