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Behavioral differences in fractalkine receptor (CX3CR1) - deficient mice

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Fractalkine is a signal molecule that plays a role in the immune system and in the central nervous system (CNS) in cell migration and cell-cell communication. CX3CR1 (fractalkine receptor) is important for sustaining normal activity of microglia in the brain, and of the monocytes, dendritic and NK cells, in the periphery. In our experiments transgenic mice (C57Bl6 background), in which a certain part of the CX3CR1 gene was replaced by a green fluorescent protein (GFP) were used. Heterozygote Cx3cr1 +/gfp animals express normal fractalkine receptor, whereas homozygote Cx3cr1 gfp/gfp mice do not have functional receptor, while cells expressing the fractalkine receptor appear in green in both genotypes. The aim of our present experiments was to characterize behavior phenotype of fractalkine deficient mice. We compared heterozygote Cx3cr1 +/gfp and homozygote Cx3cr1 gfp/gfp males with C57Bl6 males in four standard behavior tests: open field (OF), elevated plus maze (EPM), tail suspension test (TST) and forced swimming test (FST). In all tests, the heterozygotes and wild-types were not different (p-value < 0.05). However, Cx3cr1 gfp/gfp mice spent more time on the open arm of the elevated plus maze, spent more time swimming in the FST, and were immobile for less time in TST compared to heterozygotes or to wild type animals. Our results show that mice without fractalkine signaling display active coping behavior and develop resilient phenotype. We suggest that the function of neuron-microglial communication goes far beyond to neuro-immune functions.