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Investigating targets for intellectual disability in autism

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Fragile X syndrome (FSX) is the leading single gene cause of autism and intellectual disability (ID). Neurons express a high density of underdeveloped dendritic spines in FXS humans and animal models. Synaptic plasticity deficits are prevalent throughout the brains of FXS mouse models including the cortex and hippocampus; areas critical for various forms of learning and memory. Moderate to severe learning deficiencies are also characteristic in FXS patients and is paralleled in mouse models. Therefore, FXS is an ideal model in the clinical and laboratory setting to investigate therapies aimed at autism and ID. In FXS mouse models, hyperactive Rac1 has been demonstrated in hippocampus and cortex where dendritic spine abnormalities are a common feature. Herein, we study whether pharmacological regulation of Rac1 might represent a promising treatment for cognitive impairment in autism, using Fragile X syndrome (FXS) as a model. Our results show that in the Fmr1 KO mice (an animal model of FXS) deficits in memory and synaptic plasticity are associated with the presence and mislocalization of Rac1. Interestingly, treatment of Fmr1 KO mice with a specific Rac1 inhibitor improves memory and increases hippocampal LTP. Taken together these observations suggest that Rac1 might contribute to FXS related learning and memory impairments in humans. Importantly, this study proposes that targeting Rac1 in FXS may rescue cognitive impairments. Such a therapy may be translated into broader applications in autism and ID.

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