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Autism Spectrum Disorder (ASD) is a common neurodevelopmental disorder, which despite years of research is still diagnosed based only on behavioural criteria. This delays the onset of any aimed therapies. Only about 10% of ASD cases have known monogenic causes. The remainder of patients are described as idiopathic cases. Many of these share common clinical features, including learning disabilities and stereotypies, which share common synaptic plasticity impairment background. In order to study the role of synaptic plasticity in the development of ASD-like symptoms, the use of animal models of the disorder is necessary. Here we would like to employ two distinct models of ASD: 1. Fmr1KO mice (a mouse model for Fragile X syndrome, the most common genetic cause of autism) and 2. BTBR T+ Itpr3tf/J mice, the best studied model of idiopathic autism. To address the question of the mechanisms underlying their autism-like behaviors we will compare their dendritic spine morphology and functionality to animals with locally altered synaptic plasticity within the amygdalar complex. The amygdala was chosen, as our preliminary data show that both of the autistic mouse strains display aberrant functional synaptic plasticity in this region. The manipulation in control mice will be obtained by infusing lentiviral vectors carrying additional copies of matrix metalloproteinase-9 gene (MMP-9), an enzyme responsible for extracellular matrix remodelling necessary for changes in dendritic spine morphology in response to external stimulation. If the results prove similar, we will proceed with an attempt to reverse the observed phenotype by infusing tissue inhibitor of metalloproteinases (TIMP-1) into the same region. If morphological and functional synaptopathies are reversed by this treatment we will assess the effect of TIMP-1 supplementation on autism-like behaviors in all of our experimental mouse strains. This will be done using between-subject transfer of emotional information test, designed to study emotional contagion (simple form of empathy) in mice. Mice with autism-like phenotype do not display that type of behavior. If TIMP-1 treatment rescues this behavior it will open a new venue for possible early diagnosis and treatment of ASD.