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A behavioral exploration of intergenerational transmission of folate receptor antibody-induced neurodevelopmental disorder

It has been reported that the majority of children with autism spectrum disorder (ASD) produce autoantibodies against folate receptor alpha (FRaAb), the main transporter of folate into the central nervous system. Low levels of folate within the central nervous system at critical stages of development have severe consequences such as neural tube defects and cerebral folate deficiency syndrome. It has also been shown that family members of ASD children are more likely to produce FRaAb than families where ASD is not present. The main question that arises from these findings is does FRaAb play a role in hereditary transmission of neurodevelopmental disorders like ASD? Our lab has produced a rat model of FRaAb-induced behavioral phenotype that is analogous to the core deficits observed in ASD. By testing rats on a battery of behavioral tests and mating those with the severe phenotypes across one generation, we have seen transmission of behavioral features that arise in the first generation as well as a heterogeneous severity of behavioral deficits. Our findings suggest that our rat model can be a useful tool in investigating the other mechanisms of hereditary transmission of behavioral deficits that are associated with neurodevelopmental disorders like ASD.