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Intergenerational transmission of behavioral deficits in rats exposed to folate receptor alpha antibodies in utero.

Autism Spectrum Disorder (ASD) is a neurodevelopmental disorder characterized by the core symptoms of social deficits, communication impairments, and repetitive behaviors. Folate deficiency during the neural development has been widely known as a link to many neurological disorders, including ASD. Folate as an essential nutrient is necessary for many cellular mechanisms which include DNA synthesis and methylation. One mechanism that can lead to folate deficiency is autoimmunity against folate receptor alpha (FR□), the main transporter of folate. Recent evidence suggests that there is a 70% prevalence of autoantibody production against (FR□) in children with ASD, their parents, and normal siblings. Preliminary treatment studies found significant improvement in language and communication when ASD children who are positive for FR□ autoantibodies are given folinic acid, a reduced form of folate. Moreover, our laboratory has developed a rat model of FR□ antibody exposure during gestation day 8 that produces progeny with behavioral deficits like the human condition of ASD. Therefore, this presentation will investigate whether the impact FR□IgG-Ab on social communication in this model can transfer across a generation of offspring. We will be measuring ultrasonic vocalizations (USVs) at pre-weaning and adult stages of life as a measure of social communication. We expect that animals exposed directly and indirectly to FR□IgG-Ab will have altered social communication at both stages of life. By establishing this effect, we can further investigate how ASD is inherited and how it may be potentially prevented with folinic acid treatment.