Behavioral and morphological changes in a rodent developmental disruption model of schizophrenia

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Epidemiological and clinical studies suggest that a neurodevelopmental dysfunction could be one of the main exploratory hypotheses of schizophrenia (SCZ), which symptoms lead to severe personal and social dysfunctions. Even though the symptoms resulting from this detrimental neuronal development remain relatively dormant until the psychosis in adulthood is manifested, a possible identification of certain premorbid neurodevelopmental signs has been suggested. In the present study, we aimed to investigate the potential effects of prenatal administration of the mitotoxin methylazoxymethanol acetate (MAM) on early neurophenotypic presentations using a set of behavioral test battery. At birth, neonatal reflexes (righting, cliff aversion, forelimb placing, bar holding, forelimb grasping, negative geotaxis) had a delayed onset (i.e. percent of appearance) in prenatally MAM-exposed rats, as compared to the control group. During adolescence, they engaged in less social interaction as well as they shown cognitive impairment, which were correlates with morphological changes (i.e. enlargement of ventricles), as revealed by magnetic resonance imaging (MRI). These results suggest that behavioral abnormalities resulting from a MAM environmental challenge, which resemble to a schizophrenia-like phenotype, could be due to structural abnormalities.

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