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Sustained increase of neuronal polysialic acid level does not affect the nervous system development and maintenance but induces a mild behavioral deficit that may be attributed to synaptic dysfunction

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The polysialic acid (PSA) modification of the neural cell adhesion molecule (NCAM) is an abundant posttranslational modification during development. During postnatal development, PSA is rapidly down-regulated but remains expressed in certain brain regions that are involved in neurogenesis and display structural plasticity. This significant down regulation suggests that removal of PSA after modeling of the nervous system is an important task during postnatal development of the brain. In contrast, increase of PSA level is observed in some neurological disorder including multiple sclerosis and Alzheimer's diseases. We therefore hypothesized that prevention of postnatal down regulation of PSA will affects the structural and functional properties of the nervous system. To test our hypothesis, we generated transgenic mice overexpressing the polysialyl transferase (ST8SiaIV) in neurons. The transgene expression prevented the postnatal down regulation of PSA and most NCAM-140 and NCAM-180 in the forebrain was polysialylated. Morphological examinations did not reveal structural abnormalities in transgenic mice as compared to wild-type controls. Behavioral studies revealed a reduced rearing activity and exploratory behavior while parameters of motor activity like distance traveled and mean velocity in an open field or rotarod performance were not affected in transgenic mice. These results demonstrate that preventing the postnatal down regulation of PSA has a significant effect on exploratory behavior which may be attributed to some synaptic dysfunction in the brain. The absence of a neurodevelopmental phenotype makes the transgenic mouse a useful model system suited for further exploring the effects of sustained increased of PSA level in the brain homeostasis.

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