

The Nogo-A protein belongs among the most important regulatory molecules in the brain, regulating development of neuronal and glial cells, axon guidance and adult synaptic plasticity. Although it has been studied mainly as an obstacle to axon regeneration after CNS injury, it plays a role in many pathological conditions, including neurodegenerative and neuropsychiatric diseases. This work offers a literature review of the current knowledge about functions of Nogo-A and related proteins, and then recapitulates the results of experiments focused on the impact on decreased expression of Nogo-A on behavior in a transgenic rat model. The most important finding is that the Carousel Maze performance, tapping higher cognitive functions such as cognitive coordination and cognitive flexibility, is remarkably impaired in this model, while other cognitive functions, such as spatial navigation and both spatial and non-spatial memory are spared in the Nogo-A deficient rats. The results are discussed in the context of a hypothesis linking Nogo-A mutations or abnormal expression to human schizophrenia. We conclude that the Nogo-A deficient rats constitute a very promising animal model of schizophrenia and deserve further attention.