

Animal Model

MAG $-/-$ mice: a model for studying pathological mechanisms of acquired demyelinating diseases

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Abstract

Challenge

Genetically modified animals are essential research tools in modern neuroscience, since they allow researchers to study the role of specific genes in pathological processes leading to neurodegenerative diseases. The myelin-associated glycoprotein (MAG) is a transmembrane glycoprotein localized in the myelin sheath membranes of periaxonal Schwann cells and oligodendroglial cells. MAG functions both as a ligand for an axonal receptor needed for the maintenance of myelinated axons and as a receptor for an axonal signal promoting differentiation, maintenance and survival of oligodendrocytes. MAG has also been implicated to play a major role in acquired neurological disorders caused by autoimmune mechanisms (e.g. IgM paraproteinaemic neuropathies).

In situ-hybridization studies show that Mag mRNA is expressed by oligo-dendrocytes in the cerebellum.

Technology

MAG $-/-$ mice were generated by inactivating the Mag gene via homologous recombination in embryonic stem cells and creating transgenic mice. MAG $-/-$ display mild cognitive and locomotor impairment and subtle structural abnormalities in the periaxonal region of myelin sheaths. Furthermore, degeneration of periaxonal oligodendroglial processes has been observed in aging MAG $-/-$ mice, suggesting a "dying-back" oligodendroglipathy which is typically seen as a reaction to certain toxins (e.g. Cuprizone toxicity), but also in early-stage multiple sclerosis. MAG $-/-$ mice are therefore a valuable tool for studying the pathological mechanisms of acquired demyelinating diseases.

Commercial Opportunity

Breeding pairs are available under a Tangible Property License Agreement.

Further Reading

- Montag et al.: Mice deficient for the myelin-associated glycoprotein show subtle abnormalities in myelin. Neuron, Vol. 13, 229-246, July, 1994.
- Quarles RH: Myelin-associated glycoprotein (MAG): past, present and beyond. J of Neurochemistry, 2007, 100, 1431 - 1448.