

3050 Spruce Street
Saint Louis, Missouri 63103 USA
Telephone 800-325-5832 • (314) 771-5765
Fax (314) 286-7828
email: techserv@sial.com
sigma-aldrich.com

ProductInformation

Anti-MOG (C-terminal)

produced in rabbit, affinity isolated antibody

Catalog Number M0820

Product Description

Anti-MOG (C-terminal) is produced in rabbit using a synthetic peptide corresponding to amino acids 231-247 located at the C-terminus of human MOG α 1 isoform, conjugated to KLH, as immunogen. This sequence is identical in human MOG α 4 and MOG β 1 isoforms, partially found (six amino acids at the C-terminus) in human MOG α 2 and MOG α 3 isoforms, and is identical in rat, mouse and bovine MOG. The antibody is affinity-purified using the immunizing peptide immobilized on agarose.

Anti-MOG (C-terminal) recognizes MOG, 26 kDa, by immunoblotting. Staining of the MOG band is specifically inhibited with the MOG immunizing peptide.

Myelin-oligodendrocyte glycoprotein (MOG) is a member of the immunoglobulin (Ig) superfamily, exclusively expressed in the central nervous system (CNS). 1,2 MOG is an intrinsic membrane protein characterized by a N-terminal extracellular immunoglobulin-like variable (Ig-like V-type) domain, two hydrophobic transmembrane domains and a cytoplasmic C-terminal region. 1,3 The MOG gene is localized to chromosome 6p22-p21.3 at the distal end of the MHC class lb region. ^{2,4,5} It contains nine exons and eight separating introns, giving rise to at least eight alternatively spliced variants encoding for the $MOG\alpha1-4$ and $MOG\beta1-4$ isoforms (16-26 kDa). The different MOG isoforms may interact to form homo- and heterodimers and trimers (55 and 78 kDa). During the last step of myelinogenesis, MOG is expressed in the CNS on the outermost surface (external lamella) of mature myelin sheaths and on the cell surface of myelinating oligodendrocytes. 1-3,6 MOG has a variety of functions including a role as a cellular adhesion molecule. It may be involved also in the completion and/or maintenance of the myelin sheath and in cell-cell communication. MOG is also thought to function as a regulator of oligodendrocyte microtubule stability and as a mediator of interactions between myelin and the

immune system in the complement cascade. Although MOG is a relatively minor component of the myelin membrane (0.05-0.1% of total myelin protein), it is considered a primary auto-antigen target involved in the pathogenesis of immune-mediated demyelinating diseases including experimental autoimmune encephalomyelitis (EAE) and multiple sclerosis. 4,8,9

Reagent

Supplied as a solution in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide as a preservative.

Antibody concentration: ~2 mg/ml

Precautions and Disclaimer

Due to the sodium azide content a material safety data sheet (MSDS) for this product has been sent to the attention of the safety officer of your institution. Consult the MSDS for information regarding hazardous and safe handling practices.

Storage/Stability

For continuous use, store at $-20\,^{\circ}\text{C}$. For extended storage freeze in working aliquots. Repeated freezing and thawing, or storage in "frost-free" freezers, is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilution samples should be discarded if not used within 12 hours.

Product Profile

Immunoblotting: a working concentration of 2.5-5 μ g/ml is determined using a rat brain homogenate (S1 fraction) and rat spinal cord homogenate (P2 fraction).

Note: In order to obtain best results and assay sensitivity in different techniques and preparations we recommend determining optimal working concentrations by titration test.

References

- Pham-Dinh, D., et al., *Proc. Natl. Acad. Sci. USA*, 90, 7990-7994 (1993).
- Pham-Dinh, D., et al., Genomics, 29, 345-352 (1995).
- 3. Della-Gaspera, B., et al., *Eur. J. Biochem.*, **258**, 478-484 (1998)
- Pham-Dinh, D., et al., *Immunogenetics*, **42**, 386-391 (1995).
- 5. Roth, M.P., et al., Genomics, 28, 241-250 (1995).

- 6. Li, G., et al., Brain Pathol., 12, 463-471 (2002).
- 7. Johns, T.G., and Bernard, C.C., *J. Neurochem.*, **72**, 1-9 (1999).
- 8. Stefferl, A, et al., *J. Neural Transm.*, suppl., **58**, 123-133 (2000).
- 9. Reindl, M., et al., Brain, 122, 2047-2056 (1999).

ER,AH,PHC 06/06-1