



Catalog Number:	MO22102	Host:	Mouse
Product Type:	Mouse Monoclonal IgG	Species Reactivity:	Human, Rat, Mouse, and Cow
Immunogen Sequence:	Purified recombinant rat α-internexin expressed in and purified from <i>E. coli</i> .	Format:	Purified liquid antibody in PBS, 50% glycerol plus 5mM of Sodium Azide. Concentration: 1mg/ml.
Applications:	Immunofluorescent: 1:5,000 Immunocytochemistry: 1:5,000 Immunohistochemistry: 1:5,000 Western Blot: 1:10,000		

Dilutions listed as a recommendation. Optimal dilution should be determined by investigator.

Storage: Antibody can also be aliquotted and stored frozen at -20° C in a manual defrost freezer for six months without detectable loss of activity. The antibody can be stored at 2° - 8° C for 1 month without detectable loss of activity. Avoid repeated freeze-thaw cycles.

Application Notes

Description/Data:

α-internexin is a Class IV intermediate filament protein originally discovered by two different groups of researchers as it copurifies with NF-L, NF-M and NF-H, the then better known major neurofilament "triplet" subunits. It is expressed only in neurons and in large amounts early in neuronal development, but is down-regulated in many neurons as development proceeds. Some neurons express α-internexin in the absence of NF-L, NF-M and NF-H, though most mature neurons express all four proteins. This α-internexin antibody has been shown, in peer reviewed publications, to reveal the upregulation of α-internexin in facial neurons following experimental axotomy followed by down regulation on axonal regeneration. It is also the standard reagent used to identify and classify patients with neurofilament inclusion body disease, a specific form of frontotemporal lobar dementia. Finally, it has been used to confirm the presence of circulating antibodies to α-internexin in the blood of certain patients with endocrine autoimmunity.

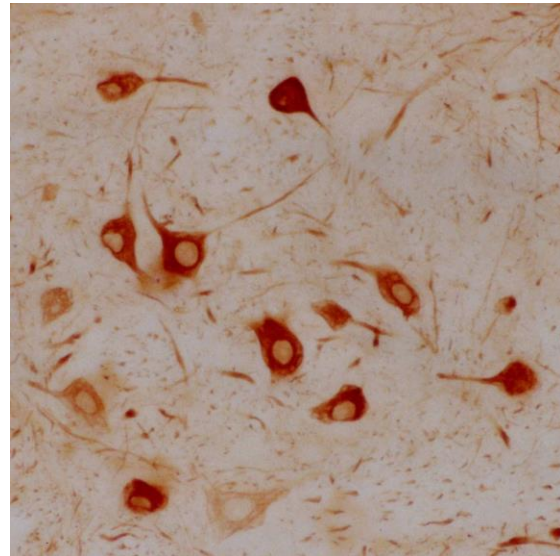


Image: Immunohistochemistry of a section of rat facial nucleus 7 days following axotomy. These neurons are capable of regenerating their axons and also, concomitant with regeneration, strongly upregulate α-internexin in their perikarya. Other central neurons which are not able to regenerate their axons do not upregulate this protein after axotomy and untreated facial neurons normally show only very low levels of α-internexin. Both findings suggest that α-internexin has a role in axonal regeneration.

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