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ProductInformation

Anti-Myopodin
Developed in Rabbit
Affinity Isolated Antibody

Product Number **M 9818**

Product Description

Anti-Myopodin is developed in rabbit using as immunogen a synthetic peptide encoding amino acids 566-585 located at the mid-region of human myopodin, conjugated to KLH. This sequence is highly conserved (77% sequence identity) in mouse myopodin and is not found in human or rat synaptopodin. The antibody is affinity-purified using the immunizing peptide immobilized on agarose.

Anti-Myopodin recognizes myopodin by immunoblotting (80 kDa) and immunohistochemistry. By immunoblotting, additional lower bands may be observed in some cell extracts, which may represent myopodin degradation products. Staining of myopodin in immunoblotting is specifically inhibited with the myopodin immunizing peptide (human, amino acids 566-585).

Myopodin (80-95 kDa), a novel actin bundling protein, is an additional member of the synaptopodin gene family.[1] Myopodin shows no significant homology to any known protein except synaptopodin. The overall homology between synaptopodin and myopodin is approx. 48%. Myopodin is expressed in skeletal and cardiac muscle. Myopodin contains one PPXY motif, mutiple PXXP motifs, and a nuclear export sequence (NES). Myopodin colocalizes with α-actinin and is found at the Z-disc. It can directly bind to actin and contains an actin-binding site in the center of the protein. Myopodin has actin bundling activity as shown by lantraculin-A-sensitive cytosolic actin bundles and nuclear actin loops in transfected cells expressing GFP-myopodin. A dual role for myopodin has been suggested as a structural protein also participating in signaling pathways between the Z-disc and the nucleus.

Myopodin, like several actin-bundling proteins, has been shown to shuttle between the nucleus and cytoplasm.[1-3] Myopodin redistributes between the nucleus and cytoplasm in a differentiation and stress-induced fashion. In myoblasts, myopodin shows preferential nuclear localization. During myotube differentiation, myopodin binds to stress fibers in a punctuated pattern before incorporating into the Z-disc. Under stress conditions, myopodin accumulates in the nucleus and is depleted from the cytoplasm. Myopodin is frequently down-regulated in invasive stages of some types of cancers.[4-6] Invasive bladder tumors decreased nuclear expression as compared to superficial lesions. Frequent complete or partial deletions of the myopodin gene have been shown to occur in 80% of invasive prostate cancer cases. Expression of myopodin induces suppression of tumor growth both *in vivo* and *in vitro*. It has been suggested that myopodin functions as a tumor suppressor gene to limit the growth and inhibit the metastasis of cancer cells.

Reagent

Anti-Myopodin is supplied as a solution in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide.

Antibody Concentration: approx. 1.0 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 2-8 °C for up to one month. For extended storage, freeze in working aliquots. Repeated freezing and thawing is not recommended. Storage in frost-free freezers is also not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilutions should be discarded if not used within 12 hours.

Product Profile
By immunoblotting, a working antibody concentration of 1-2 µg/ml is recommended using a cytosolic fraction of rat skeletal muscle.

By immunohistochemistry, a working antibody concentration of 1-2 µg/ml is recommended using frozen sections of rat skeletal muscle and rat kidney.

Note: In order to obtain the best results using various techniques and preparations, we recommend determining the optimal working dilutions by titration.

References

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6. Jing, L., et al., Am. J. Pathol., **164**, 1799-1806 (2004).

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