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ProductInformation

Anti-ADAM-12, N-Terminal
Developed in Rabbit
Affinity Isolated Antibody

Product Number **A 2976**

Product Description

Anti-ADAM-12, N-Terminal is developed in rabbit using a synthetic peptide corresponding to the N-terminal of human ADAM12 (A Disintegrin And Metalloproteinase-12) as immunogen. Affinity isolated antigen specific antibody is obtained from rabbit anti-ADAM-12 antiserum by immuno-specific purification which removes essentially all rabbit serum proteins, including immunoglobulins, which do not specifically bind to the peptide.

Anti-ADAM-12, N-Terminal may be used for the detection and localization of human ADAM12 and does not react with other ADAMs. By immunoblotting against the reduced protein, the antibody recognizes bands of 116 kDa (minor band), 92 kDa (major band), and cleaved products at 68 kDa and 40 kDa in cell lysates.

ADAM12, also known as Meltrin α, is a member of the ADAM (a disintegrin and metalloprotease-like domain) family. ADAM12 was first described as a protein participating in myoblast fusion, similar to sperm-egg fusion aided by ADAMs 1 and 2. Later, two forms of ADAM12 were described: ADAM12-S and ADAM12-L (short and long forms). The short form is a soluble, alternatively spliced form lacking the transmembrane and cytoplasmic domains. The short form of ADAM12 was reported to provoke myogenesis, and the lack of cytoplasmic domain suggests different regulation pathways (although both forms can be expressed in the same tissue). Other papers investigated the SH3 ligand domains in the cytoplasmic portion of ADAM12, demonstrating regulation routes for ADAM12 via Src and Src tyrosine kinase.[1]

ADAM12 is thought to have more limited expression than the other ADAMs (ADAM9 and ADAM19) originally described in myoblasts. It is primarily expressed in muscle and bone (although placenta, osteoblasts, and many tumor cell lines also express ADAM12).[2] ADAM12 supports cell adhesion by acting as a cell-attachment molecule and binding integrins through the cysteine-rich domain.[3] ADAM12, a marker of skeletal muscle regeneration, has been implicated in myoblast differentiation and fusion.[4]

ADAM-12 contains the canonical HExxHxxxxxH zinc metalloproteinase motif, and has been shown to be proteolytically active. Full length ADAM12 (909 amino acids) has a predicted mass of 99.5 kDa, but due to glycosylation and cysteine-rich regions, the reduced protein migrates to 114 kDa (unprocessed) and 84 kDa (processed). The soluble form of ADAM12 (738 amino acids, predicted mass of 80.5 kDa) is seen as a 92 kDa zymogen and 68 kDa furin-processed form.

Reagent

Anti-ADAM-12, N-Terminal is supplied in phosphate buffered saline containing 50% glycerol and 0.05% sodium azide. The protein concentration is approximately 1 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 hazards and safe handling practices.

Storage/Stability

For continuous use, store at 2-8 °C for up to six months. For extended storage, the solution may be stored −20 °C. Do not store below −22 °C. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use.

Product Profile

A minimum working antibody dilution of 1:1,000 is determined by immunoblotting tissue or cell lysates with an alkaline phosphatase conjugated secondary antibody and BCIP/NBT as the substrate. A starting dilution of 1:5,000 of the antibody is recommended for chemiluminescent substrates

Note: Higher antibody dilutions may be necessary for non-human samples. EDTA/EGTA treatment of tissues or lysates is required to see latent zymogen.

In order to obtain the best results and assay sensitivity in various techniques and preparations we recommend determining optimum working dilutions by titration.

References

1. Suzuki, A., et al., Oncogene, **19**, 5842-5850 (2000).
2. Tian, B.L., et al., Mol. Pathol., **55**, 394-397 (2002).
3. Iba, K., et al., Am. J. Pathol., **154**, 1489-1501 (1999).
4. Galliano, M. et al., J. Biol. Chem., **275**, 13933-13939 (2000).

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