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ProductInformation

Anti-ADAM-19, Cytoplasmic Domain

Developed in Rabbit Affinity Isolated Antibody

Product Number A 4101

Product Description

Anti-ADAM-19, Cytoplasmic Domain is developed in rabbit using a synthetic peptide corresponding to the cytoplasmic domain of human ADAM19 (A Disintegrin And Metalloproteinase-19) as immunogen. Affinity isolated antigen specific antibody is obtained from rabbit anti-ADAM-19 antiserum by immuno-specific purification which removes essentially all rabbit serum proteins, including immunoglobulins, which do not specifically bind to the peptide.

Anti-ADAM-19, Cytoplasmic Domain may be used for the detection and localization of human ADAM19. By immunoblotting against the reduced protein, the antibody recognizes bands at 95 kDa, 120 kDa, and breakdown products at 36 kDa and 20 kDa from cell lysates. A 50 kDa band may be seen in some lysates, perhaps representing a cleaved form of ADAM19.

ADAM19, also known as Meltrin-β), is a member of the ADAM (a disintegrin and metalloprotease-like domain) family. It has been cloned from mouse and human. ADAM19 was first described in muscle cells as a protein with homology to the fertilins (ADAMs 1 and 2). Initial observations indicated a role for ADAM19 in myoblast fusion, similar to sperm-egg fusion aided by ADAMs 1 and 2. Later works describe ADAM19 in the bone, muscle, lung, heart, brain, kidney, and a wide range of tissues. ADAM19 may be important in osteoblast differentiation, and in the intracellular processing of neuregulin. 5

The ADAMs proteins are structurally similar, possessing a signal sequence, metalloprotease domain (inactive in some ADAMs), disintegrin domain, cysteinrich domain, EGF-like repeat, type-I transmembrane domain, and cytoplasmic domain. ADAM-19 contains the canonical HExxHxxxxxH zinc metalloproteinase motif, and has been shown to be proteolytically active.

The cytoplasmic domain of ADAM19, like ADAMs 9, 12, and 15, contains SH3 ligand domains. These are thought to interact with PKC-δ, suggesting a specific regulation route for ADAM19. Also reported is a sequence of ADAM19 lacking the transmembrane and cytoplasmic domains, indicating that a soluble form is produced.

The full length ADAM19 (956 amino acids) contains a type-I transmembrane domain, with a predicted molecular mass of 105 kDa. Two shorter sequences have been reported: a 918 amino acid sequence that differs at the carboxyterminal end, and the soluble form, a 538 amino acid sequence with predicted molecular mass of 59.9 kDa. Mouse ADAM19 (920 amino acids) has a predicted molecular mass of 100.9 kDa. Human and mouse ADAM19 share approximately 84% amino acid sequence identity. ^{1, 4} Human ADAM19 maps to chromosome 5, and mouse ADAM19 maps to chromosome 11. ⁶

Reagent

Anti-ADAM-19, Cytoplasmic Domain 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 necessary 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

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