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ProductInformation

Anti-MTA1
Developed in Rabbit
Affinity Isolated Antibody

Product Number **M 7693**

Product Description

Anti-MTA1 is developed in rabbit using as immunogen a synthetic peptide corresponding to amino acids 626-641 of human MTA1, conjugated to KLH via an N-terminal added cysteine residue. The immunizing sequence is not present in the other members of the family, MTA2 and MTA3. The antibody is affinity purified on the immunizing peptide immobilized on agarose.

Anti-MTA1 recognizes specifically MTA1. Applications include immunoblotting (doublet at 75-80 kDa) and immunoprecipitation. The antibody does not cross react with MTA2 or MTA3. Staining of the MTA1 band in immunoblotting is specifically inhibited by the immunizing peptide.

Metastasis-associated genes (MTAs) comprise a novel gene family with a growing number of members. Currently, there are three known genes encoding six isoforms (MTA1, MTA1S, MTA-ZG29p, MTA2/MTA1L1, MTA3, MTA3L).[1-3] MTA1, also known as NuRD-70**,** was originally identified in rat metastatic adenocarcinomas as a differentially expressed gene.[1] It encodes a 715 amino acids protein that shares about 70% overall homology to human MTA2 and MTA3 proteins, the C-terminus being more divergent than the N-terminus.[2] Although it is clear that MTA1 is associated with cancer metastasis, its exact role in the process remains elusive.[4-6] The discovery that both MTA1 and MTA2/MTA1L1 interact with the deacetylases HDAC1 and HDAC2 within the nuclear remodeling and deacetylation complexes Mi2/NuRD, suggests that these proteins are involved in transcriptional repression.[7-9] MTA1 interacts with CAK, a component of the TFIIH regulatory complex, suggesting that MTA1 may also act as a signal transducer to mediate cross-talk between corepressor complexes and the general transcription machinery.[10] In addition, estrogen receptor (ER) is transcriptionally repressed by MTA1, this having serious implications for the development of an aggressive breast cancer phenotype through trans-activation of the HER-2 receptor by Heregulin-β1 (HRG).[11] Mechanistically, HRG regulates expression of MTA1 in the Nurd complex, which in turn represses ER-mediated transcription by recruiting HDAC's.[11] Expression in breast cancer cells of MTA1s, a naturally occurring short form of MTA1, can be also a cause for the development of the malignant phenotype. MTA1 localizes in the cytoplasm and sequesters ER in the cytoplasm, preventing ligand-induced translocation of ER and stimulating malignant phenotypes.[12] MTA3 was characterized as being part of another NuRD complex, and also shown to be an ER-regulated gene, which targets the transcription factor Snail. Snail in turn represses E-cadherin expression leading to epithelial dedifferentiation and increased metastasis.[2]

Reagent

Anti-MTA1 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 0.5-1.0 µg/ml is recommended using nuclear extracts of MCF7 cells.

By immunoprecipitation, 5-10 µg of the antibody immunoprecipitates MTA1 from extracts of 293-T cells.

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

References

1. Toh, Y., et al., J. Biol. Chem., **269**, 22958-22963 (1994).
2. Fujita, N., et al, Cell, **113**, 207-219 (2003).
3. Luo, J., et al., Nature, **408**, 377-381 (2000).
4. Nawa, A., et al., J. Cell. Biochem., **79**, 202-212 (2000).
5. Mishra, et al., J. Biol. Chem., **279**, 31, 32709-32715 (2004).
6. Mahoney, et al., Oncogene, **21**, 2162-2170 (2002).
7. Yao, Y-L., et al., J. Biol. Chem., **278**, 42560-42568 (2003).
8. Toh, Y., et al., J. Exp. Clin. Cancer Res., **19**, 105-111 (2000).
9. Xue, Y., et al., Molec. Cell, **2**, 851-861 (1998).
10. Talukder, et al., J. Biol. Chem., **278**, 11676-11685 (2003).
11. Mazumdar, A., et al., Nature Cell Biol., **3**, 30-37 (2001).
12. Kumar, R., et al., Nature, **428**, 654-657 (2002).

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