3050 Spruce Street, St. Louis, MO 63103 USA
Tel: (800) 521-8956 (314) 771-5765 Fax: (800) 325-5052 (314) 771-5757
email: techservice@sial.com sigma-aldrich.com

Product Information

Monoclonal Anti-Matrix Metalloproteinase-1
Mixed Clones
produced in mouse, purified immunoglobulin

Catalog Number **M6427**

Synonym: Anti-MMP-1

Product Description
Monoclonal Anti-Matrix Meltalloproteinase-1 (mouse IgG1κ isotype) is derived from ascites fluid from mice immunized with native human matrix metalloproteinase-1 (collagenase-1) as immunogen. The purified antibody is derived from ascites fluid by protein G affinity chromatography.

Monoclonal Anti-MMP-1 may be used for the detection and localization of human MMP-1 by imunoblotting, The antibody specifically binds to collagenase and does not cross-react with other MMP family members. By immunoblotting, the antibody detects a band at 52 kDa (unglycosylated and a band at 57 kDa (glycosylated, proform).

The matrix metalloproteinases (MMPs) are a family of at least eighteen secreted and membrane-bound zinc endopeptidases. Collectively, these enzymes can degrade all the components of the extracellular matrix, including fibrillar and non-fibrillar collagens, fibronectin, laminin and basement membrane glycoproteins. In general, the structure of MMPs is characterized by a single peptide, propeptide, and catalytic domain containing the highly conserved zinc-binding site. In addition, fibronectin-like repeats, a hinge region, and C-terminal hemopexin-like domain allow categorization of MMPs into the collagenase, gelatinase, stomelysin and membrane-type MMP subfamilies.[1-3]

MMPs contain the motif His-Glu-Xaa-His that binds zinc in the catalytic site, as well as another zinc molecule and two calcium molecules structurally. They fall within the matrixin subfamily, and are EC designated 3.4.24.x. This group also contains astacin, reprolysin, and serralysin, as well as other more divergent metalloproteinases. All MMPs are synthesized as proenzymes, and most of them are secreted from the cells as proenzymes. Thus, the activation of these proenzymes is a critical step that leads to extracellular matrix breakdown.

MMPs are considered to play an important role in wound healing, apoptosis, bone elongation, embryo development, uterine involution, angiogenesis,[4] and tissue remodeling, and in diseases such as multiple sclerosis,[2, 5] Alzheimer's,[2] malignant gliomas,[2] lupus, arthritis, periodontis, glumerulonephritis, atherosclerosis, tissue ulceration, and in cancer cell invasion and metastasis.[6] Numerous studies have shown that there is a close association between expression of various members of the MMP family by tumors and their proliferative and invasive behavior and metastaic potential. Among agents that induce MMP and TIMP production are the inflammatory cytokines TNFα and IL-1β. A marked cell type specificity is a hallmark of both MMP and TIMP gene expression (i.e., a limited number of cell types can be induced to make these proteins).

Matrix Metalloproteinase-1 (MMP-1) is also known as interstitial collagenase and collagenase-1. MMP-1 degrades fibrillar collagens types I, II, III, VII, VIII, X, aggrecan, serpins and α_2-macroglobulin. All collagenases cleave fibrillar collagens at one specific site resulting in generation of N-terminal ¾ and C-terminal ¼ fragments, which then denature to gelatin at body temperature. The substrate specificity of collagenases is variable: MMP-1 degrades type III collagen more efficiently than type I or type II collagen, whereas MMP-8 is more potent in degrading type I collagen than type III or type II collagen.[7, 8]

MMP-13, in turn degrades type II collagen 6-fold more efficiently than type I and type II collagens and displays almost 50-fold stronger gelatinolytic activity than MMP-1 and MMP-8.[9,10] Increased synthesis of MMP-1 is caused by a wide variety of reagents that include: TNF,[11,12] IL-1,[13] serum, EGF and TGF-β,[14,15,16] phorbol ester tumor promoter, PMA,[17] ECM components,[18] and polyoma and RSV infections. In contrast to these activators, several well-known antagonists, dexamethasone and all-trans-retinoic acid (RA) block the induced gene expression.[19]

The human MMP-1 gene, about 17 kb, has the chromosomal location of 11q22.2-22.3.

Reagent
Supplied in 10 mM PBS, pH 7.4, containing 0.2% BSA and 0.05% sodium azide.

Protein concentration: ~0.2 mg/mL.

Precautions and Disclaimer
This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability
For continuous use, store at 2-8 °C for up to one year. For extended storage, the solution may be aliquotted and stored –20 °C. Avoid repeated freezing and thawing.

Product Profile
Immunoblotting: the recommended working concentration is 1-2 μg/mL for 2 hours at room temperature using conditioned, serum-free medium from (TPA-treated) HFL-1 cells.

Note: 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. Borkakoti, N., *Prog. Biophy. Mol. Biol*., **70**, 73 (1998).
2. Yong, V.W., et al., *Trends in Neuroscience*, **2**1, 75 (1998).
3. Kähäri, V.M., and Saarialho-Kere, U., *Exp. Dermatol*., **6**, 199 (1997).
4. Halbert, I., et al., *Proc. Natl. Acad. Sci., USA*, **93**, 9748 (1996).
5. Chandler, S., et al., *J. Neuroimmunol*., **72**, 155 (1997).
6. Birkedal-Hansen, H., et al., *Crit. Rev. Oral. Biol. Me*d., **4**, 197 (1993).
7. Hasty, K.A., et al., *J. Exp. Med*., **159**, 1455 (1984).
8. Mallya, S.K., et al., *Biochemistry*, **29**, 10628 (1990).
9. Knauper, V., et al., *J. Biol. Chem*., **271**, 1544 (1996).
10. Mitchell, P.G., et al., *J. Clin. Invest*., **97**, 761 (1996).
11. Brenner, D.A., et al., *Nature*, **337**, 661 (1989).
12. Sciavolina, P.J., et al., *J. Biol. Chem*., **269**, 21627 (1994).
13. Dayer, J.M., et al., J*. Clin. Invest*., **77**, 645 (1986).
14. Delany, A.M., and Brickerhoff, C.E., *J. Cell Biochem*., **50**, 400 (1992).
15. Edwards, D.R., et al., *EMBO J*., **6**, 1899 (1987).
16. Partridge, N.C., et al., *Endocrinology*, **120**, 1956 (1987).
17. Brickerhoff, C.E., et al., *Biochemistry*, **21**, 2674 (1982).
18. Shapiro, S.D., et al., *J. Biol. Chem*., **268**, 8170 (1993).
19. Brickerhoff, C.E., et al., *N. Engl. J. Med*., **303**, 432 (1980).

KAA,PHC 04/08-1