

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

Anti-Matrix Metalloproteinase-9 produced in goat, affinity isolated antibody

Catalog Number M9570

Product Description

Anti-Matrix Metalloproteinase-9 (MMP-9) is produced in goat using purified, NSO-derived, recombinant mouse matrix metalloproteinase-9 specific IgG. MMP-9 specific IgG was purified by mouse MMP-9 affinity chromatography.

Anti-MMP-9 may be used for the detection and localization of MMP-9 by various immunochemical techniques such as immunoblotting, immunoprecipitation, and immunohistochemistry.

Anti-MMP-9 specifically binds to mouse MMP-9. This antibody recognizes pro and active mouse MMP-9. By immunoblotting, the antibody shows ~10% cross-reactivity with recombinant human MMP-9 and less than 2% cross-reactivity with recombinant human MMP-1, MMP-2, MMP-3, MMP-3, MMP-7, MMP-8, MMP-10, MMP-12, and MMP-13.

The matrix metalloproteinases (MMPs) are a family of at least eighteen secreted and membrane-bound zincendopeptidases. 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, a signal peptide, a propeptide, and a catalytic domain containing the highly conserved zinc-binding site characterizes the structure of the MMPs. In addition, fibronectin-like repeats, a hinge region, and a 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-X-X-His (X represents any amino acid) 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.

Mouse MMP-9 (gelatinase-B) is also known as 105 kDa gelatinase. Human MMP-9 (gelatinase-B) is also known as 92 kDa gelatinase and 92 kDa type IV collagenase. The expression of MMP-9 is more restricted than MMP-2. MMP-9 is produced by keratinocytes and stored in the granules of neutrophils and eosinophils. but not expressed by dermal fibroblasts. MMP-9 degrades gelatin, type IV, V and XIV collagens, α₂-macroglobulin, elastin, vitronectin and proteoglycans. MMP-2 and MMP-9 are thought to play an important role in the final degradation of fibrillar collagens after initial cleavage by collagenases. Interestingly, reports provide evidence that the gelatinases also possess collagenolytic activity. MMP-2 cleaves native type I collagen to N-terminal 3/4 and C-terminal \(\frac{1}{4} \) fragments identical to those generated by collagenases. In addition, MMP-9, which is expressed specifically by osteoclasts during murine fetal development and in adult human bone, has shown to cleave type I, II and V collagens in the N-terminal nonhelical telopeptide.8 Because of their ability to initiate and continue degradation of fibrillar collagen type I, MMP-2 and MMP-9 play an important role in the remodeling of collagenous ECM (extracellular matrix). In general, inducers such as PMA, EGF, IL-1 β , or TNF α enhance MMP-9 production without altering MMP-2 levels, whereas $TGF\beta$, which down-regulates most MMPs, enhances the expression of both MMP-2 and MMP-9.

Reagent

Supplied lyophilized from a 0.2 µm filtered solution in phosphate buffered saline containing 5% trehalose.

Storage/Stability

Store at $-20\,^{\circ}$ C. For extended storage, the reconstituted solution can be aliquotted and stored at $-20\,^{\circ}$ C or below. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use.

Preparation Instructions

Reconstitute with sterile phosphate buffered saline (PBS). If I mL of phosphate buffered saline is used, the antibody concentration will be 0.1 mg/mL.

Product Profile

Immunoblotting: a working antibody concentration of 0.1 μg/mL is recommended to detect mouse MMP-9. The detection limit for recombinant mouse MMP-9 is ~2 ng/lane under non-reducing and reducing conditions.

 $\frac{Immunoprecipitation:}{25~\mu g/mL} \ is \ recommended \ to \ immunoprecipitate$ recombinant mouse MMP-9 from conditioned media of transfected NSO cells.

 $\frac{Immunohistochemistry}{5-15~\mu g/mL} \ \ a \ working \ concentration \ of \\ 5-15~\mu g/mL \ \ is \ recommended \ to \ detect \ mouse \ MMP-9 \ in \\ cells \ \ or \ tissues.$

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

- Borkakoti, N., *Prog. Biophy. Mol. Biol.*, **70**, 73 (1998).
- 2. Yong, V.W., et al., *Trends in Neuroscience*, **21**, 75 (1998).
- Kähäri, V.M., and Saarialho-Kere, U., *Exp. Dermatol.*, 6, 199 (1997).
- Halbert, I., et al., Proc. Natl. Acad. Sci., USA, 93, 9748 (1996).
- 5. Chandler, S., et al., *J. Neuroimmunol.*, **72**, 155 (1997).
- Birkedal-Hansen, H., et al., Crit. Rev. Oral. Biol. Med., 4, 197 (1993).
- 7. Aimes, R.T., and Quigley, J.P., *J.Biol. Chem.*, **270**, 5872 (1995).
- 8. Okada, Y., et al., *Lab Invest.*, **72**, 311 (1995).
- 9. Borden, P., and Heller, R.A., *Crit. Rev. Eukar. Gene Exp.*, **7**, 159 (1997).

SC,PHC 04/12-1