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Product Information

MMP-9, human
recombinant, expressed in HEK 293 cells

Catalog Number **SAE0077**
Storage Temperature –20 °C

EC 3.4.24.35
Synonyms: GELBCLG4B, Gelatinase B, Gelatinase, MANDP2, Matrix Metalloproteinase-9, Type IV collagenase, GELB
Uniprot: P14780

Product Description

Matrix Metalloproteinase-9 (MMP-9) is a member of the matrix metalloproteinase (MMP) family of proteins. MMPs participate in the breakdown of extracellular matrix in normal physiological processes, such as embryonic development, reproduction, and tissue remodeling, as well as in disease processes, such as arthritis and metastasis.[1] MMP-9 is secreted from neutrophils, macrophages, and a number of transformed cells. MMP-9 is the most complex family member in terms of domain structure and regulation of its activity.[2]

Structurally, MMP-9 may be divided into five distinct domains:[3,4]

- a pro-domain which is cleaved upon activation
- a gelatin-binding domain consisting of three contiguous fibronectin type II units
- a catalytic domain containing the zinc binding site
- a proline-rich linker region
- a carboxyl terminal hemopexin-like domain

Studies in rhesus monkeys suggest that MMP-9 is involved in interleukin-8 (IL-8)-induced mobilization of hematopoietic progenitor cells from bone marrow, and murine studies suggest a role in tumor-associated tissue remodeling.[2] Thrombospondins, intervertebral disc proteins, regulate the effective levels of MMP-2 and MMP-9, which are key effectors of extracellular matrix (ECM) remodeling.[5]

MMP-9 degrades various substrates, including gelatin, collagen types IV and V, and elastin.[3] MMP-9 is involved in various autoimmune diseases, such as systemic lupus erythematosus, rheumatoid arthritis, and multiple sclerosis. MMP-9 thus may be regarded as a potential therapeutic target.[6]

As with most MMPs, MMP-9 is secreted as an inactive pro-protein which is activated when cleaved by extracellular proteinases.[1] This recombinant MMP-9 is **not pre-activated**.

This product is expressed in human HEK 293 cells as a glycoprotein with a calculated molecular mass of 76 kDa (amino acids 20-707). The DTT-reduced protein migrates as a ~92 kDa polypeptide on SDS-PAGE because of glycosylation. This protein is produced in human cells, without the use of serum. The human cells expression system allows human-like glycosylation and folding, and often supports higher specific activity of the protein. This recombinant protein is expressed without artificial tags.

This product is supplied in a 0.22 μm-filtered solution, containing 25 mM Trizma®, pH 7.5, with 10 mM $CaCl_2$, 150 mM NaCl, and 0.05% Brij® 35.

The specific activity of this recombinant human MMP-9 is measured by its ability to cleave the fluorogenic peptide Mca-Pro-Leu-Gly-Leu-Dpa-Ala-Arg-NH_2 (Mca-PLGL-Dpa-AR-NH_2; Mca = 7-Methoxycoumarin-4-yl)acetyl; Dpa = *N*-3-(2, 4-Dinitrophenyl)-L-2,3-diaminopropionyl).

Specific activity: ≥1,300 pmol/min/μg

Purity: ≥95% (SDS-PAGE)

(The related product SAE0078 is an AMPA-treated, pre-activated form of this recombinant human MMP-9.)

Precautions and Disclaimer

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

Preparation Instructions

This product can be activated *in vitro* by overnight incubation with 4-aminophenylmercuric acetate (APMA, e.g. Catalog Number A9563) at 1 mM APMA.

Storage/Stability
Store the product at –20 °C. The product retains its activity for at least 2 years as supplied. After initial thawing it is recommended to store the protein in working aliquots at –20 °C.

References

1. Vos, M.C. *et al.*, *Reprod. Biol. Endocrinol.*, **12**, 12 (2014).
2. Pruijt, J.F. *et al.*, *Proc. Nat. Acad. Sci. USA*, **96(19)**, 10863-10868 (1999).
3. Somerville, R.P. *et al.*, *Genome Biol.*, **4(6)**, 216-216 (2003).
4. Tallant, C. *et al.*, *Biochim. Biophys. Acta*, **1803(1)**, 20-28 (2010).
5. Rodriguez-Manzaneque, J.C. *et al.*, *Proc. Nat. Acad. Sci. USA*, **98(22)**, 12485-12490 (2001).
6. Chang, Y.H. *et al.*, *Clin. Biochem.*, **41(12)**, 955-959 (2008).

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