Recombinant Mouse Legumain/LGMN Protein (His Tag)

Catalog Number: PKSM040307

Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

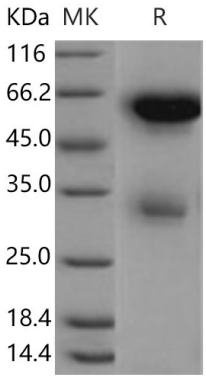
Description

Species	Mouse
Source	HEK293 Cells-derived Mouse Legumain/LGMN protein Val 18-Tyr 435, with an C-terminal His
Calculated MW	49.8 kDa
Observed MW	55 kDa
Accession	NP_035305.1
Bio-activity	Measured by its ability to cleave the fluorogenic peptide substrate, N-carbobenzyloxy-Ala-Ala-Asn-7-amido-4-methyl coumarin(Z-AAN-AMC). The specific activity is > 350 pmoles/min/μg. (Activation description: The enzyme achieves its activity under acidic pH)

Properties

Purity	> 75 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per μg of the protein as determined by the LAL method.
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from sterile 25mM Tris, 0.15M NaCl, 20% Glycerol, pH 7.5 Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 75 % as determined by reducing SDS-PAGE.

Background

The Mammalian Legumain, also known as LGMN, also called asparaginyl endopeptidase (AEP), is a cysteine protease belonging to peptidase family C13 with a strict specificity for hydrolysis of asparaginyl bonds. Known previously only from plants and invertebrates, Legumain is discovered as a lysosomal endopeptidase in mammals. Mammalian Legumain is a cysteine endopeptidase, inhibited by iodoacetamide and maleimides, but unaffected by compound E64. The Mammalian Legumain is involved in the processing of bacterial peptides and endogenous proteins for MHC class II presentation in the lysosomal/endosomal systems. Legumain has been observed to be highly expressed in several types of solid tumors. It was demonstrated in membrane-associated vesicles concentrated at the invadopodia of tumor cells and on cell surfaces where it colocalized with integrins. Legumain was demonstrated to activate progelatinase A. Cells overexpressing Legumain possessed increased migratory and invasive activity in vitro and adopted an invasive and metastatic phenotype in vivo, inferring significance of Legumain in tumor invasion and metastasis. In addition, Legumain is expressed in both murine and human atherosclerotic lesions. The macrophage-specific expression of Legumain in vivo and ability of Legumain to induce chemotaxis of monocytes and endothelial cells in vitro suggest that Legumain may play a functional role in atherogenesis.

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