

Recombinant Human CMA1/Chymase 1 Protein (His Tag)



Catalog Number:PKSH031112

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

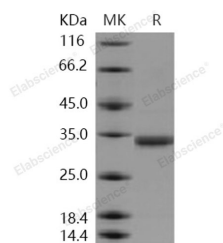
Description

| | |
|------------------------------------|--------------------------|
| Synonyms | chymase;CYH;MCT1 |
| Species | Human |
| Expression Host | Baculovirus-Insect Cells |
| Sequence | Met 1-Asn 247 |
| Accession | P23946 |
| Calculated Molecular Weight | 26.6 kDa |
| Observed molecular weight | 33 kDa |
| Tag | C-His |

Properties

| | |
|-----------------------|--|
| Purity | > 92 % as determined by reducing SDS-PAGE. |
| Endotoxin | < 1.0 EU per µg of the protein as determined by the LAL method. |
| Storage | Store at < -20°C, stable for 6 months. Please minimize freeze-thaw cycles. |
| Shipping | This product is provided as liquid. It is shipped at frozen temperature with blue ice/gel packs. Upon receipt, store it immediately at < - 20°C. |
| Formulation | Supplied as sterile solution of 20mM Tris, 500mM NaCl, pH 7.4, 10% glycerol |
| Reconstitution | Not Applicable |

Data



> 92 % as determined by reducing SDS-PAGE.

Background

Chymotrypsin C (abbreviated for CTRC), also known as caldecrin or elastase4, is a digestive enzyme of the peptidase S1 family. This enzyme is synthesized as an inactivate chymotrypsinogen. On cleavage by trypsin into two parts that activate each other by removing two small peptides in a trans-proteolysis, chymotrypsin C produced. N-linked glycosylation of human CTRC is required for efficient folding and secretion, however, the N-linked glycan is unimportant for enzyme activity or inhibitor binding. It has been proposed that CTRC is a key regulator of digestive zymogen activation and a physiological co-activator of digestive carboxypeptidases proCPA1 and proCPA2. Mutations that abolish activity or secretion of CTRC increase the risk for chronic pancreatitis. It's speculated that CTRC might regulate pancreatic cancer cell migration in relation to cytokeratin 18 expression. The pancreatic cancer cell migration ability was downregulated in pancreatic cancer Aspc-1 cells that overexpressed CTRC, whereas the cell migration ability was upregulated in Aspc-1 cells in which CTRC was suppressed.

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