

Recombinant Human Chymotrypsin C Protein (His Tag)



Catalog Number:PKSH031117

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

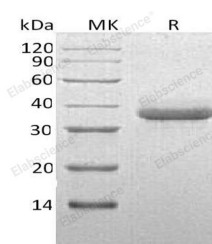
Description

Synonyms	Chymotrypsin-C;Caldecrin;CTRC;CLCR
Species	Human
Expression Host	HEK293 Cells
Sequence	Met 1-Leu 268
Accession	Q99895
Calculated Molecular Weight	29.3 kDa
Observed molecular weight	36 kDa
Tag	C-His
Bioactivity	Measured by its ability to cleave the fluorogenic peptide substrate, SUCAlaAlaPro-PheAMC. The specific activity is > 300 pmol/min/μg.

Properties

Purity	> 97 % 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 PBS, pH 7.4 Normally 5 % - 8 % trehalose, mannitol and 0.01 % Tween80 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



> 97 % 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

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secretion of CTSC increase the risk for chronic pancreatitis. It's speculated that CTSC 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 CTSC; whereas the cell migration ability was upregulated in Aspc-1 cells in which CTSC was suppressed.

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