Recombinant Protein Technical Manual

Recombinant Human Chymotrypsin C Protein (His Tag)(Active)

RPES1292

Product Data:

Product SKU: RPES1292

Size: 10µg

Species: Human

Expression host: HEK293 Cells

Uniprot: Q99895

Protein Information:

Molecular Mass:	29.3 kDa
AP Molecular Mass:	36 kDa
Tag:	C-His
Bio-activity:	Measured by its ability to cleave the fluorogenic peptide substrate, SUC-Ala-Ala-Pro-Phe-AMC. The specific activity is >300 pmol/min/µg.
Purity:	> 97 % as determined by reducing SDS-PAGE.
Endotoxin:	< 1.0 EU per µg of the protein as determined by the LAL method.
Storage:	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℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ 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
Reconstitution:	Please refer to the printed manual for detailed information.
Application:	
Synonyms:	Chymotrypsin-C; Caldecrin; CTRC; CLCR

Immunogen Information:

Sequence: Met 1-Leu 268

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 cells that overexpressed CTRC, whereas the cell migration ability was upregulated in Aspc cells in which CTRC was suppressed.