

Recombinant Human ECE1 Protein (His Tag)

Catalog Number:PKSH031372



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

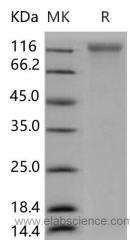
Description

Synonyms	Endothelin-converting enzyme 1; ECE-1
Species	Human
Expression Host	HEK293 Cells
Sequence	Gln 90-Trp 770
Accession	P42892-1
Calculated Molecular Weight	80 kDa
Observed molecular weight	125 kDa
Tag	N-His

Properties

Purity	> 95 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per µg 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°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
Reconstitution	Please refer to the printed manual for detailed information.

Data



Background

Endothelin-converting enzyme 1, also known as ECE-1, is a single-pass type I I membrane protein which belongs to the peptidase M13 family. ECE-1 converts big endothelin-1 to endothelin-1. ECE-1 is a membrane metalloprotease that generates endothelin from its direct precursor big endothelin. Four isoforms of ECE-1 are produced from a single gene through the use of alternate promoters. These isoforms share the same extracellular catalytic domain and contain unique cytosolic tails, which results in their specific subcellular targeting. All isoforms of ECE-1 are expressed in umbilical vein endothelial cells, polynuclear neutrophils, fibroblasts, atrium cardiomyocytes and ventricles. Isoforms A, B and C of ECE-1 are also expressed in placenta, lung, heart, adrenal gland and pheochromocytoma; isoforms A and C of ECE-1 in liver, testis and small intestine; isoform B, C and D of ECE-1 in endothelial cells and umbilical vein smooth muscle cells; isoforms C and D in saphenous vein cells, and isoform C in kidney. Defects in ECE1 are a cause of Hirschsprung disease, cardiac defects and autonomic dysfunction. It is a form of Hirschsprung disease with skip-lesions defects, craniofacial abnormalities and other dysmorphic features, and autonomic dysfunction.

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