Recombinant Human CART/CARTPT Protein

Catalog Number: PKSH030681

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

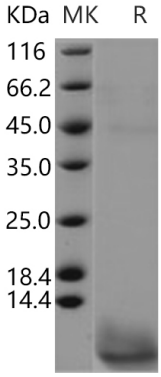
Description

Species	Human
Source	HEK293 Cells-derived Human CART/CARTPT protein Met 1-Leu116
Calculated MW	10.6 kDa
Observed MW	11 kDa
Accession	NP_004282.1
Bio-activity	Not validated for activity

Properties

Purity	> 90 % 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 ℃. 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 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



> 90 % as determined by reducing SDS-PAGE.

Background

Z-farnesyl diphosphate synthase (FDPS) is an enzyme belonging to the family of transferases; specifically those transferring aryl or alkyl groups other than methyl groups. Z-farnesyl diphosphate synthase (FDPS) functions as key enzyme in isoprenoid biosynthesis which catalyzes the formation of farnesyl diphosphate; a precurcor for several classes of essential metabolites. FDPS catalyzes the production of geranyl pyrophosphate and farnesyl pyrophosphate from isopentenyl pyrophosphate and dimethylallyl pyrophosphate. The resulting product; farnesyl pyrophosphate; is a key intermediate in cholesterol and sterol biosynthesis; a substrate for protein farnesylation and geranylgeranylation; and a ligand or agonist for certain hormone receptors and growth receptors. Drugs that inhibit this enzyme prevent the post-translational modifications of small GTPases and have been used to treat diseases related to bone resorption. Functions of FDPS may be inactivated by interferon-induced RSAD2. This inactivation may result of disruption of lipid rafts at the plasma membrane; and thus have an antiviral effect since many enveloped viruses need lipid rafts to bud efficiently out of the cell.

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