



# Recombinant Protein Technical Manual

## Recombinant Human CART/CARTPT Protein

RPES0558

### Product Data:

**Product SKU:** RPES0558

**Size:** 20µg

**Species:** Human

**Expression host:** HEK293 Cells

**Uniprot:** NP\_004282.1

### Protein Information:

**Molecular Mass:** 10.6 kDa

**AP Molecular Mass:** 11 kDa

**Tag:**

**Bio-activity:**

**Purity:** > 90 % 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°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.

**Application:**

**Synonyms:** CART

## Immunogen Information:

**Sequence:** Met 1-Leu116

## 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 precursor 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.