

Recombinant Human FDPS Protein (His Tag)

Catalog Number: PKSH030685

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

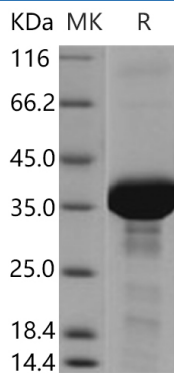
Description

Species	Human
Source	E.coli-derived Human FDPS protein Met 1-Lys 353, with an N-terminal His
Calculated MW	42.4 kDa
Observed MW	38 kDa
Accession	NP_001129294.1
Bio-activity	Not validated for activity

Properties

Purity	> 85 % as determined by reducing SDS-PAGE.
Endotoxin	Please contact us for more information.
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 7.5 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



> 85 % as determined by reducing SDS-PAGE.

Background

For Research Use Only

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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.