Data Sheet (Cat.No.TMPY-00475)

TargetMol

PPT1 Protein, Human, Recombinant (His)

General Information

Synonyms:	PPT;INCL;palmitoyl-protein thioesterase 1;CLN1;PPT1
Protein Construction:	A DNA sequence encoding the human PPT1 (P50897-1)(Met1-Gly306) was expressed with a C-terminal polyhistidine tag. Predicted N terminal: Asp 28
Species:	Human
Expression Host:	HEK293 Cells
Accession:	P50897-1
Molecular Weight:	32.7 kDa (predicted); 35-41 kDa (reducing conditions)

QC Testing

Biological Activity:	Activity testing is in progress. It is theoretically active, but we cannot guarantee it. If you require protein activity, we recommend choosing the eukaryotic expression version first.
Purity:	> 90 % as determined by SDS-PAGE
Endotoxin:	< 1.0 EU/μg of the protein as determined by the LAL method.
Formulation:	Lyophilized from a solution filtered through a 0.22 μm filter, containing PBS, pH 7.4. Typically, a mixture containing 5% to 8% trehalose, mannitol, and 0.01% Tween 80 is incorporated as a protective agent before lyophilization.

Preparation and Storage

Reconstitution:

A Certificate of Analysis (CoA) containing reconstitution instructions is included with the products. Please refer to the CoA for detailed information.

Stability & Storage:

It is recommended to store recombinant proteins at -20°C to -80°C for future use. Lyophilized powders can be stably stored for over 12 months, while liquid products can be stored for 6-12 months at -80°C. For reconstituted protein solutions, the solution can be stored at -20°C to -80°C for at least 3 months. Please avoid multiple freeze-thaw cycles and store products in aliquots.

Actual storage temperature shall be subject to the COA.

Shipping:

In general, lyophilized powders are shipped with blue ice, while solutions are shipped with dry ice.

Protein Background

Mutations in the depalmitoylating enzyme gene, PPT1, cause the infantile form of Neuronal Ceroid Lipofuscinosis (NCL), an early onset neurodegenerative disease. Mutations in palmitoyl protein thioesterase-1 (PPT1) have been found to cause the infantile form of neuronal ceroid lipofuscinosis, which is a lysosomal storage disorder characterized by impaired degradation of fatty acid-modified proteins with accumulation of amorphous granular deposits in cortical neurons, leading to mental retardation and death. PPT1 catalyzes the cleavage of thioester

linkages in S-acylated (palmitoylated) proteins and its deficiency leads to abnormal accumulation of thioesterified polypeptides (ceroid) in lysosomes causing INCL pathogenesis.

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