Recombinant Human TPP1/CLN2 Protein (His Tag)

Catalog Number: PKSH030613

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

Description	
Species	Human
Source	Baculovirus-Insect Cells-derived Human TPP1/CLN2 protein Met 1-Pro 563, with an C-
	terminal His
Calculated MW	60.7 kDa
Observed MW	60 kDa
Accession	AAH14863.1
Bio-activity	Measured by the cleavage of AlaAlaPheAMC. The specific activity is > 850
	pmoles/min/µg.
Properties	
Purity	>95 % 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
	°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of
	reconstituted samples are stable at $< -20^{\circ}$ C for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from sterile 20mM Tris, 500mM NaCl, pH 7.4, 10% glycerol
	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	

KDa	MK	R
116	-	
66.2	-,	-
45.0	-	
35.0	-	
25.0	-	
18.4 14.4	=	

> 95 % as determined by reducing SDS-PAGE.

Background

For Research Use Only

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Tripeptidyl-peptidase 1 (TPP1 / CLN2) is a member of the sedolisin family of serine proteases. The protease functions in the lysosome to cleave N-terminal tripeptides from substrates, and has weaker endopeptidase activity. It is synthesized as a catalytically-inactive enzyme which is activated and auto-proteolyzed upon acidification. TPP1 / CLN2 May act as a non-specific lysosomal peptidase which generates tripeptides from the breakdown products produced by lysosomal proteinases. Defects in TPP1 / CLN2 are the cause of neuronal ceroid lipofuscinosis type 2 (CLN2), a form of neuronal ceroid lipofuscinosis which is associated with the failure to degrade specific neuropeptides and a subunit of ATP synthase in the lysosome. Neuronal ceroid lipofuscinoses are progressive neurodegenerative, lysosomal storage diseases characterized by intracellular accumulation of autofluorescent liposomal material, and clinically by seizures, dementia, visual loss, and/or cerebral atrophy.