

Recombinant Human PTPMT1 Protein (His Tag)

Catalog No. PKSH030769

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

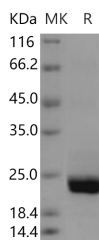
Description

Synonyms	DUSP23;FLJ46081;MOSP;PLIP;PNAS-129
Species	Human
Expression Host	E.coli
Sequence	Lys 28-Thr 201
Accession	Q8WUK0-1
Calculated Molecular Weight	21.7 kDa
Observed molecular weight	20 kDa
Tag	N-His
Bioactivity	Measured by its ability to cleave pNPP. The specific activity is > 200 pmoles/min/μg.

Properties

Purity	> 94 % 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, 10% glycerol, 1mM DTT, pH 7.5 Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 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



> 94 % as determined by reducing SDS-PAGE.

Background

PTPMT1 (PTP localized to the Mitochondrion 1) is a member of the protein tyrosine phosphatase superfamily that is

For Research Use Only

localized exclusively to the mitochondrion. It has been recently reported that PTPMT1 dephosphorylates phosphatidylglycerol phosphate, an essential intermediate of cardiolipin biosynthesis. PTPMT1 deficiency in mouse embryonic fibroblasts compromises mitochondrial respiration and results in abnormal mitochondrial morphology. Lipid analysis of PTPMT1-deficient fibroblasts reveals an accumulation of PGP along with a concomitant decrease in phosphatidylglycerol. Modulation of mitochondrial ATP synthesis by PTPMT1 suggests a novel approach for the treatment of pancreatic cancers, which represent some of the deadliest forms of human tumors. The gluttony of cancer cells for energy is well established, and with the development of a modulator of expression, one may hope that we could also achieve the synthetic induction of PTPMT1 expression. It would then be expected that this effect would attenuate, if not abolish, the growth of pancreas-derived tumor cells and support the establishment of a novel regimen for pancreatic cancers.