

PITRM1 Antibody (Center)
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP12390c

Specification

PITRM1 Antibody (Center) - Product Information

Application	WB,E
Primary Accession	Q5JRX3
Other Accession	NP_055704.2
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	117413
Antigen Region	490-518

PITRM1 Antibody (Center) - Additional Information

Gene ID 10531

Other Names

Presequence protease, mitochondrial, hPreP, 3424-, Pitrilysin metalloproteinase 1, Metalloprotease 1, hMP1, PITRM1, KIAA1104, MP1

Target/Specificity

This PITRM1 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 490-518 amino acids from the Central region of human PITRM1.

Dilution

WB~~1:1000

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

PITRM1 Antibody (Center) is for research use only and not for use in diagnostic or therapeutic procedures.

PITRM1 Antibody (Center) - Protein Information

Name PITRM1 ([HGNC:17663](#))

Function Metalloendopeptidase of the mitochondrial matrix that functions in peptide cleavage

and degradation rather than in protein processing (PubMed:[10360838](#), PubMed:[16849325](#), PubMed:[19196155](#), PubMed:[24931469](#)). Has an ATP-independent activity (PubMed:[16849325](#)). Specifically cleaves peptides in the range of 5 to 65 residues (PubMed:[19196155](#)). Shows a preference for cleavage after small polar residues and before basic residues, but without any positional preference (PubMed:[10360838](#), PubMed:[19196155](#), PubMed:[24931469](#)). Degrades the transit peptides of mitochondrial proteins after their cleavage (PubMed:[19196155](#)). Also degrades other unstructured peptides (PubMed:[19196155](#)). It is also able to degrade amyloid-beta protein 40, one of the peptides produced by APP processing, when it accumulates in mitochondrion (PubMed:[16849325](#), PubMed:[24931469](#), PubMed:[26697887](#)). It is a highly efficient protease, at least toward amyloid-beta protein 40 (PubMed:[24931469](#), PubMed:[29764912](#), PubMed:[29383861](#)). Cleaves that peptide at a specific position and is probably not processive, releasing digested peptides intermediates that can be further cleaved subsequently (PubMed:[24931469](#)). It is also able to degrade amyloid-beta protein 42 (PubMed:[29764912](#)).

Cellular Location

Mitochondrion. Mitochondrion matrix

Tissue Location

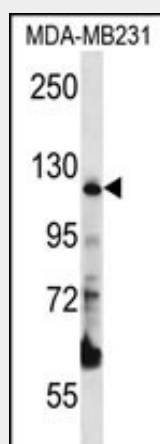
Widely expressed. Expressed at higher level in muscle and heart compared to brain, pancreas, liver, lung and placenta

PITRM1 Antibody (Center) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

PITRM1 Antibody (Center) - Images



PITRM1 Antibody (Center) (Cat. #AP12390c) western blot analysis in MDA-MB231 cell line lysates (35ug/lane). This demonstrates the PITRM1 antibody detected the PITRM1 protein (arrow).

PITRM1 Antibody (Center) - Background

ATP-independent protease that degrades mitochondrial transit peptides after their cleavage. Also degrades other unstructured peptides. Specific for peptides in the range of 10 to 65 residues. Able to degrade amyloid beta A4 (APP) protein when it accumulates in mitochondrion, suggesting a link with Alzheimer disease. Shows a preference for cleavage after small polar residues and before basic residues, but without any positional preference.

PITRM1 Antibody (Center) - References

Yoshida, T., et al. Int. J. Mol. Med. 25(4):649-656(2010)
Pinho, C.M., et al. Neurosci. Lett. 469(2):204-208(2010)
Oguri, M., et al. Am. J. Hypertens. 23(1):70-77(2010)
Yoshida, T., et al. Int. J. Mol. Med. 24(4):539-547(2009)
Chow, K.M., et al. Biochemistry 48(13):2868-2877(2009)