

Mouse Pink1 Antibody (N-term)
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP14257a

Specification

Mouse Pink1 Antibody (N-term) - Product Information

Application	WB,E
Primary Accession	O99MQ3
Other Accession	NP_081156.2
Reactivity	Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	63181
Antigen Region	118-146

Mouse Pink1 Antibody (N-term) - Additional Information

Gene ID 68943

Other Names

Serine/threonine-protein kinase PINK1, mitochondrial, BRPK, PTEN-induced putative kinase protein 1, Pink1

Target/Specificity

This Mouse Pink1 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 118-146 amino acids from the N-terminal region of mouse Pink1.

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

Mouse Pink1 Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

Mouse Pink1 Antibody (N-term) - Protein Information

Name Pink1

Function Serine/threonine-protein kinase which protects against mitochondrial dysfunction during

cellular stress by phosphorylating mitochondrial proteins such as PRKN and DNM1L, to coordinate mitochondrial quality control mechanisms that remove and replace dysfunctional mitochondrial components (PubMed:[24652937](#), PubMed:[24784582](#), PubMed:[25474007](#), PubMed:[32484300](#)). Depending on the severity of mitochondrial damage and/or dysfunction, activity ranges from preventing apoptosis and stimulating mitochondrial biogenesis to regulating mitochondrial dynamics and eliminating severely damaged mitochondria via mitophagy (By similarity). Mediates the translocation and activation of PRKN at the outer membrane (OMM) of dysfunctional/d depolarized mitochondria (PubMed:[24652937](#), PubMed:[24784582](#), PubMed:[25474007](#), PubMed:[32484300](#)). At the OMM of damaged mitochondria, phosphorylates pre-existing polyubiquitin chains at 'Ser-65', the PINK1-phosphorylated polyubiquitin then recruits PRKN from the cytosol to the OMM where PRKN is fully activated by phosphorylation at 'Ser-65' by PINK1 (PubMed:[24652937](#), PubMed:[24784582](#), PubMed:[25474007](#), PubMed:[32484300](#)). In damaged mitochondria, mediates the decision between mitophagy or preventing apoptosis by promoting PRKN-dependent poly- or monoubiquitination of VDAC1; polyubiquitination of VDAC1 by PRKN promotes mitophagy, while monoubiquitination of VDAC1 by PRKN decreases mitochondrial calcium influx which ultimately inhibits apoptosis (By similarity). When cellular stress results in irreversible mitochondrial damage, functions with PRKN to promote clearance of damaged mitochondria via selective autophagy (mitophagy) (PubMed:[24784582](#), PubMed:[25474007](#)). The PINK1-PRKN pathway also promotes fission of damaged mitochondria by phosphorylating and thus promoting the PRKN-dependent degradation of mitochondrial proteins involved in fission such as MFN2 (By similarity). This prevents the refusion of unhealthy mitochondria with the mitochondrial network or initiates mitochondrial fragmentation facilitating their later engulfment by autophagosomes (By similarity). Also promotes mitochondrial fission independently of PRKN and ATG7-mediated mitophagy, via the phosphorylation and activation of DNM1L (PubMed:[32484300](#)). Regulates motility of damaged mitochondria by promoting the ubiquitination and subsequent degradation of MIRO1 and MIRO2; in motor neurons, this likely inhibits mitochondrial intracellular anterograde transport along the axons which probably increases the chance of the mitochondria undergoing mitophagy in the soma (By similarity). Required for ubiquinone reduction by mitochondrial complex I by mediating phosphorylation of complex I subunit NDUFA10 (PubMed:[24652937](#)). Phosphorylates LETM1, positively regulating its mitochondrial calcium transport activity (PubMed:[29123128](#)).

Cellular Location

Mitochondrion outer membrane {ECO:0000250|UniProtKB:Q9BXM7}; Single-pass membrane protein. Mitochondrion inner membrane; Single-pass membrane protein. Cytoplasm, cytosol {ECO:0000250|UniProtKB:Q9BXM7} Note=Localizes mostly in mitochondrion and the two smaller proteolytic processed fragments localize mainly in cytosol. When mitochondria lose mitochondrial membrane potential following damage, PINK1 import is arrested, which induces its accumulation in the outer mitochondrial membrane, where it acquires kinase activity {ECO:0000250|UniProtKB:Q9BXM7}

Tissue Location

High levels expressed in testis, lower levels in brain, heart, lung, liver and kidney.

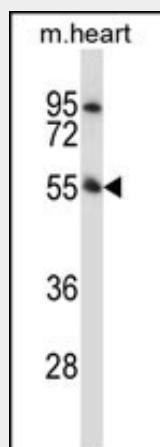
Mouse Pink1 Antibody (N-term) - Protocols

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](#)

Mouse Pink1 Antibody (N-term) - Images



Mouse Pink1 Antibody (N-term) (Cat. #AP14257a) western blot analysis in mouse heart tissue lysates (35ug/lane). This demonstrates the Pink1 antibody detected the Pink1 protein (arrow).

Mouse Pink1 Antibody (N-term) - Background

Pink1 protects against mitochondrial dysfunction during cellular stress, potentially by phosphorylating mitochondrial proteins (By similarity).

Mouse Pink1 Antibody (N-term) - References

Matsuda, N., et al. J. Cell Biol. 189(2):211-221(2010)
Kawajiri, S., et al. FEBS Lett. 584(6):1073-1079(2010)
Kim, K.H., et al. Neurosci. Lett. 468(3):272-276(2010)
Morais, V.A., et al. EMBO Mol Med 1(2):99-111(2009)
Chiba, M., et al. Cytogenet. Genome Res. 126(3):259-270(2009)