10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

Mouse PINK Antibody (Center) Blocking peptide

Synthetic peptide
Catalog # BP11315c

Specification

Mouse PINK Antibody (Center) Blocking peptide - Product Information

Primary Accession Q99MQ3

Mouse PINK Antibody (Center) Blocking peptide - Additional Information

Gene ID 68943

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

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

Mouse PINK Antibody (Center) Blocking peptide - 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/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 PINK Antibody (Center) Blocking peptide - Protocols

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

- Blocking Peptides

Mouse PINK Antibody (Center) Blocking peptide - Images

Mouse PINK Antibody (Center) Blocking peptide - Background

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

Mouse PINK Antibody (Center) Blocking peptide - 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)