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

PINK1 Recombinant Rabbit mAb

Catalog # AP99573

Product Information

Application	WB, IHC-P, IHC-F, IF, IP, E
Primary Accession	Q9BXM7
Other Accession	Q9BXM7
Reactivity	Rat, Human, Mouse
Host	Rabbit
Clonality	Recombinant
Calculated MW	62769
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SIMILARITY	Belongs to the protein kinase superfamily. Ser/Thr protein kinase family.
SUBUNIT	Interacts with PRKN. Interacts with FBXO7. Forms a complex with PRKN and PARK7.
Post-translational modifications	Autophosphorylation at Ser-228 and Ser-402 is essential for Parkin/PRKN recruitment to depolarized mitochondria. Two shorter forms of 55 kDa and 48 kDa seem to be produced by proteolytic cleavage and localize mainly in cytosol.
DISEASE	Defects in PINK1 are the cause of Parkinson disease type 6 (PARK6) [MIM:605909]. A neurodegenerative disorder characterized by parkinsonian signs such as rigidity, resting tremor and bradykinesia. A subset of patients manifest additional symptoms including hyperreflexia, autonomic instability, dementia and psychiatric disturbances. Symptoms show diurnal fluctuation and can improve after sleep.
Background Descriptions	This gene encodes a serine/threonine protein kinase that localizes to mitochondria. It is thought to protect cells from stress-induced mitochondrial dysfunction. Mutations in this gene cause one form of autosomal recessive early-onset Parkinson disease. [provided by RefSeq, Jul 2008],

Additional Information

Gene ID	65018
Other Names	PINK1_HUMAN; PINK1; BRPK; PTEN-induced putative kinase protein 1; 2.7.11.1; PINK1_MOUSE; PTEN induced kinase 1; Parkinson disease (autosomal recessive) 6; PTEN induced putative kinase 1; Serine/threonine-protein kinase PINK1, mitochondrial (BRPK) (PTEN-induced putative kinase protein 1)
Dilution	IHC-P=1:50-200,IHC-F=1:50-200,WB=1:500-2000,IF=1:50-200,ELISA=1:5000-10000,IP=1:50-200
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody

is stable for at least two weeks at 2-4 °C.

Protein Information

Name	PINK1
Function	Serine/threonine-protein kinase which acts as a sensor of mitochondrial damage and protects against mitochondrial dysfunction during cellular stress (PubMed:40080546). It phosphorylates mitochondrial proteins to coordinate mitochondrial quality control mechanisms that remove and replace dysfunctional mitochondrial components (PubMed:14607334, PubMed:15087508, PubMed:18443288, PubMed:18957282, PubMed:19229105, PubMed:19966284, PubMed:20404107, PubMed:20547144, PubMed:20798600, PubMed:22396657, PubMed:23620051, PubMed:23754282, PubMed:23933751, PubMed:24660806, PubMed:24751536, PubMed:24784582, PubMed:24896179, PubMed:24898855, PubMed:25527291, PubMed:32484300). In healthy mitochondria, PINK1 is translocated across the mitochondrial outer membrane (MOM) via the translocase of the outer membrane (TOM) complex, and inserted into the mitochondrial inner membrane (MIM) via the translocase of the inner membrane (TIM23) complex where it is cleaved and released into the cytosol (PubMed:40080546). Depending on the severity of mitochondrial damage, activity ranges from preventing apoptosis and stimulating mitochondrial biogenesis to eliminating severely damaged mitochondria via PINK1-PRKN- dependent mitophagy (PubMed:14607334, PubMed:15087508, PubMed:18443288, PubMed:19966284, PubMed:20404107, PubMed:20798600, PubMed:22396657, PubMed:23620051, PubMed:23933751, PubMed:24898855, PubMed:32047033, PubMed:32484300). When cellular stress results in irreversible mitochondrial damage, PINK1 accumulates at the outer mitochondrial membrane (OMM) where it phosphorylates pre-existing polyubiquitin chains at 'Ser-65', recruits PRKN from the cytosol to the OMM and activates PRKN by phosphorylation at 'Ser-65'; activated PRKN then ubiquitinates VDAC1 and other OMM proteins to initiate mitophagy (PubMed:14607334, PubMed:15087508, PubMed:19966284, PubMed:20404107, PubMed:20798600, PubMed:23754282, PubMed:23933751, PubMed:24660806, PubMed:24751536, PubMed:24784582, PubMed:25474007, PubMed:25527291, PubMed:32047033, PubMed:40080546). The PINK1-PRKN pathway also promotes fission of damaged mitochondria through phosphorylation and PRKN- dependent degradation of mitochondrial proteins involved in fission such as MFN2 (PubMed:18443288, PubMed:23620051, PubMed:24898855). This prevents the refusion of unhealthy mitochondria with the mitochondrial network or initiates mitochondrial fragmentation facilitating their later engulfment by autophagosomes (PubMed:18443288, PubMed:23620051). Also promotes mitochondrial fission independently of PRKN and ATG7-mediated mitophagy, via the phosphorylation and activation of DNM1L (PubMed:18443288, 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 (PubMed:22396657). Required for ubiquinone reduction by mitochondrial complex I by mediating phosphorylation of complex I subunit NDUFA10 (By similarity). Phosphorylates LETM1, positively regulating its mitochondrial calcium transport activity (PubMed:29123128).
Cellular Location	Mitochondrion outer membrane Mitochondrion inner membrane

	{ECO:0000250|UniProtKB:Q99MQ3}. Cytoplasm, cytosol Note=Localizes mostly in mitochondrion and the two smaller proteolytic processed fragments localize mainly in cytosol (PubMed:19229105). In healthy mitochondria, PINK1 is translocated across the mitochondrial membranes (PubMed:40080546). Upon mitochondrial membrane depolarization following damage, PINK1 import is stalled, which induces its accumulation in the outer mitochondrial membrane and the activation of its kinase activity (PubMed:18957282, PubMed:40080546)
Tissue Location	Highly expressed in heart, skeletal muscle and testis, and at lower levels in brain, placenta, liver, kidney, pancreas, prostate, ovary and small intestine. Present in the embryonic testis from an early stage of development

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.