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PINK1 (PARK6) Antibody (Center)

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP6406B

Product Information

Application	WB, E
Primary Accession	Q9BXM7
Other Accession	NP_115785
Reactivity	Human, Mouse
Predicted	Rat, Canine, Rabbit
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	62769
Antigen Region	237-266

Additional Information

Gene ID	65018
Other Names	Serine/threonine-protein kinase PINK1, mitochondrial, BRPK, PTEN-induced putative kinase protein 1, PINK1
Target/Specificity	This PINK1 (PARK6) antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 237-266 amino acids from the Central region of human PINK1 (PARK6).
Dilution	WB~~1:1000 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.
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	PINK1 (PARK6) Antibody (Center) is for research use only and not for use in diagnostic or therapeutic procedures.

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

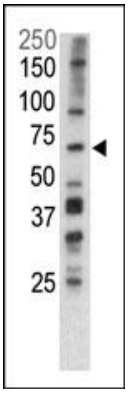
Background

Parkinson is the second most common neurodegenerative disease after Alzheimers. About 1 percent of people over the age of 65 and 3 percent of people over the age of 75 are affected by the disease. The mutation is the most common cause of Parkinson disease identified to date. Defects in PINK1 are the cause of autosomal recessive early-onset Parkinson's disease 6 (PARK6). Six novel pathogenic PINK1 mutations suggest that PINK1 may be the second most common causative gene next to parkin in parkinsonism with the recessive mode of inheritance. Strong evidence indicates that, although important in mendelian forms of Parkinson's disease (PD), PINK1 does not influence the cause of sporadic nonmendelian forms of PD.

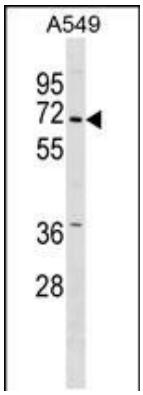
References

Rogaeva,E.,et al. Arch. Neurol. 61 (12), 1898-1904 (2004) Hatano,Y., et al. Ann. Neurol. 56 (3), 424-427 (2004) Healy,D.G., et al. Ann. Neurol. 56 (3), 329-335 (2004) Valente,E.M., et al. Science 304 (5674), 1158-1160 (2004) Nakajima,A., et al. Cancer Lett. 201 (2), 195-201 (2003) Unoki,M. and Nakamura,Y. Oncogene 20 (33), 4457-4465 (2001)

Images



The anti-PINK1 C-term Pab (Cat. #AP6406b) is used in Western blot to detect PINK1 in mouse heart tissue lysate.



Park6 (PINK1) Antibody (Center) (Cat. #AP6406b) western blot analysis in A549 cell line lysates (35ug/lane).This demonstrates the Park6 (PINK1) antibody detected the Park6 (PINK1) protein (arrow).

Citations

- PINK1/Parkin-mediated mitophagy is dependent on VDAC1 and p62/SQSTM1.
- MKK3 regulates mitochondrial biogenesis and mitophagy in sepsis-induced lung injury.
- Defective mitochondria-lysosomal axis enhances the release of extracellular vesicles containing mitochondrial DNA and proteins in Huntington's disease

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