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PINK1 (PARK6) Antibody (N-term T133)

Purified Rabbit Polyclonal Antibody (Pab)

Catalog # AP6406A

Specification

PINK1 (PARK6) Antibody (N-term T133) - Product Information

Application	**WB, IHC-P,E**
Primary Accession	Q9BXM7
Other Accession	NP_115785
Reactivity	**Human**
Host	**Rabbit**
Clonality	**Polyclonal**
Isotype	**Rabbit IgG**
Calculated MW	**62769**
Antigen Region	**118-147**

PINK1 (PARK6) Antibody (N-term T133) - 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 118-147 amino acids from the N-terminal region of human PINK1 (PARK6).

Dilution
WB~~1:1000
IHC-P~~1:50~100

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 (N-term T133) is for research use only and not for use in diagnostic or therapeutic procedures.

PINK1 (PARK6) Antibody (N-term T133) - 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:14607334, PubMed:18957282, PubMed:18443288, PubMed:15087508, PubMed:19229105, PubMed:19966284, PubMed:20404107, PubMed:22396657, PubMed:20798600, PubMed:23620051, PubMed:23754282, PubMed:23933751, PubMed:24660806, PubMed:24898855, PubMed:24751536, PubMed:24784582, PubMed:24896179, PubMed:25527291, PubMed:32484300, PubMed:20547144). 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 (PubMed:18443288, PubMed:23620051, PubMed:24898855, PubMed:20798600, PubMed:20404107, PubMed:19966284, PubMed:32484300, PubMed:22396657, PubMed:32047033, PubMed:15087508). Mediates the translocation and activation of PRKN at the outer membrane (OMM) of dysfunctional/depolarized mitochondria (PubMed:19966284, PubMed:20404107, PubMed:20798600, PubMed:23754282, PubMed:24660806, PubMed:24751536, PubMed:24784582, PubMed:25474007, PubMed:25527291). 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:19966284, PubMed:20404107, PubMed:20798600, PubMed:23754282, PubMed:24660806, PubMed:24751536, PubMed:24784582, PubMed:25474007, PubMed:25527291). 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 (PubMed:32047033). When cellular stress results in irreversible mitochondrial damage, functions with PRKN to promote clearance of damaged mitochondria via selective autophagy (mitophagy) (PubMed:14607334, PubMed:20798600, PubMed:20404107, PubMed:19966284, PubMed:23933751, PubMed:15087508). 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 (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; Single-pass membrane protein. Mitochondrion inner membrane {ECO:0000250|UniProtKB:Q99MQ3}; Single-pass membrane protein. Cytoplasm, cytosol. Note=Localizes mostly in mitochondrion and the two smaller proteolytic processed fragments localize mainly in cytosol (PubMed:19229105). 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 (PubMed:18957282)

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

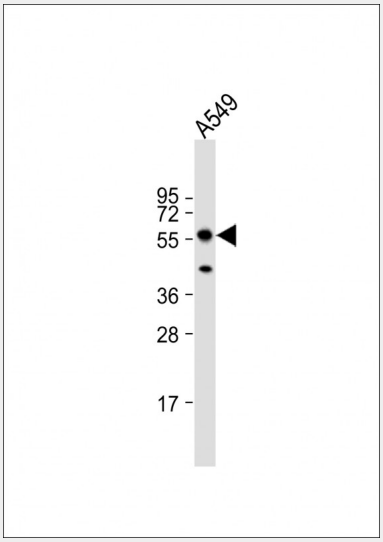
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PINK1 (PARK6) Antibody (N-term T133) - Protocols

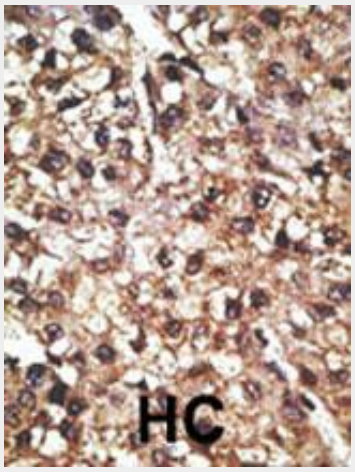
Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

PINK1 (PARK6) Antibody (N-term T133) - Images



Anti-Park6 (PINK1) Antibody (N-term) at 1:1000 dilution + A549 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 63 kDa Blocking/Dilution buffer: 5% NFDM/TBST.



Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was peroxidase-conjugated to the secondary antibody, followed by AEC staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.

PINK1 (PARK6) Antibody (N-term T133) - Background

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.

PINK1 (PARK6) Antibody (N-term T133) - 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)

PINK1 (PARK6) Antibody (N-term T133) - Citations

- The post-therapeutic effect of rapamycin in mild traumatic brain injured rats ensuing in the upregulation of autophagy and mitophagy.
- High expression of PINK1 promotes proliferation and chemoresistance of NSCLC.