

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

Purified Rabbit Polyclonal Antibody (Pab)

Catalog # AP6406A

## Product Information

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<b>Application</b>	IHC-P, WB, E
<b>Primary Accession</b>	<a href="#">Q9BXM7</a>
<b>Other Accession</b>	<a href="#">NP_115785</a>
<b>Reactivity</b>	Human
<b>Host</b>	Rabbit
<b>Clonality</b>	Polyclonal
<b>Isotype</b>	Rabbit IgG
<b>Calculated MW</b>	62769
<b>Antigen Region</b>	118-147

## Additional Information

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<b>Gene ID</b>	65018
<b>Other Names</b>	Serine/threonine-protein kinase PINK1, mitochondrial, BRPK, PTEN-induced putative kinase protein 1, PINK1
<b>Target/Specificity</b>	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).
<b>Dilution</b>	IHC-P~~1:100~500 WB~~1:1000 E~~Use at an assay dependent concentration.
<b>Format</b>	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.
<b>Storage</b>	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.
<b>Precautions</b>	PINK1 (PARK6) Antibody (N-term T133) is for research use only and not for use in diagnostic or therapeutic procedures.

## Protein Information

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<b>Name</b>	PINK1
<b>Function</b>	Serine/threonine-protein kinase which acts as a sensor of mitochondrial damage and protects against mitochondrial dysfunction during cellular stress. 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](#)). 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](#)). 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 DNMT1 (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 NDUF10 (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](#)). Upon mitochondrial membrane depolarization following damage, PINK1 import into the mitochondria 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

## 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.

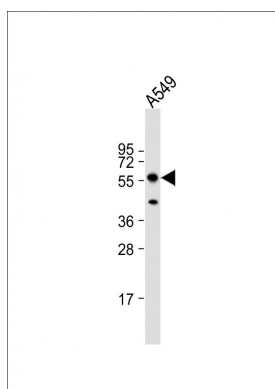
## References

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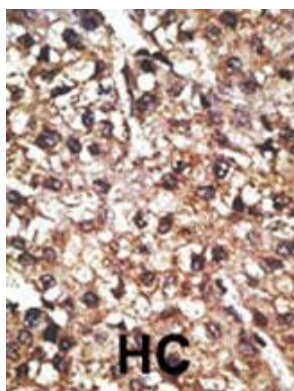
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Hatano,Y., et al. Ann. Neurol. 56 (3), 424-427 (2004)  
Healy,D.G., et al. Ann. Neurol. 56 (3), 329-335 (2004)  
Valente,E.M., et al. Science 304 (5674), 1158-1160 (2004)  
Nakajima,A., et al. Cancer Lett. 201 (2), 195-201 (2003)  
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## Images

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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.

## Citations

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- [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.](#)

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