

**PINK1 (PARK6) Antibody (Center)**  
**Purified Rabbit Polyclonal Antibody (Pab)**  
**Catalog # AP6406B**

**Specification**

**PINK1 (PARK6) Antibody (Center) - Product Information**

|                   |                           |
|-------------------|---------------------------|
| Application       | WB,E                      |
| Primary Accession | <a href="#">Q9BXM7</a>    |
| Other Accession   | <a href="#">NP_115785</a> |
| Reactivity        | Human                     |
| Host              | Rabbit                    |
| Clonality         | Polyclonal                |
| Isotype           | Rabbit Ig                 |
| Antigen Region    | 237-266                   |

**PINK1 (PARK6) Antibody (Center) - 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

**Format**

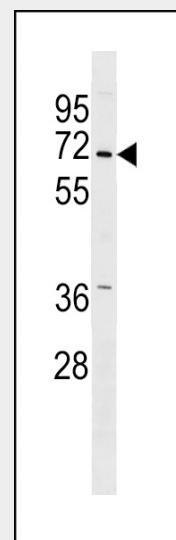
Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.

**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



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).

**PINK1 (PARK6) Antibody (Center) - 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.

**PINK1 (PARK6) Antibody (Center) -**

research use only and not for use in diagnostic or therapeutic procedures.

#### **PINK1 (PARK6) Antibody (Center) - 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 DNML1, to coordinate mitochondrial quality control mechanisms that remove and replace dysfunctional mitochondrial components (PubMed:<a href="http://www.uniprot.org/citations/14607334" target="\_blank">14607334</a>, PubMed:<a href="http://www.uniprot.org/citations/18957282" target="\_blank">18957282</a>, PubMed:<a href="http://www.uniprot.org/citations/18443288" target="\_blank">18443288</a>, PubMed:<a href="http://www.uniprot.org/citations/15087508" target="\_blank">15087508</a>, PubMed:<a href="http://www.uniprot.org/citations/19229105" target="\_blank">19229105</a>, PubMed:<a href="http://www.uniprot.org/citations/19966284" target="\_blank">19966284</a>, PubMed:<a href="http://www.uniprot.org/citations/20404107" target="\_blank">20404107</a>, PubMed:<a href="http://www.uniprot.org/citations/22396657" target="\_blank">22396657</a>, PubMed:<a href="http://www.uniprot.org/citations/20798600" target="\_blank">20798600</a>, PubMed:<a href="http://www.uniprot.org/citations/23620051" target="\_blank">23620051</a>, PubMed:<a href="http://www.uniprot.org/citations/23754282" target="\_blank">23754282</a>, PubMed:<a href="http://www.uniprot.org/citations/23933751" target="\_blank">23933751</a>, PubMed:<a href="http://www.uniprot.org/citations/24660806" target="\_blank">24660806</a>, PubMed:<a href="http://www.uniprot.org/ci

##### **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)

tations/24898855"  
target="\_blank">24898855</a>,  
PubMed:<a href="http://www.uniprot.org/ci  
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PubMed:<a href="http://www.uniprot.org/ci  
tations/32484300"  
target="\_blank">32484300</a>,  
PubMed:<a href="http://www.uniprot.org/ci  
tations/20547144"  
target="\_blank">20547144</a>).  
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:<a href="http://www.u  
niprot.org/citations/18443288"  
target="\_blank">18443288</a>,  
PubMed:<a href="http://www.uniprot.org/ci  
tations/23620051"  
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target="\_blank">32047033</a>,  
PubMed:<a href="http://www.uniprot.org/ci  
tations/15087508"  
target="\_blank">15087508</a>). Mediates  
the translocation and activation of PRKN at

the outer membrane (OMM) of dysfunctional/depolarized mitochondria (PubMed:<a href="http://www.uniprot.org/citations/19966284" target="\_blank">19966284</a>, PubMed:<a href="http://www.uniprot.org/citations/20404107" target="\_blank">20404107</a>, PubMed:<a href="http://www.uniprot.org/citations/20798600" target="\_blank">20798600</a>, PubMed:<a href="http://www.uniprot.org/citations/23754282" target="\_blank">23754282</a>, PubMed:<a href="http://www.uniprot.org/citations/24660806" target="\_blank">24660806</a>, PubMed:<a href="http://www.uniprot.org/citations/24751536" target="\_blank">24751536</a>, PubMed:<a href="http://www.uniprot.org/citations/24784582" target="\_blank">24784582</a>, PubMed:<a href="http://www.uniprot.org/citations/25474007" target="\_blank">25474007</a>, PubMed:<a href="http://www.uniprot.org/citations/25527291" target="\_blank">25527291</a>). 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:<a href="http://www.uniprot.org/citations/19966284" target="\_blank">19966284</a>, PubMed:<a href="http://www.uniprot.org/citations/20404107" target="\_blank">20404107</a>, PubMed:<a href="http://www.uniprot.org/citations/20798600" target="\_blank">20798600</a>, PubMed:<a href="http://www.uniprot.org/citations/23754282" target="\_blank">23754282</a>, PubMed:<a href="http://www.uniprot.org/citations/24660806" target="\_blank">24660806</a>, PubMed:<a href="http://www.uniprot.org/citations/24751536" target="\_blank">24751536</a>, PubMed:<a href="http://www.uniprot.org/citations/24784582" target="\_blank">24784582</a>, PubMed:<a href="http://www.uniprot.org/citations/25474007" target="\_blank">25474007</a>, PubMed:<a href="http://www.uniprot.org/citations/25527291" target="\_blank">25527291</a>).

PubMed:<a href="http://www.uniprot.org/citations/25474007" target="\_blank">25474007</a>, PubMed:<a href="http://www.uniprot.org/citations/25527291" target="\_blank">25527291</a>). 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:<a href="http://www.uniprot.org/citations/32047033" target="\_blank">32047033</a>). When cellular stress results in irreversible mitochondrial damage, functions with PRKN to promote clearance of damaged mitochondria via selective autophagy (mitophagy) (PubMed:<a href="http://www.uniprot.org/citations/14607334" target="\_blank">14607334</a>, PubMed:<a href="http://www.uniprot.org/citations/20798600" target="\_blank">20798600</a>, PubMed:<a href="http://www.uniprot.org/citations/20404107" target="\_blank">20404107</a>, PubMed:<a href="http://www.uniprot.org/citations/19966284" target="\_blank">19966284</a>, PubMed:<a href="http://www.uniprot.org/citations/23933751" target="\_blank">23933751</a>, PubMed:<a href="http://www.uniprot.org/citations/15087508" target="\_blank">15087508</a>). 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:<a href="http://www.uniprot.org/citations/18443288" target="\_blank">18443288</a>, PubMed:<a href="http://www.uniprot.org/citations/23620051" target="\_blank">23620051</a>, PubMed:<a href="http://www.uniprot.org/citations/24898855" target="\_blank">24898855</a>). This prevents the refusion of unhealthy mitochondria with the mitochondrial network or initiates mitochondrial

fragmentation facilitating their later engulfment by autophagosomes (PubMed:<a href="http://www.uniprot.org/citations/18443288" target="\_blank">18443288</a>, PubMed:<a href="http://www.uniprot.org/citations/23620051" target="\_blank">23620051</a>). Also promotes mitochondrial fission independently of PRKN and ATG7-mediated mitophagy, via the phosphorylation and activation of DNM1L (PubMed:<a href="http://www.uniprot.org/citations/18443288" target="\_blank">18443288</a>, PubMed:<a href="http://www.uniprot.org/citations/32484300" target="\_blank">32484300</a>). 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:<a href="http://www.uniprot.org/citations/22396657" target="\_blank">22396657</a>). Required for ubiquinone reduction by mitochondrial complex I by mediating phosphorylation of complex I subunit NDUFA10 (By similarity).

#### **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

**PINK1 (PARK6) Antibody (Center) -  
Protocols**

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

**PINK1 (PARK6) Antibody (Center) - Citations**

- [MKK3 regulates mitochondrial biogenesis and mitophagy in sepsis-induced lung injury.](#)
- [PINK1/Parkin-mediated mitophagy is dependent on VDAC1 and p62/SQSTM1.](#)