

**PINK1 Antibody (N-Terminus)**  
**Rabbit Polyclonal Antibody**  
**Catalog # ALS16436**

### Specification

#### PINK1 Antibody (N-Terminus) - Product Information

Application	IHC, WB
Primary Accession	<a href="#">Q9BXM7</a>
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Calculated MW	63kDa KDa

#### PINK1 Antibody (N-Terminus) - Additional Information

##### Gene ID 65018

##### Other Names

Serine/threonine-protein kinase PINK1, mitochondrial, 2.7.11.1, BRPK, PTEN-induced putative kinase protein 1, PINK1

##### Target/Specificity

PINK1 antibody is human, mouse and rat reactive. At least two isoforms are known to exist; this antibody will only detect the longer isoform. PINK1 antibody will detect the cleaved and uncleaved form of PINK1.

##### Reconstitution & Storage

Long term: -20°C; Short term: +4°C. Avoid repeat freeze-thaw cycles.

##### Precautions

PINK1 Antibody (N-Terminus) is for research use only and not for use in diagnostic or therapeutic procedures.

#### PINK1 Antibody (N-Terminus) - 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:<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>)

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/citations/24898855" target="\_blank">>24898855</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/24896179" target="\_blank">>24896179</a>, PubMed:<a href="http://www.uniprot.org/citations/25527291" target="\_blank">>25527291</a>, PubMed:<a href="http://www.uniprot.org/citations/32484300" target="\_blank">>32484300</a>, PubMed:<a href="http://www.uniprot.org/citations/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.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>, 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/32484300" target="\_blank">>32484300</a>, PubMed:<a href="http://www.uniprot.org/citations/22396657" target="\_blank">>22396657</a>, PubMed:<a href="http://www.uniprot.org/citations/32047033" target="\_blank">>32047033</a>, PubMed:<a href="http://www.uniprot.org/citations/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>). 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). Phosphorylates LETM1, positively regulating its mitochondrial calcium transport activity (PubMed:<a href="http://www.uniprot.org/citations/29123128" target="\_blank">>29123128</a>).

### 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

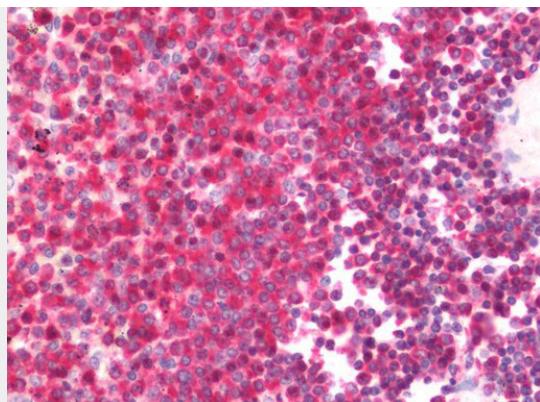
Highly expressed in heart, skeletal muscle and testis, and at lower levels in brain, placenta, liver, kidney, pancreas, prostate, ovary and small intestine. Present in the embryonic testis from an early stage of development

### PINK1 Antibody (N-Terminus) - Protocols

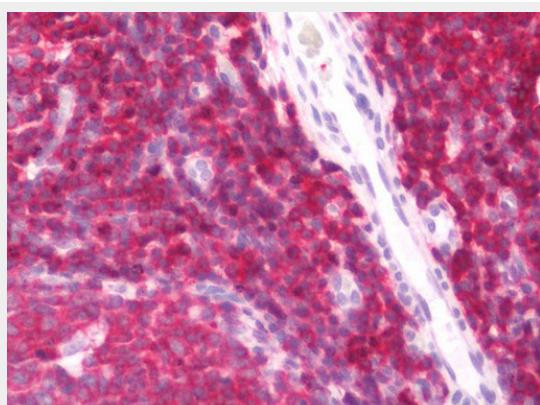
Provided below are standard protocols that you may find useful for product applications.

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

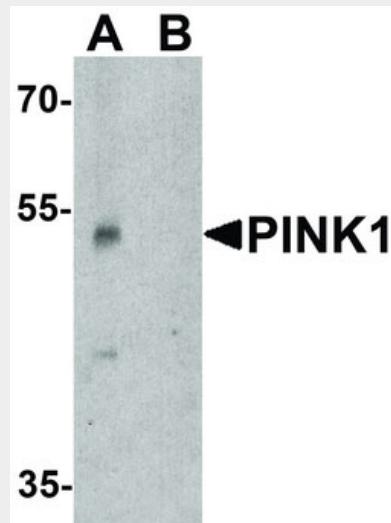
### PINK1 Antibody (N-Terminus) - Images



Human Spleen: Formalin-Fixed, Paraffin-Embedded (FFPE)



Human Tonsil: Formalin-Fixed, Paraffin-Embedded (FFPE)



Western blot analysis of PINK1 in A431 cell lysate with PINK1 antibody at 1 ug/ml in (A) the...

#### PINK1 Antibody (N-Terminus) - Background

Protects against mitochondrial dysfunction during cellular stress by phosphorylating mitochondrial proteins. Involved in the clearance of damaged mitochondria via selective autophagy (mitophagy) by mediating activation and translocation of PARK2. Targets PARK2 to dysfunctional depolarized mitochondria through the phosphorylation of MFN2. Activates PARK2 in 2 steps: (1) by mediating phosphorylation at 'Ser-65' of PARK2 and (2) mediating phosphorylation of ubiquitin, converting PARK2 to its fully-active form (PubMed:24660806, PubMed:24751536, PubMed:24784582).

### PINK1 Antibody (N-Terminus) - References

- Unoki M.,et al.Oncogene 20:4457-4465(2001).  
Nakajima A.,et al.Cancer Lett. 201:195-201(2003).  
Ota T.,et al.Nat. Genet. 36:40-45(2004).  
Gregory S.G.,et al.Nature 441:315-321(2006).  
Zhou C.,et al.Proc. Natl. Acad. Sci. U.S.A. 105:12022-12027(2008).