

PINK1 Antibody
Catalog # ASC11814**Specification****PINK1 Antibody - Product Information**

Application	IF, IHC
Primary Accession	Q9BXM7
Other Accession	NP_115785 , 65018
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	Predicted: 55 kDa

Application Notes	Observed: 53 kDa KDa PINK1 antibody can be used for detection of PINK1 by Western blot at 1 - 2 µg/ml. Antibody can also be used for immunohistochemistry starting at 5 µg/mL. For immunofluorescence start at 20 µg/mL.
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PINK1 Antibody - Additional InformationGene ID **65018****Target/Specificity**

PINK1 antibody was raised against a 16 amino acid peptide near the amino terminus of human PINK1.

The immunogen is located within amino acids 120 - 170 of PINK1.

Reconstitution & Storage

PINK1 antibody can be stored at 4°C for three months and -20°C, stable for up to one year.

Precautions

PINK1 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

PINK1 Antibody - 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. 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)

target="_blank">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 DNM1L (PubMed:<a

[18443288](http://www.uniprot.org/citations/18443288), PubMed: [32484300](http://www.uniprot.org/citations/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](http://www.uniprot.org/citations/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](http://www.uniprot.org/citations/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

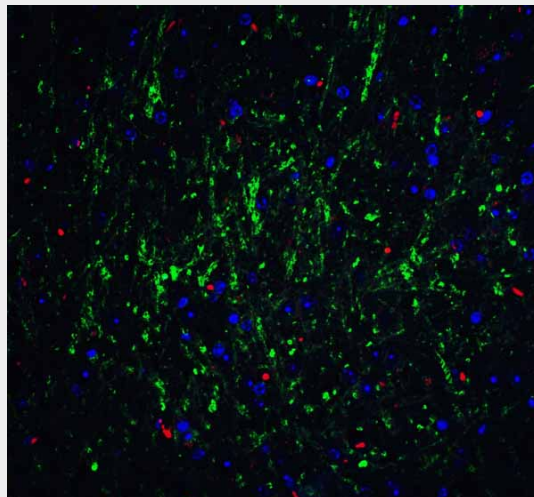
Highly expressed in heart, skeletal muscle and testis, and at lower levels in brain, placenta, liver, kidney, pancreas, prostate, ovary and small intestine. Present in the embryonic testis from an early stage of development

PINK1 Antibody - Protocols

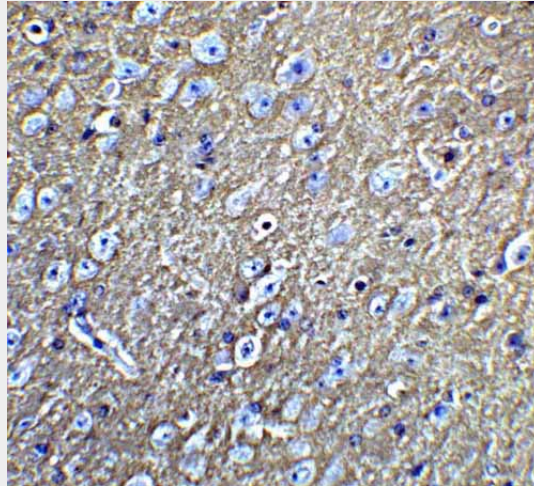
Provided below are standard protocols that you may find useful for product applications.

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

PINK1 Antibody - Images



Immunofluorescence of RPSA in mouse brain tissue with RPSA Antibody at 20 µg/mL.



Immunohistochemistry of LMX1B in mouse brain tissue with LMX1B Antibody at 5 µg/mL.

PINK1 Antibody - Background

The PTEN-induced putative kinase 1 (PINK1) is a serine/threonine protein kinase that localizes to mitochondria and is thought to protect cells from stress-induced mitochondrial dysfunction (reviewed in 1). PINK1 recruits the E3 ubiquitin ligase Parkin to mitochondria to initiate mitophagy, an autophagic process that clears damaged mitochondria within a cell (2). PINK1 is cleaved by the mitochondrial protease PARL (3). Mutations in this gene cause one form of autosomal recessive early-onset Parkinson disease (4).

PINK1 Antibody - References

- Matsuda S, Kitagishi Y, and Kobayashi M. Function and characteristics of PINK1 in mitochondria. *Oxid. Med. Cell. Longev.* 2013;60:1587.
- Corti O, Lesage S, and Brice A. What genetics tells us about the causes and mechanism of Parkinson's disease. *Physiol. Rev.* 2011; 91:1161-218.
- Deas E, Plun-Favreau H, and Gandhi S, et al. PINK1 cleavage at position A103 by the mitochondrial protease PARL. *Hum. Mol. Genet.* 2011; 20:867-79.
- Valente EM, Abou-Sleiman PM, Caputo V, et al. Hereditary early-onset Parkinson's disease caused by mutations in PINK1. *Science* 2004; 304:1158-60.