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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** **Observed: 53 kDa KDa**
Application Notes	**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.**

PINK1 Antibody - Additional Information

Gene 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℃ for three months and -20℃, 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 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:15087508, PubMed:18443288, PubMed:18957282, PubMed:19229105, PubMed:<a href="http://www.uniprot.org/citations/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 and/or dysfunction, activity ranges from preventing apoptosis and stimulating mitochondrial biogenesis to regulating mitochondrial dynamics and eliminating severely damaged mitochondria via mitophagy (PubMed:15087508, PubMed:18443288, PubMed:19966284, PubMed:20404107, PubMed:20798600, PubMed:22396657, PubMed:23620051, PubMed:24898855, PubMed:32047033, PubMed:32484300). 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:<a

href="http://www.uniprot.org/citations/14607334" target="_blank">14607334, PubMed:15087508, PubMed:19966284, PubMed:20404107, PubMed:20798600, PubMed:23933751). 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

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 Cytomety
- Cell Culture

PINK1 Antibody - Images

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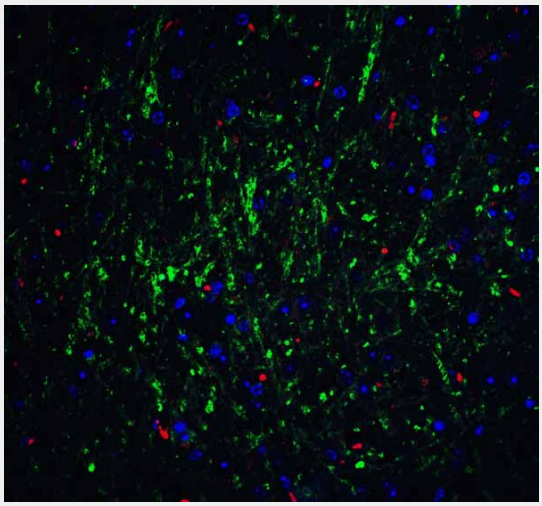
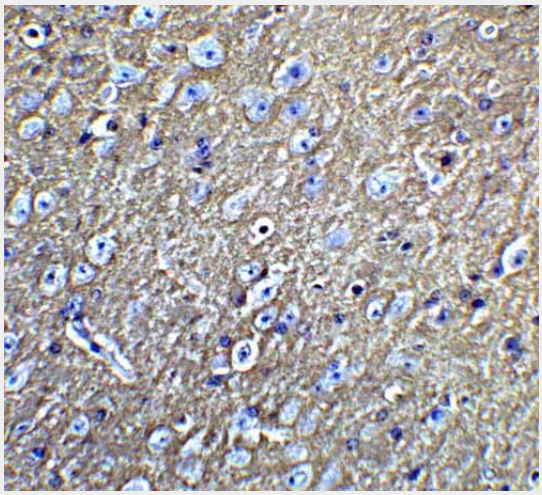


Immunofluorescence of RPSA in mouse brain tissue with RPSA Antibodyat 20 µg/mL.



Immunohistochemistry of LMX1B in mouse brain tissue with LMX1B Antibodyat 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;601587.
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.