

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
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**

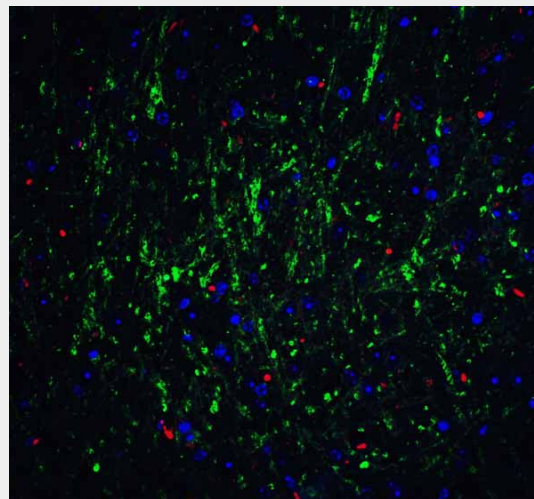
Target/Specificity
PINK1 antibody was raised against a 16 amino acid peptide near the amino terminus of human PINK1. The immunogen is located within amino acids 120 - 170 of PINK1.

Reconstitution & Storage

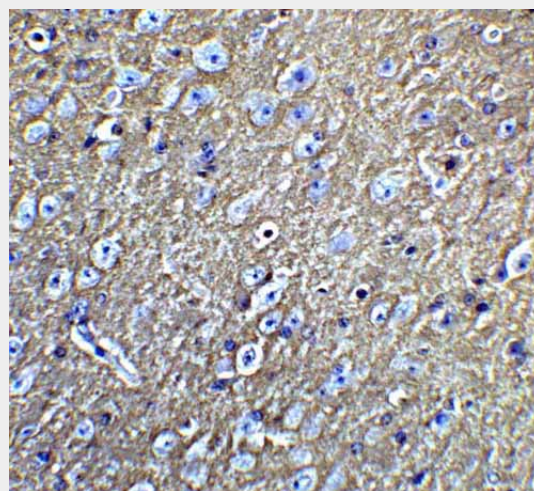
PINK1 antibody can be stored at 4°C for three months and -20°C, stable for up to one year.

Precautions

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



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

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 DNML1, to coordinate mitochondrial quality control mechanisms that remove and replace dysfunctional mitochondrial components (PubMed:14607334, PubMed:18957282, PubMed:18443288, PubMed:15087508, PubMed:19229105, PubMed:19966284, PubMed:20404107, PubMed:22396657, PubMed:20798600, PubMed:23620051, PubMed:23754282, PubMed:23933751, PubMed:24660806, PubMed:24898855, PubMed:24751536)

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.

target="_blank">24751536,
PubMed:<a href="http://www.uniprot.org/citations/24784582"
target="_blank">24784582,
PubMed:<a href="http://www.uniprot.org/citations/24896179"
target="_blank">24896179,
PubMed:<a href="http://www.uniprot.org/citations/25527291"
target="_blank">25527291,
PubMed:<a href="http://www.uniprot.org/citations/32484300"
target="_blank">32484300,
PubMed:<a href="http://www.uniprot.org/citations/20547144"
target="_blank">20547144).
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,
PubMed:<a href="http://www.uniprot.org/citations/23620051"
target="_blank">23620051,
PubMed:<a href="http://www.uniprot.org/citations/24898855"
target="_blank">24898855,
PubMed:<a href="http://www.uniprot.org/citations/20798600"
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PubMed:<a href="http://www.uniprot.org/citations/20404107"
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PubMed:<a href="http://www.uniprot.org/citations/19966284"
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PubMed:<a href="http://www.uniprot.org/citations/32484300"
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PubMed:<a href="http://www.uniprot.org/citations/22396657"
target="_blank">22396657,
PubMed:<a href="http://www.uniprot.org/citations/32047033"
target="_blank">32047033,
PubMed:<a href="http://www.uniprot.org/citations/15087508"
target="_blank">15087508). 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,
PubMed:<a href="http://www.uniprot.org/citations/20404107"
target="_blank">20404107,
PubMed:<a href="http://www.uniprot.org/citations/20798600"
target="_blank">20798600,
PubMed:<a href="http://www.uniprot.org/citations/23754282"
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PubMed:<a href="http://www.uniprot.org/citations/24660806"
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PubMed:<a href="http://www.uniprot.org/citations/24751536"
target="_blank">24751536,
PubMed:<a href="http://www.uniprot.org/citations/24784582"
target="_blank">24784582,
PubMed:<a href="http://www.uniprot.org/citations/25474007"
target="_blank">25474007,
PubMed:<a href="http://www.uniprot.org/citations/25527291"
target="_blank">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:<a href="http://www.uniprot.org/citations/19966284"
target="_blank">19966284,
PubMed:<a href="http://www.uniprot.org/citations/20404107"
target="_blank">20404107,
PubMed:<a href="http://www.uniprot.org/citations/20798600"
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PubMed:<a href="http://www.uniprot.org/citations/23754282"
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PubMed:<a href="http://www.uniprot.org/citations/24784582"
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PubMed:<a href="http://www.uniprot.org/citations/25474007"
target="_blank">25474007,
PubMed:<a href="http://www.uniprot.org/citations/25527291"

tations/25527291" target="_blank">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:14607334, PubMed:20798600, PubMed:20404107, PubMed:19966284, PubMed:23933751, PubMed:15087508). 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).

target="_blank">18443288,
PubMed:<a href="http://www.uniprot.org/citations/23620051"
target="_blank">23620051). 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,
PubMed:<a href="http://www.uniprot.org/citations/32484300"
target="_blank">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:<a href="http://www.uniprot.org/citations/22396657"
target="_blank">22396657). 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 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](#)