

**Anti-PINK1 (pS228) Antibody**  
Rabbit polyclonal antibody to PINK1 (pS228)  
Catalog # AP61086

### Specification

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#### Anti-PINK1 (pS228) Antibody - Product Information

Application	WB, E
Primary Accession	<a href="#">O9BXM7</a>
Other Accession	<a href="#">O99MQ3</a>
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Calculated MW	62769

#### Anti-PINK1 (pS228) Antibody - Additional Information

Gene ID 65018

#### Other Names

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

#### Target/Specificity

Recognizes endogenous levels of PINK1 (pS228) protein.

#### Dilution

WB~~WB (1/500 - 1/1000)

E~~WB (1/500 - 1/1000)

#### Format

Liquid in 0.42% Potassium phosphate, 0.87% Sodium chloride, pH 7.3, 30% glycerol, and 0.09% (W/V) sodium azide.

#### Storage

Store at -20 °C. Stable for 12 months from date of receipt

#### Anti-PINK1 (pS228) 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 DNMI1L, to coordinate mitochondrial quality control mechanisms that remove and replace dysfunctional mitochondrial components (PubMed: [14607334](http://www.uniprot.org/citations/14607334) target="\_blank">14607334</a>, PubMed: [15087508](http://www.uniprot.org/citations/15087508) target="\_blank">15087508</a>, PubMed: [18443288](http://www.uniprot.org/citations/18443288) target="\_blank">18443288</a>, PubMed: [18957282](http://www.uniprot.org/citations/18957282) target="\_blank">18957282</a>)

target="\_blank">18957282</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/20547144" target="\_blank">20547144</a>, PubMed:<a href="http://www.uniprot.org/citations/20798600" target="\_blank">20798600</a>, PubMed:<a href="http://www.uniprot.org/citations/22396657" target="\_blank">22396657</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/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/24898855" target="\_blank">24898855</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>). 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/15087508" target="\_blank">15087508</a>, PubMed:<a href="http://www.uniprot.org/citations/18443288" target="\_blank">18443288</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/20798600" target="\_blank">20798600</a>, PubMed:<a href="http://www.uniprot.org/citations/22396657" target="\_blank">22396657</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/32047033" target="\_blank">32047033</a>, PubMed:<a href="http://www.uniprot.org/citations/32484300" target="\_blank">32484300</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/15087508" target="\_blank">15087508</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/20798600" target="\_blank">20798600</a>, PubMed:<a href="http://www.uniprot.org/citations/23933751" target="\_blank">23933751</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

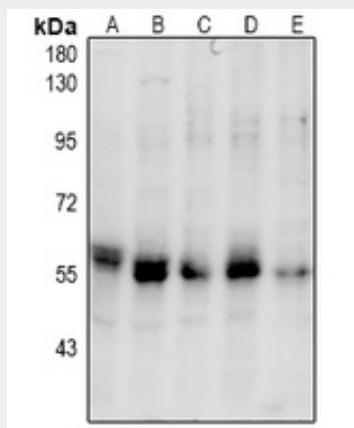
Highly expressed in heart, skeletal muscle and testis, and at lower levels in brain, placenta, liver, kidney, pancreas, prostate, ovary and small intestine. Present in the embryonic testis from an early stage of development

#### Anti-PINK1 (pS228) Antibody - Protocols

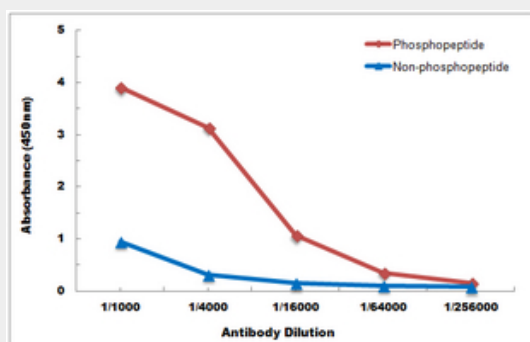
Provided below are standard protocols that you may find useful for product applications.

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

## Anti-PINK1 (pS228) Antibody - Images



Western blot analysis of PINK1 (pS228) expression in mouse testis (A), rat testis (B), HEK293T (C), MCF7 (D), U87MG (E) whole cell lysates.



Direct ELISA antibody dose-response curve using Anti-PINK1 (pS228) Antibody. Antigen (phosphopeptide and non-phosphopeptide) concentration is 5 ug/ml. Goat Anti-Rabbit IgG (H&L) - HRP was used as the secondary antibody, and signal was developed by TMB substrate.

## Anti-PINK1 (pS228) Antibody - Background

KLH-conjugated synthetic peptide encompassing a sequence within the center region of human PINK1. The exact sequence is proprietary.