# PINK1 Rabbit mAb [Pow0]

Cat NO. :A58164

#### Information:

Applications	Reactivity:	UniProt ID:	MW(kDa)	Host	Isotype	Size
WB,IHC	Human,Mouse,R	Q9BXM7	63kd	Rabbit	lgG	50ul,100ul
	at					

#### Applications detail:

Application	Dilution		
WB	1:1000-2000		
нс	1:100		
The optimal dilutions should be determined by the end user			

## Conjugate:

UnConjugate

Form:

Liquid

sensitivity:

Endogenous

#### **Purification**:

Protein A affinity purified.

#### Specificity:

Antibody is produced by immunizing animals with a synthetic peptide at the sequence of Human PINK1

## Storage buffer and conditions:

Antibody store in 10 mM PBS, 0.5mg/ml BSA, 50% glycerol (buffer) .

Shipped at 4°C. Store at-20°C or -80°C.

Products are valid for one natural year of receipt. Avoid repeated freeze / thaw cycles.

#### **Tissue specificity:**

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

#### Subcellular location:

Mitochondrion outer membrane, Single-pass membrane protein. Mitochondrion inner membrane, Single-pass membrane protein. Cytoplasm, cytosol.

**Function**:

Introduction: WB: Western Blot IP: Immunoprecipitation IHC: Immunohistochemistry ChIP: Chromatin Immunoprecipitation ICC/IF: Immunocytochemistry/ Immunofluorescence F: Flow Cytometry

Cross Reactivity: H: human M: mouse R: rat Hm: hamster Mk: monkey Vir: virus MI: mink C: chicken Dm D. melanogaster X: Xenopus Z: zebrafish B: bovine Dg: dog Pg: pig Hr: horse

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# Nature Biosciences

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: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, PubMed:24784582, PubMed:24896179, PubMed:25527291, PubMed:32484300, PubMed: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:18443288, PubMed:23620051, PubMed:24898855, PubMed:20798600, PubMed:20404107, PubMed:19966284, PubMed:32484300, PubMed:22396657, PubMed:32047033, PubMed:15087508). 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: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, PubMed:23620051). Also promotes mitochondrial fission independently of PRKN and ATG7-mediated mitophagy, via the phosphorylation and activation of DNM1L (PubMed:18443288, PubMed:32484300).

## Validation Data:

### PINK1 Rabbit mAb [Pow0] Images



Western blot (SDS PAGE) analysis of extracts from HeLa cells treated with CCCP (10  $\mu$  M 24h) lysates.Using NCOA4 Rabbit mAb [P4P1] at dilution of 1:1000 incubated at 4°C over night.

View more information on http://naturebios.com

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 1% w/v Milk, 1X TBST at 4°C overnight.

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