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PINK1 Antibody

Purified Mouse Monoclonal Antibody (Mab)
Catalog # AW5456

Product Information

Application	WB, IHC-P, IF
Primary Accession	Q9BXM7
Reactivity	Human, Mouse
Host	Mouse
Clonality	Monoclonal
Calculated MW	62769
Isotype	IgG1
Antigen Source	HUMAN

Additional Information

Gene ID	65018
Antigen Region	Unknown
Other Names	Serine/threonine-protein kinase PINK1, mitochondrial, BRPK, PTEN-induced putative kinase protein 1, PINK1
Dilution	WB~~1:1000 IHC-P~~1:100~500 IF~~1:25
Target/Specificity	Recombinant PINK1 protein was used to produced this monoclonal antibody.
Format	Purified monoclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein G column, followed by dialysis against PBS.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	PINK1 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

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 (PubMed:40080546). 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, 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). In healthy mitochondria, PINK1 is translocated across the mitochondrial outer membrane (MOM) via the translocase of the outer membrane (TOM) complex, and inserted into the mitochondrial inner membrane (MIM) via the translocase of the inner membrane (TIM23) complex where it is cleaved and released into the cytosol (PubMed:40080546). 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, PubMed:40080546). 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: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 Mitochondrion inner membrane {ECO:0000250|UniProtKB:Q99MQ3}. Cytoplasm, cytosol Note=Localizes mostly in mitochondrion and the two smaller proteolytic processed fragments localize mainly in cytosol (PubMed:19229105). In healthy mitochondria, PINK1 is translocated across the mitochondrial membranes (PubMed:40080546). Upon mitochondrial membrane depolarization following damage, PINK1 import is stalled, which induces its accumulation in the outer mitochondrial membrane and the activation of its kinase activity (PubMed:18957282, PubMed:40080546)

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

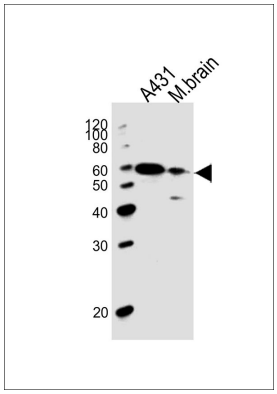
Background

This gene encodes a serine/threonine protein kinase that localizes to mitochondria. It is thought to protect cells from stress-induced mitochondrial dysfunction. Mutations in this gene cause one form of autosomal recessive early-onset Parkinson disease.

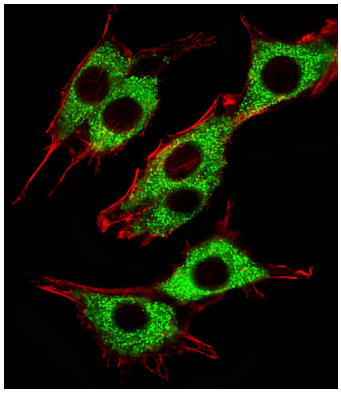
References

Oxidative stress alters the regulatory control of p66Shc and Akt in PINK1 deficient cells. Maj MC, et al. Biochem Biophys Res Commun, 2010 Aug 27. PMID 20637729.
Assessing the prevalence of PINK1 genetic variants in South African patients diagnosed with early- and late-onset Parkinson's disease. Keyser RJ, et al. Biochem Biophys Res Commun, 2010 Jul 16. PMID 20558144.
Progression of subtle motor signs in PINK1 mutation carriers with mild dopaminergic deficit. Eggers C, et al. Neurology, 2010 Jun 1. PMID 20513816.
Structural imaging in the presymptomatic stage of genetically determined parkinsonism. Reetz K, et al. Neurobiol Dis, 2010 Sep. PMID 20483373.
Clinical and demographic characteristics of PINK1 mutation carriers--a meta-analysis. Kasten M, et al. Mov Disord, 2010 May 15. PMID 20461815.

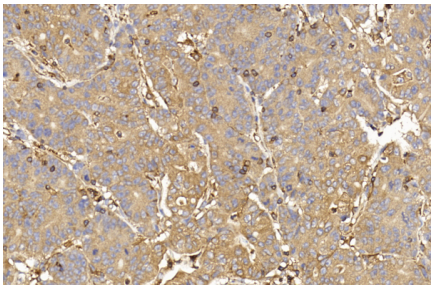
Images



All lanes : Anti-Pink1 Antibody at 1:1000 dilution Lane 1: A431 whole cell lysates Lane 2: mouse brain lysates Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Mouse IgG, (H+L),Peroxidase conjugated at 1/10000 dilution Predicted band size : 63 kDa Blocking/Dilution buffer: 5% NFDM/TBST.



Fluorescent image of PC12 cells stained with Pink1(115-213) Antibody (Cat#AW5456). AW5456 was diluted at 1:25 dilution. An Alexa Fluor 488-conjugated goat anti-mouse lgG at 1:400 dilution was used as the secondary antibody (green). Cytoplasmic actin was counterstained with Alexa Fluor® 555 conjugated with Phalloidin (red).



Immunohistochemical analysis of paraffin-embedded Human colon cancer section using Pink1(Cat#AW5456). AW5456 was diluted at 1:200 dilution. A undiluted biotinylated goat polyvalent antibody was used as the secondary, followed by DAB staining.

Citations

- Deficient tRNA posttranscription modification dysregulated the mitochondrial quality controls and apoptosis
- Deafness-associated tRNA mutation impaired mitochondrial and cellular integrity
- Sustained adenosine exposure causes endothelial mitochondrial dysfunction via equilibrative nucleoside transporters
- OPA1 haploinsufficiency due to a novel splicing variant resulting in mitochondrial dysfunction without mitochondrial DNA depletion
- Global Landscape and Dynamics of Parkin and USP30-Dependent Ubiquitylomes in iNeurons during Mitophagic Signaling
- RAB7A phosphorylation by TBK1 promotes mitophagy via the PINK-PARKIN pathway.
- Mitophagy receptor FUNDC1 is regulated by PGC-1α/NRF1 to fine tune mitochondrial homeostasis
- Leber's hereditary optic neuropathy-associated ND6 14484T C mutation caused pleiotropic effects on the complex I, RNA homeostasis, apoptosis and mitophagy

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