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Park6(PINK1) Antibody(C-term)

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP6406D

Specification

Park6(PINK1) Antibody(C-term) - Product Information

Application	**WB,E**
Primary Accession	Q9BXM7
Reactivity	**Human, Mouse**
Host	**Rabbit**
Clonality	**Polyclonal**
Isotype	**Rabbit IgG**
Antigen Region	**493-526**

Park6(PINK1) Antibody(C-term) - Additional Information

Gene ID 65018

Other Names
Serine/threonine-protein kinase PINK1, mitochondrial, BRPK, PTEN-induced putative kinase protein 1, PINK1

Target/Specificity
This Park6(PINK1) antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 493-526 amino acids from the C-terminal region of human Park6(PINK1).

Dilution
WB~~1:1000
E~~Use at an assay dependent concentration.

Format
Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

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
Park6(PINK1) Antibody(C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

Park6(PINK1) Antibody(C-term) - 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. 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). 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 ubiquinates 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). 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; 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). Upon mitochondrial membrane depolarization following damage, PINK1 import into the mitochondria 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

Park6(PINK1) Antibody(C-term) - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry

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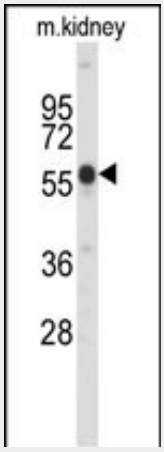
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

Park6(PINK1) Antibody(C-term) - Images



Western blot analysis of Park6 (PINK1) C-term (Cat. #AP6406d) in mouse kidney tissue lysates (35ug/lane). Park6 (arrow) was detected using the purified Pab.

Park6(PINK1) Antibody(C-term) - Background

Parkinson is the second most common neurodegenerative disease after Alzheimers. About 1 percent of people over the age of 65 and 3 percent of people over the age of 75 are affected by the disease. The mutation is the most common cause of Parkinson disease identified to date. Defects in PINK1 are the cause of autosomal recessive early-onset Parkinson's disease 6 (PARK6). Six novel pathogenic PINK1 mutations suggest that PINK1 may be the second most common causative gene next to parkin in parkinsonism with the recessive mode of inheritance. Strong evidence indicates that, although important in mendelian forms of Parkinson's disease (PD), PINK1 does not influence the cause of sporadic nonmendelian forms of PD.

Park6(PINK1) Antibody(C-term) - References

Hatano, Y., et al., Ann. Neurol. 56(3):424-427 (2004).
Healy, D.G., et al., Ann. Neurol. 56(3):329-335 (2004).
Valente, E.M., et al., Science 304(5674):1158-1160 (2004).
Nakajima, A., et al., Cancer Lett. 201(2):195-201 (2003).
Unoki, M., et al., Oncogene 20(33):4457-4465 (2001).

Park6(PINK1) Antibody(C-term) - Citations

- CLEC16A regulates splenocyte and NK cell function in part through MEK signaling.
- PINK1 regulates histone H3 trimethylation and gene expression by interaction with the polycomb protein EED/WAIT1.