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Park6(PINK1) Antibody(C-term) Blocking peptide

Synthetic peptide
Catalog # BP6406d

Specification

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

Primary Accession Q9BXM7

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

Gene ID 65018

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

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP6406d was selected from the PINK1 region of human Park6 (PINK1) C-term. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

Park6(PINK1) Antibody(C-term) Blocking peptide - 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 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:<a href="http://www.uniprot.org/citations/22396657"

target="_blank">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:<a

href="http://www.uniprot.org/citations/20404107" target="_blank">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). 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). 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

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

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

- Blocking Peptides

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

Park6(PINK1) Antibody(C-term) Blocking peptide - 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) Blocking peptide - 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).