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Drosophila Parkin Antibody (C-term) Blocking Peptide

Synthetic peptide
Catalog # BP6414b

Specification

Drosophila Parkin Antibody (C-term) Blocking Peptide - Product Information

Primary Accession Q7KTX7

Drosophila Parkin Antibody (C-term) Blocking Peptide - Additional Information

Gene ID 40336

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP6414b was selected from the C-term region of human Drosophila Parkin. 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.

Drosophila Parkin Antibody (C-term) Blocking Peptide - Protein Information

Name PRKN

Function
E3 ubiquitin-protein ligase which accepts ubiquitin from E2 ubiquitin-conjugating enzymes in the form of a thioester and then directly transfers the ubiquitin to targeted substrates, such as Paris, Marf, Opa1, Miro, pnut, Septin1, Tom20 and porin (PubMed:16002472, PubMed:17456438, PubMed:25474007, PubMed:20194754, PubMed:24192653, PubMed:24901221, PubMed:27906179, PubMed:31714929, PubMed:32138754, PubMed:32047033, PubMed:23770917). Mediates monoubiquitination as well as 'Lys-6', 'Lys-11', 'Lys-48'-linked and 'Lys-63'-linked

polyubiquitination of substrates, depending on the context (PubMed:18957282, PubMed:24901221, PubMed:25474007, PubMed:23650379, PubMed:27906179, PubMed:31714929, PubMed:32047033). Protects against mitochondrial dysfunction during cellular stress, by acting downstream of Pink1, to coordinate mitochondrial quality control mechanisms that remove and replace dysfunctional mitochondrial components (PubMed:12642658, PubMed:15073152, PubMed:16672980, PubMed:16672981, PubMed:17123504, PubMed:18957282, PubMed:18799731, PubMed:18230723, PubMed:18443288, PubMed:20496123, PubMed:20194754, PubMed:23509287, PubMed:24192653, PubMed:24901221, PubMed:25474007, PubMed:27906179, PubMed:29497364, PubMed:32047033). 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:12642658, PubMed:15073152, PubMed:16002472, PubMed:16672980, PubMed:16672981, PubMed:17123504, PubMed:18957282, PubMed:18799731, PubMed:18230723, PubMed:18443288, PubMed:20496123, PubMed:20194754, PubMed:23509287, PubMed:24192653, PubMed:24901221, PubMed:25474007, PubMed:27906179, PubMed:29497364, PubMed:32047033). Appears to be particularly important in maintaining the physiology and function of cells with high energy demands that are undergoing stress or altered metabolic environment, including spermatids, muscle cells and neurons such as the dopaminergic (DA) neurons (PubMed:12642658, PubMed:15073152, PubMed:16002472, PubMed:16672980, PubMed:<a

href="http://www.uniprot.org/citations/17123504" target="_blank">17123504, PubMed:18799731, PubMed:20483372, PubMed:22396657, PubMed:24901221, PubMed:28435104, PubMed:29497364, PubMed:31714929). Activation and recruitment onto the outer membrane of damaged/dysfunctional mitochondria (OMM) requires Pink1-mediated phosphorylation of both park and ubiquitin (PubMed:18957282, PubMed:24901221, PubMed:20194754, PubMed:22396657, PubMed:18799731, PubMed:18230723, PubMed:25474007, PubMed:27906179). In depolarized mitochondria, mediates the decision between mitophagy or preventing apoptosis by inducing either the poly- or monoubiquitination of porin/VDAC; polyubiquitination of porin promotes mitophagy, while monoubiquitination of porin decreases mitochondrial calcium influx which ultimately inhibits apoptosis (PubMed:32047033). When cellular stress results in irreversible mitochondrial damage, promotes the autophagic degradation of dysfunctional depolarized mitochondria (mitophagy) by promoting the ubiquitination of mitochondrial proteins (PubMed:16672980, PubMed:16672981, PubMed:20194754, PubMed:18957282, PubMed:23509287, PubMed:24192653, PubMed:25474007, PubMed:29497364). Preferentially assembles 'Lys-6'-, 'Lys-11'- and 'Lys-63'-linked polyubiquitin chains following mitochondrial damage, leading to mitophagy (PubMed:32047033, PubMed:23650379). In developing tissues, inhibits JNK-mediated apoptosis by negatively regulating bsk transcription (PubMed:16002472, PubMed:20496123). The Pink1-park pathway also promotes fission and/or inhibits fusion of damaged mitochondria by mediating the ubiquitination and subsequent degradation of proteins involved in mitochondrial fusion/fission such as Marf, Opa1 and fzo (PubMed:18443288, PubMed:17123504, PubMed:18799731, PubMed:18230723, PubMed:20194754, PubMed:23650379, PubMed:24192653, PubMed:24901221, PubMed:29497364). This prevents the refusion of unhealthy mitochondria with the healthy mitochondrial network and/or initiates mitochondrial fragmentation facilitating their later engulfment by autophagosomes (PubMed:18443288, PubMed:17123504, PubMed:18799731,

PubMed:18230723, PubMed:20194754, PubMed:23650379, PubMed:24192653, PubMed:24901221, PubMed:29497364). Regulates motility of damaged mitochondria by phosphorylating Miro which likely promotes its park-dependent degradation by the proteasome; in motor neurons, this inhibits mitochondrial intracellular anterograde transport along the axons which probably increases the chance of the mitochondria being eliminated in the soma (PubMed:22396657). The Pink1-park pathway is also involved in mitochondrial regeneration processes such as promoting mitochondrial biogenesis, activating localized mitochondrial repair, promoting selective turnover of mitochondrial proteins and initiating the mitochondrial import of endogenous proteins (PubMed:16672980, PubMed:20496123, PubMed:20869429, PubMed:23509287, PubMed:23650379, PubMed:24192653, PubMed:25565208, PubMed:29497364). Involved in mitochondrial biogenesis via the ubiquitination of transcriptional repressor Paris which leads to its subsequent proteasomal degradation and allows activation of the transcription factor srl (PubMed:23509287, PubMed:29497364, PubMed:32138754). Promotes localized mitochondrial repair by activating the translation of specific nuclear-encoded mitochondrial RNAs (nc-mtRNAs) on the mitochondrial surface, including several key electron transport chain component nc-mtRNAs (PubMed:23509287, PubMed:25565208).

Cellular Location
Mitochondrion. Cytoplasm, cytosol Note=Translocates from the cytosol to dysfunctional mitochondria that have lost their mitochondrial membrane potential; recruitment to mitochondria is Pink1-dependent.

Tissue Location
In oocytes, accumulates in early egg chambers where it is enriched until stages 9-10, localizing mainly to the posterior pole and anterior margin (at protein level) (PubMed:20869429). After stage 10 it is no longer detected in the oocyte (at protein level) (PubMed:20869429). In embryos, ubiquitously expressed in the early stages (stages 2 to 5) (at protein level) (PubMed:14646593). Expression levels decrease at later stages and becomes restricted to the brain and nerve cord from stage 9 (at protein level) (PubMed:14646593) Relatively higher levels of expression in the head compared to the body (PubMed:16002472). Enriched in the dorsomedial (DM) dopaminergic neurons (PubMed:16002472).

Drosophila Parkin Antibody (C-term) Blocking Peptide - Protocols

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

- Blocking Peptides

Drosophila Parkin Antibody (C-term) Blocking Peptide - Images

Drosophila Parkin Antibody (C-term) Blocking Peptide - Background

Parkin is thought to play a role in the ubiquitin/proteasome pathway for protein degradation. The amino terminus bears sequence homology to ubiquitin while functionally it acts as a RING-type ubiquitin protein ligase (E3) that coordinates the transfer of ubiquitin to substrate proteins, thus targeting them for degradation by the proteasome. Mutations in the human version of the protein are known to cause autosomal recessive juvenile parkinsonism.

Drosophila Parkin Antibody (C-term) Blocking Peptide - References

Zhong,L. et al. J. Biol. Chem. 280 (10), 9425-9430 (2005) Pesah,Y. et al. Development 131 (9), 2183-2194 (2004) Haywood,A.F. et al. BMC Neurosci 5, 14 (2004) Finney,N. et al. J. Biol. Chem. 278 (18), 16054-16058 (2003) Yang,Y. et al. Neuron 37 (6), 911-924 (2003)