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Drosophila Parkin Antibody (N-term)

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP6414a

Product Information

Application	WB, E
Primary Accession	Q7KTX7
Reactivity	Drosophila
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Clone Names	RB8213
Calculated MW	54105
Antigen Region	82-111

Additional Information

Gene ID	40336
Other Names	CG10523-PB; isoform B; Cg10523-pc; isoform c; PARKIN; park; parkin;
Target/Specificity	This Drosophila Parkin antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 82-111 amino acids from the N-terminal region of human Drosophila Parkin.
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 prepared by Saturated Ammonium Sulfate (SAS) precipitation 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	Drosophila Parkin Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

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:20194754, PubMed:23770917, PubMed:24192653,

PubMed:24901221, PubMed:25474007, PubMed:27906179, PubMed:31714929, PubMed:32047033, PubMed:32138754). 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:23650379, PubMed:24901221, PubMed:25474007, 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:18230723, PubMed:18443288, PubMed:18799731, PubMed:18957282, PubMed:20194754, PubMed:20496123, 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:18230723, PubMed:18443288, PubMed:18799731, PubMed:18957282, PubMed:20194754, PubMed:20496123, 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: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:18230723, PubMed:18799731, PubMed:18957282, PubMed:20194754, PubMed:22396657, PubMed:24901221, 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:18957282, PubMed:20194754, 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:23650379, PubMed:32047033). 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:17123504, PubMed:18230723, PubMed:18443288, PubMed:18799731, 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:17123504, PubMed:18230723, PubMed:18443288, PubMed:18799731, 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).

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.

References

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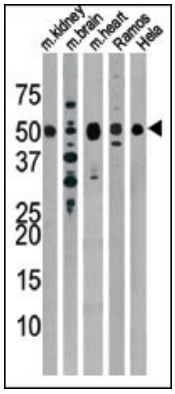
Pesah,Y. et al. Development 131 (9), 2183-2194 (2004)
Haywood,A.F. et al. BMC Neurosci 5, 14 (2004)
Finney,N. et al. J. Biol. Chem. 278 (18), 16054-16058 (2003)
Yang,Y. et al. Neuron 37 (6), 911-924 (2003)

Images



The anti-Drosophila Parkin Pab (Cat. #AP6414a) is used in Western blot to detect Drosophila Parkin in, from left to right, mouse kidney, mouse brain, mouse heart, Ramos, and Hela tissue lysates.