

**Parkin Rabbit mAb**  
Catalog # AP78993**Specification**

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**Parkin Rabbit mAb - Product Information**

Application	<b>WB</b>
Primary Accession	<a href="#">O60260</a>
Reactivity	<b>Human, Mouse, Rat</b>
Host	<b>Rabbit</b>
Clonality	<b>Monoclonal Antibody</b>
Calculated MW	<b>51641</b>

**Parkin Rabbit mAb - Additional Information**

Gene ID 5071

**Other Names**  
PRKN**Dilution**  
WB~~1/500-1/1000**Format**  
Liquid**Parkin Rabbit mAb - Protein Information**Name PRKN ([HGNC:8607](#))**Synonyms** PARK2**Function**

Functions within a multiprotein E3 ubiquitin ligase complex, catalyzing the covalent attachment of ubiquitin moieties onto substrate proteins (PubMed:<a href="http://www.uniprot.org/citations/10888878" target="\_blank">10888878</a>, PubMed:<a href="http://www.uniprot.org/citations/10973942" target="\_blank">10973942</a>, PubMed:<a href="http://www.uniprot.org/citations/11431533" target="\_blank">11431533</a>, PubMed:<a href="http://www.uniprot.org/citations/12150907" target="\_blank">12150907</a>, PubMed:<a href="http://www.uniprot.org/citations/12628165" target="\_blank">12628165</a>, PubMed:<a href="http://www.uniprot.org/citations/15105460" target="\_blank">15105460</a>, PubMed:<a href="http://www.uniprot.org/citations/16135753" target="\_blank">16135753</a>, PubMed:<a href="http://www.uniprot.org/citations/21376232" target="\_blank">21376232</a>, PubMed:<a href="http://www.uniprot.org/citations/21532592" target="\_blank">21532592</a>, PubMed:<a href="http://www.uniprot.org/citations/22396657" target="\_blank">22396657</a>, PubMed:<a href="http://www.uniprot.org/citations/23620051" target="\_blank">23620051</a>, PubMed:<a href="http://www.uniprot.org/citations/23754282" target="\_blank">23754282</a>, PubMed:<a href="http://www.uniprot.org/citations/24660806" target="\_blank">24660806</a>, PubMed:<a href="http://www.uniprot.org/citations/24751536" target="\_blank">24751536</a>, PubMed:<a

href="http://www.uniprot.org/citations/29311685" target="\_blank">29311685</a>, PubMed:<a href="http://www.uniprot.org/citations/32047033" target="\_blank">32047033</a>). Substrates include SYT11 and VDAC1 (PubMed:<a href="http://www.uniprot.org/citations/29311685" target="\_blank">29311685</a>, PubMed:<a href="http://www.uniprot.org/citations/32047033" target="\_blank">32047033</a>). Other substrates are BCL2, CCNE1, GPR37, RHOT1/MIRO1, MFN1, MFN2, STUB1, SNCAIP, SEPTIN5, TOMM20, USP30, ZNF746, MIRO1 and AIMP2 (PubMed:<a href="http://www.uniprot.org/citations/10888878" target="\_blank">10888878</a>, PubMed:<a href="http://www.uniprot.org/citations/10973942" target="\_blank">10973942</a>, PubMed:<a href="http://www.uniprot.org/citations/11431533" target="\_blank">11431533</a>, PubMed:<a href="http://www.uniprot.org/citations/12150907" target="\_blank">12150907</a>, PubMed:<a href="http://www.uniprot.org/citations/12628165" target="\_blank">12628165</a>, PubMed:<a href="http://www.uniprot.org/citations/15105460" target="\_blank">15105460</a>, PubMed:<a href="http://www.uniprot.org/citations/16135753" target="\_blank">16135753</a>, PubMed:<a href="http://www.uniprot.org/citations/21376232" target="\_blank">21376232</a>, PubMed:<a href="http://www.uniprot.org/citations/21532592" target="\_blank">21532592</a>, PubMed:<a href="http://www.uniprot.org/citations/22396657" target="\_blank">22396657</a>, PubMed:<a href="http://www.uniprot.org/citations/23620051" target="\_blank">23620051</a>, PubMed:<a href="http://www.uniprot.org/citations/23754282" target="\_blank">23754282</a>, PubMed:<a href="http://www.uniprot.org/citations/24660806" target="\_blank">24660806</a>, PubMed:<a href="http://www.uniprot.org/citations/24751536" target="\_blank">24751536</a>). Mediates monoubiquitination as well as 'Lys-6', 'Lys-11', 'Lys-48'-linked and 'Lys-63'-linked polyubiquitination of substrates depending on the context (PubMed:<a href="http://www.uniprot.org/citations/19229105" target="\_blank">19229105</a>, PubMed:<a href="http://www.uniprot.org/citations/20889974" target="\_blank">20889974</a>, PubMed:<a href="http://www.uniprot.org/citations/25474007" target="\_blank">25474007</a>, PubMed:<a href="http://www.uniprot.org/citations/25621951" target="\_blank">25621951</a>, PubMed:<a href="http://www.uniprot.org/citations/32047033" target="\_blank">32047033</a>). Participates in the removal and/or detoxification of abnormally folded or damaged protein by mediating 'Lys-63'-linked polyubiquitination of misfolded proteins such as PARK7: 'Lys-63'-linked polyubiquitinated misfolded proteins are then recognized by HDAC6, leading to their recruitment to aggresomes, followed by degradation (PubMed:<a href="http://www.uniprot.org/citations/17846173" target="\_blank">17846173</a>, PubMed:<a href="http://www.uniprot.org/citations/19229105" target="\_blank">19229105</a>). Mediates 'Lys-63'-linked polyubiquitination of a 22 kDa O-linked glycosylated isoform of SNCAIP, possibly playing a role in Lewy-body formation (PubMed:<a href="http://www.uniprot.org/citations/11431533" target="\_blank">11431533</a>, PubMed:<a href="http://www.uniprot.org/citations/11590439" target="\_blank">11590439</a>, PubMed:<a href="http://www.uniprot.org/citations/15105460" target="\_blank">15105460</a>, PubMed:<a href="http://www.uniprot.org/citations/15728840" target="\_blank">15728840</a>, PubMed:<a href="http://www.uniprot.org/citations/19229105" target="\_blank">19229105</a>). Mediates monoubiquitination of BCL2, thereby acting as a positive regulator of autophagy (PubMed:<a href="http://www.uniprot.org/citations/20889974" target="\_blank">20889974</a>). 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:<a href="http://www.uniprot.org/citations/11439185" target="\_blank">11439185</a>, PubMed:<a href="http://www.uniprot.org/citations/18957282" target="\_blank">18957282</a>, PubMed:<a href="http://www.uniprot.org/citations/19029340" target="\_blank">19029340</a>, PubMed:<a href="http://www.uniprot.org/citations/19966284" target="\_blank">19966284</a>, PubMed:<a href="http://www.uniprot.org/citations/21376232" target="\_blank">21376232</a>, PubMed:<a href="http://www.uniprot.org/citations/22082830" target="\_blank">22082830</a>, PubMed:<a href="http://www.uniprot.org/citations/22396657" target="\_blank">22396657</a>, PubMed:<a href="http://www.uniprot.org/citations/23620051" target="\_blank">23620051</a>, PubMed:<a href="http://www.uniprot.org/citations/23933751" target="\_blank">23933751</a>, PubMed:<a href="http://www.uniprot.org/citations/24660806" target="\_blank">24660806</a>, PubMed:<a href="http://www.uniprot.org/citations/24784582" target="\_blank">24784582</a>, PubMed:<a href="http://www.uniprot.org/citations/24896179" target="\_blank">24896179</a>, PubMed:<a href="http://www.uniprot.org/citations/25474007" target="\_blank">25474007</a>

target="\_blank">25474007</a>, PubMed:<a href="http://www.uniprot.org/citations/25527291" target="\_blank">25527291</a>, PubMed:<a href="http://www.uniprot.org/citations/32047033" target="\_blank">32047033</a>). 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:<a href="http://www.uniprot.org/citations/11439185" target="\_blank">11439185</a>, PubMed:<a href="http://www.uniprot.org/citations/19029340" target="\_blank">19029340</a>, PubMed:<a href="http://www.uniprot.org/citations/19801972" target="\_blank">19801972</a>, PubMed:<a href="http://www.uniprot.org/citations/19966284" target="\_blank">19966284</a>, PubMed:<a href="http://www.uniprot.org/citations/21376232" target="\_blank">21376232</a>, PubMed:<a href="http://www.uniprot.org/citations/22082830" target="\_blank">22082830</a>, PubMed:<a href="http://www.uniprot.org/citations/22396657" target="\_blank">22396657</a>, PubMed:<a href="http://www.uniprot.org/citations/23620051" target="\_blank">23620051</a>, PubMed:<a href="http://www.uniprot.org/citations/23685073" target="\_blank">23685073</a>, PubMed:<a href="http://www.uniprot.org/citations/23933751" target="\_blank">23933751</a>, PubMed:<a href="http://www.uniprot.org/citations/24896179" target="\_blank">24896179</a>, PubMed:<a href="http://www.uniprot.org/citations/25527291" target="\_blank">25527291</a>, PubMed:<a href="http://www.uniprot.org/citations/32047033" target="\_blank">32047033</a>, PubMed:<a href="http://www.uniprot.org/citations/33499712" target="\_blank">33499712</a>). Activation and recruitment onto the outer membrane of damaged/dysfunctional mitochondria (OMM) requires PINK1-mediated phosphorylation of both PRKN and ubiquitin (PubMed:<a href="http://www.uniprot.org/citations/24660806" target="\_blank">24660806</a>, PubMed:<a href="http://www.uniprot.org/citations/24784582" target="\_blank">24784582</a>, PubMed:<a href="http://www.uniprot.org/citations/25474007" target="\_blank">25474007</a>, PubMed:<a href="http://www.uniprot.org/citations/25527291" target="\_blank">25527291</a>). After mitochondrial damage, functions with PINK1 to mediate the decision between mitophagy or preventing apoptosis by inducing either the poly- or monoubiquitination of VDAC1, respectively; polyubiquitination of VDAC1 promotes mitophagy, while monoubiquitination of VDAC1 decreases mitochondrial calcium influx which ultimately inhibits apoptosis (PubMed:<a href="http://www.uniprot.org/citations/27534820" target="\_blank">27534820</a>, PubMed:<a href="http://www.uniprot.org/citations/32047033" target="\_blank">32047033</a>). When cellular stress results in irreversible mitochondrial damage, promotes the autophagic degradation of dysfunctional depolarized mitochondria (mitophagy) by promoting the ubiquitination of mitochondrial proteins such as TOMM20, RHOT1/MIRO1, MFN1 and USP30 (PubMed:<a href="http://www.uniprot.org/citations/19029340" target="\_blank">19029340</a>, PubMed:<a href="http://www.uniprot.org/citations/19966284" target="\_blank">19966284</a>, PubMed:<a href="http://www.uniprot.org/citations/21753002" target="\_blank">21753002</a>, PubMed:<a href="http://www.uniprot.org/citations/22396657" target="\_blank">22396657</a>, PubMed:<a href="http://www.uniprot.org/citations/23620051" target="\_blank">23620051</a>, PubMed:<a href="http://www.uniprot.org/citations/23685073" target="\_blank">23685073</a>, PubMed:<a href="http://www.uniprot.org/citations/23933751" target="\_blank">23933751</a>, PubMed:<a href="http://www.uniprot.org/citations/24896179" target="\_blank">24896179</a>, PubMed:<a href="http://www.uniprot.org/citations/25527291" target="\_blank">25527291</a>). Preferentially assembles 'Lys-6', 'Lys-11' and 'Lys-63'-linked polyubiquitin chains, leading to mitophagy (PubMed:<a href="http://www.uniprot.org/citations/25621951" target="\_blank">25621951</a>, PubMed:<a href="http://www.uniprot.org/citations/32047033" target="\_blank">32047033</a>). The PINK1-PRKN pathway also promotes fission of damaged mitochondria by PINK1-mediated phosphorylation which promotes the PRKN-dependent degradation of mitochondrial proteins involved in fission such as MFN2 (PubMed:<a href="http://www.uniprot.org/citations/23620051" target="\_blank">23620051</a>). This prevents the refusion of unhealthy mitochondria with the mitochondrial network or initiates mitochondrial fragmentation facilitating their later engulfment by autophagosomes (PubMed:<a href="http://www.uniprot.org/citations/23620051" target="\_blank">23620051</a>). Regulates motility of damaged mitochondria via 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:<a href="http://www.uniprot.org/citations/22396657" target="\_blank">22396657</a>). Involved in

mitochondrial biogenesis via the 'Lys-48'-linked polyubiquitination of transcriptional repressor ZNF746/PARIS which leads to its subsequent proteasomal degradation and allows activation of the transcription factor PPARGC1A (PubMed:<a href="http://www.uniprot.org/citations/21376232" target="\_blank">21376232</a>). Limits the production of reactive oxygen species (ROS) (PubMed:<a href="http://www.uniprot.org/citations/18541373" target="\_blank">18541373</a>). Regulates cyclin-E during neuronal apoptosis (PubMed:<a href="http://www.uniprot.org/citations/12628165" target="\_blank">12628165</a>). In collaboration with CHPF isoform 2, may enhance cell viability and protect cells from oxidative stress (PubMed:<a href="http://www.uniprot.org/citations/22082830" target="\_blank">22082830</a>). Independently of its ubiquitin ligase activity, protects from apoptosis by the transcriptional repression of p53/TP53 (PubMed:<a href="http://www.uniprot.org/citations/19801972" target="\_blank">19801972</a>). May protect neurons against alpha synuclein toxicity, proteasomal dysfunction, GPR37 accumulation, and kainate-induced excitotoxicity (PubMed:<a href="http://www.uniprot.org/citations/11439185" target="\_blank">11439185</a>). May play a role in controlling neurotransmitter trafficking at the presynaptic terminal and in calcium-dependent exocytosis. May represent a tumor suppressor gene (PubMed:<a href="http://www.uniprot.org/citations/12719539" target="\_blank">12719539</a>).

### Cellular Location

Cytoplasm, cytosol. Nucleus. Endoplasmic reticulum. Mitochondrion. Mitochondrion outer membrane {ECO:0000250|UniProtKB:Q9WVS6}. Cell projection, neuron projection. Postsynaptic density {ECO:0000250|UniProtKB:Q9WVS6}. Presynapse {ECO:0000250|UniProtKB:Q9WVS6}. Note=Mainly localizes in the cytosol (PubMed:19029340, PubMed:19229105). Co-localizes with SYT11 in neurites (PubMed:12925569). Co-localizes with SNCAIP in brainstem Lewy bodies (PubMed:10319893, PubMed:11431533). Translocates to dysfunctional mitochondria that have lost the mitochondrial membrane potential; recruitment to mitochondria is PINK1-dependent (PubMed:18957282, PubMed:19966284, PubMed:23620051, PubMed:24898855) Mitochondrial localization also gradually increases with cellular growth (PubMed:22082830).

### Tissue Location

Highly expressed in the brain including the substantia nigra (PubMed:19501131, PubMed:9560156). Expressed in heart, testis and skeletal muscle (PubMed:9560156). Expression is down-regulated or absent in tumor biopsies, and absent in the brain of PARK2 patients (PubMed:12719539, PubMed:14614460). Overexpression protects dopamine neurons from kainate-mediated apoptosis (PubMed:12628165) Found in serum (at protein level) (PubMed:19501131)

### Parkin Rabbit mAb - Protocols

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

### Parkin Rabbit mAb - Images



