

**Parkin (phospho Ser131) Polyclonal Antibody**  
Catalog # AP67894**Specification****Parkin (phospho Ser131) Polyclonal Antibody - Product Information**

Application	IHC
Primary Accession	<a href="#">O60260</a>
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal

**Parkin (phospho Ser131) Polyclonal Antibody - Additional Information****Gene ID** 5071**Other Names**

PARK2; PRKN; E3 ubiquitin-protein ligase parkin; Parkinson juvenile disease protein 2; Parkinson disease protein 2

**Dilution**

IHC~~Immunohistochemistry: 1/100 - 1/300. ELISA: 1/20000. Not yet tested in other applications.

**Format**

Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.

**Storage Conditions**

-20°C

**Parkin (phospho Ser131) Polyclonal Antibody - 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:&lt;a

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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>, 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

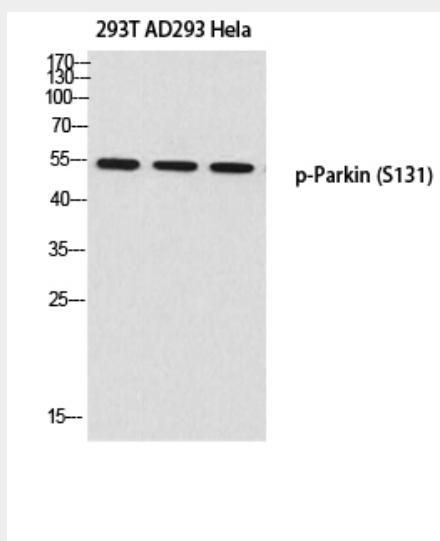
Highly expressed in the brain including the substantia nigra (PubMed:19501131, PubMed:9560156). Expressed in heart, testis and skeletal muscle (PubMed:9560156). Expression is down-regulated or absent in tumor biopsies, and absent in the brain of PARK2 patients (PubMed:12719539, PubMed:14614460). Overexpression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

#### Parkin (phospho Ser131) Polyclonal Antibody - 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 (phospho Ser131) Polyclonal Antibody - Images



### Parkin (phospho Ser131) Polyclonal Antibody - Background

Functions within a multiprotein E3 ubiquitin ligase complex, catalyzing the covalent attachment of ubiquitin moieties onto substrate proteins, such as BCL2, SYT11, CCNE1, GPR37, RHOT1/MIRO1, MFN1, MFN2, STUB1, SNCAIP, SEPT5, TOMM20, USP30, ZNF746 and AIMP2 (PubMed:10973942, PubMed:10888878, PubMed:11431533, PubMed:12150907, PubMed:12628165, PubMed:16135753, PubMed:21376232, PubMed:23754282, PubMed:23620051, PubMed:24660806, PubMed:24751536). Mediates monoubiquitination as well as 'Lys-6', 'Lys-11', 'Lys-48'-linked and 'Lys-63'-linked polyubiquitination of substrates depending on the context (PubMed:19229105, PubMed:20889974, PubMed:25621951). 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:17846173, PubMed:19229105). Mediates 'Lys-63'-linked polyubiquitination of a 22 kDa O-linked glycosylated isoform of SNCAIP, possibly playing a role in Lewy-body formation (PubMed:11590439, PubMed:11431533, PubMed:19229105, PubMed:11590439, PubMed:15728840). Mediates monoubiquitination of BCL2, thereby acting as a positive regulator of autophagy (PubMed:20889974). Promotes the autophagic degradation of dysfunctional depolarized mitochondria (mitophagy) by promoting the ubiquitination of mitochondrial proteins such as TOMM20, RHOT1/MIRO1 and USP30 (PubMed:19029340, PubMed:19966284, PubMed:23620051, PubMed:24896179, PubMed:25527291). Preferentially assembles 'Lys-6', 'Lys-11' and 'Lys-63'-linked polyubiquitin chains following mitochondrial damage, leading to mitophagy (PubMed:25621951). Mediates 'Lys-48'-linked polyubiquitination of ZNF746, followed by degradation of ZNF746 by the proteasome; possibly playing a role in the regulation of neuron death (PubMed:21376232). Limits the production of reactive oxygen species (ROS). Regulates cyclin-E during neuronal apoptosis. In collaboration with CHPF isoform 2, may enhance cell viability and protect cells from oxidative stress (PubMed:22082830). Independently of its ubiquitin ligase activity, protects from apoptosis by the transcriptional repression of p53/TP53 (PubMed:19801972). May protect neurons against alpha synuclein toxicity, proteasomal dysfunction, GPR37 accumulation, and kainate-induced excitotoxicity (PubMed:11439185). May play a role in controlling neurotransmitter trafficking at the presynaptic terminal and in calcium-dependent exocytosis. May represent a tumor suppressor gene.