



Rabbit Anti-phospho-PARK2 (Ser378) antibody

SL5553R

Product Name:	phospho-PARK2 (Ser378)
Chinese Name:	磷酸化帕金森蛋白抗体
Alias:	Parkin (phospho S378); p-Parkin (phospho S378); PARK2(phospho Ser378); AR JP; E3 ubiquitin protein ligase parkin; FRA6E; LPRS 2; LPRS2; PARK 2; PARK2; Parkinson disease (autosomal recessive juvenile) 2; Parkinson disease protein 2; Parkinson juvenile disease protein 2; PDJ; PRKN 2; PRKN; PRKN2; Ubiquitin E3 ligase PRKN.
Organism Species:	Rabbit
Clonality:	Polyclonal
React Species:	Human,Mouse,Pig,Guinea Pig,
Applications:	WB=1:500-2000ELISA=1:500-1000IHC-P=1:400-800IHC-F=1:400-800IF=1:100-500 (Paraffin sections need antigen repair) not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight:	51kDa
Cellular localization:	The nucleuscytoplasmicThe cell membrane
Form:	Lyophilized or Liquid
Concentration:	1mg/ml
immunogen:	KLH conjugated Synthesised phosphopeptide derived from human PARK2 around the phosphorylation site of Ser378:EC(p-S)AV
Lsotype:	IgG
Purification:	affinity purified by Protein A
Storage Buffer:	0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.
Storage:	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
PubMed:	PubMed
Product Detail:	Parkinson's Disease, the second most common neurodegenerative disease after Alzheimer's Disease, is characterized by the loss of dopaminergic neurons and the

presence of Lewy bodies (comprised of alpha synuclein and parkin inclusions). Autosomal Recessive Juvenile Parkinsonism (AR-JP) is a recently described form of Parkinson's Disease that has been linked to a gene that codes for parkin. Parkin, a 52 kDa protein, has a suggested 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.

Function:

Functions within a multiprotein E3 ubiquitin ligase complex, catalyzing the covalent attachment of ubiquitin moieties onto substrate proteins, such as BCL2, SYT11, CCNE1, GPR37, STUB1, a 22 kDa O-linked glycosylated isoform of SNCAIP, SEPT5, ZNF746 and AIMP2. Mediates monoubiquitination as well as 'Lys-48'-linked and 'Lys-63'-linked polyubiquitination of substrates depending on the context. 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. Mediates 'Lys-63'-linked polyubiquitination of SNCAIP, possibly playing a role in Lewy-body formation. Mediates monoubiquitination of BCL2, thereby acting as a positive regulator of autophagy. Promotes the autophagic degradation of dysfunctional depolarized mitochondria. Mediates 'Lys-48'-linked polyubiquitination of ZNF746, followed by degradation of ZNF746 by the proteasome; possibly playing a role in regulation of neuron death. Limits the production of reactive oxygen species (ROS). Loss of this ubiquitin ligase activity appears to be the mechanism underlying pathogenesis of PARK2. May protect neurons against alpha synuclein toxicity, proteasomal dysfunction, GPR37 accumulation, and kainate-induced excitotoxicity. May play a role in controlling neurotransmitter trafficking at the presynaptic terminal and in calcium-dependent exocytosis. Regulates cyclin-E during neuronal apoptosis. May represent a tumor suppressor gene.

Subunit:

Forms an E3 ubiquitin ligase complex with UBE2L3 or UBE2L6. Mediates 'Lys-63'-linked polyubiquitination by associating with UBE2V1. Part of a SCF-like complex, consisting of PARK2, CUL1 and FBXW7. Interacts with SNCAIP. Binds to the C2A and C2B domains of SYT11. Interacts and regulates the turnover of SEPT5. Part of a complex, including STUB1, HSP70 and GPR37. The amount of STUB1 in the complex increases during ER stress. STUB1 promotes the dissociation of HSP70 from PARK2 and GPR37, thus facilitating PARK2-mediated GPR37 ubiquitination. HSP70 transiently associates with unfolded GPR37 and inhibits the E3 activity of PARK2, whereas, STUB1 enhances the E3 activity of PARK2 through promotion of dissociation of HSP70 from PARK2-GPR37 complexes. Interacts with PSMD4 and PACRG. Interacts with LRRK2. Interacts with RANBP2. Interacts with SUMO1 but not SUMO2, which promotes nuclear localization and autoubiquitination. Interacts (via first RING-type domain) with AIMP2 (via N-terminus). Interacts with PSMA7 and RNF41.

Interacts with PINK1.

Subcellular Location:

Cytoplasm, cytosol. Nucleus. Endoplasmic reticulum. Mitochondrion. Note=Mainly localizes in the cytosol. Co-localizes with SYT11 in neurites. Co-localizes with SNCAIP in brainstem Lewy bodies. Relocates to dysfunctional mitochondria that have lost the mitochondrial membrane potential; recruitment to mitochondria is PINK1-dependent.

Tissue Specificity:

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

Post-translational modifications:

Auto-ubiquitinates in an E2-dependent manner leading to its own degradation. Also polyubiquitinated by RNF41 for proteasomal degradation. S-nitrosylated. The inhibition of PARK2 ubiquitin E3 ligase activity by S-nitrosylation could contribute to the degenerative process in PD by impairing the ubiquitination of PARK2 substrates.

DISEASE:

Defects in PARK2 are a cause of Parkinson disease (PARK) [MIM:168600]. A complex neurodegenerative disorder characterized by bradykinesia, resting tremor, muscular rigidity and postural instability. Additional features are characteristic postural abnormalities, dysautonomia, dystonic cramps, and dementia. The pathology of Parkinson disease involves the loss of dopaminergic neurons in the substantia nigra and the presence of Lewy bodies (intra-neuronal accumulations of aggregated proteins), in surviving neurons in various areas of the brain. The disease is progressive and usually manifests after the age of 50 years, although early-onset cases (before 50 years) are known. The majority of the cases are sporadic suggesting a multifactorial etiology based on environmental and genetic factors. However, some patients present with a positive family history for the disease. Familial forms of the disease usually begin at earlier ages and are associated with atypical clinical features.

Defects in PARK2 are the cause of Parkinson disease type 2 (PARK2) [MIM:600116]; also known as early-onset parkinsonism with diurnal fluctuation (EPDF) or autosomal recessive juvenile Parkinson disease (PDJ). A neurodegenerative disorder characterized by bradykinesia, rigidity, postural instability, tremor, and onset usually before 40. It differs from classic Parkinson disease by early DOPA-induced dyskinesia, diurnal fluctuation of the symptoms, sleep benefit, dystonia and hyper-reflexia. Dementia is absent. Pathologically, patients show loss of dopaminergic neurons in the substantia nigra, similar to that seen in Parkinson disease; however, Lewy bodies (intra-neuronal accumulations of aggregated proteins) are absent.

Similarity:

Belongs to the RBR family. Parkin subfamily.
Contains 1 IBR-type zinc finger.
Contains 2 RING-type zinc fingers.
Contains 1 ubiquitin-like domain.

SWISS:
O60260

Gene ID:
5071

Database links:

[Entrez Gene: 5071](#) Human

[Omim: 602544](#) Human

[SwissProt: O60260](#) Human

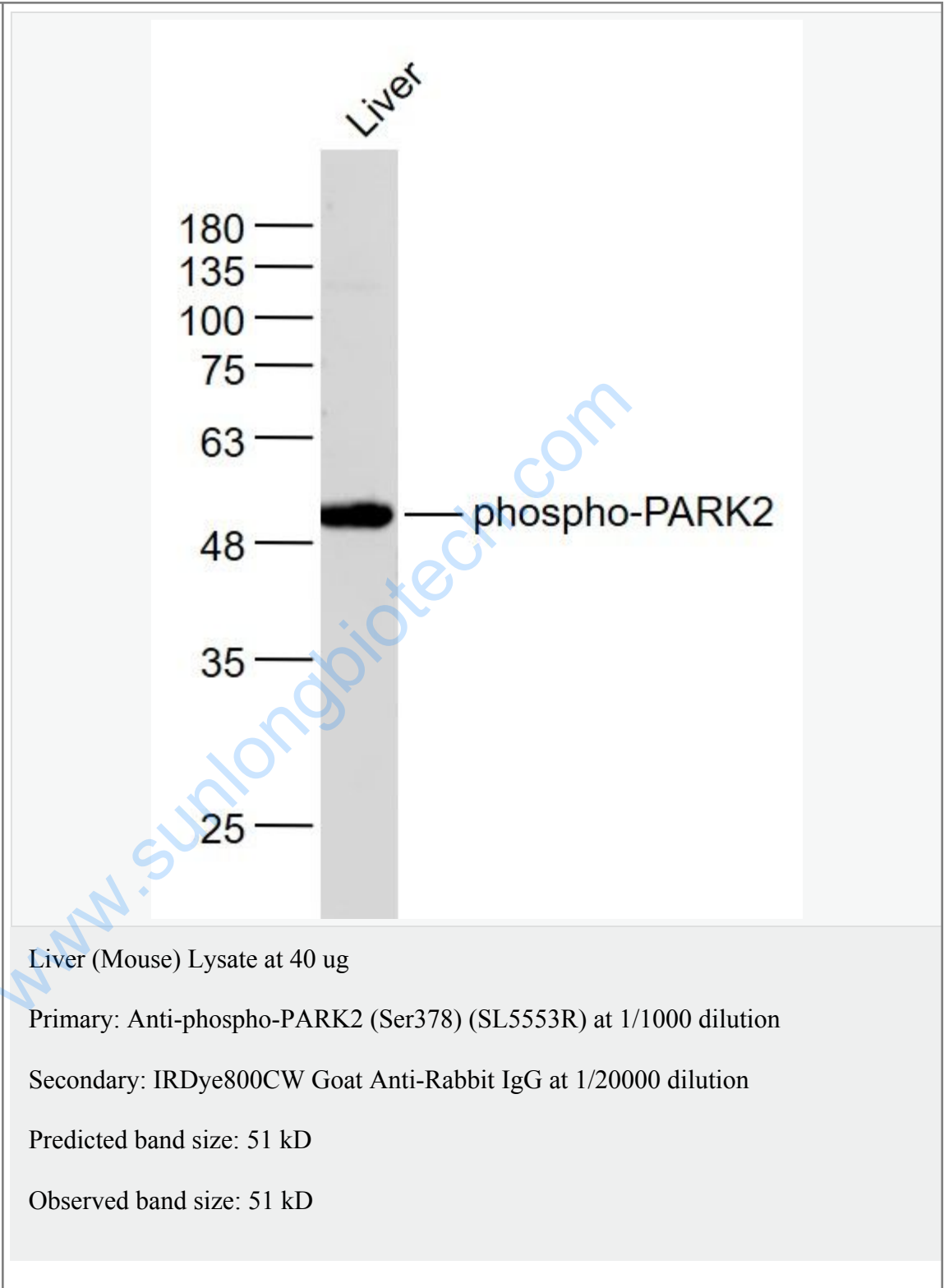
[Unigene: 132954](#) Human

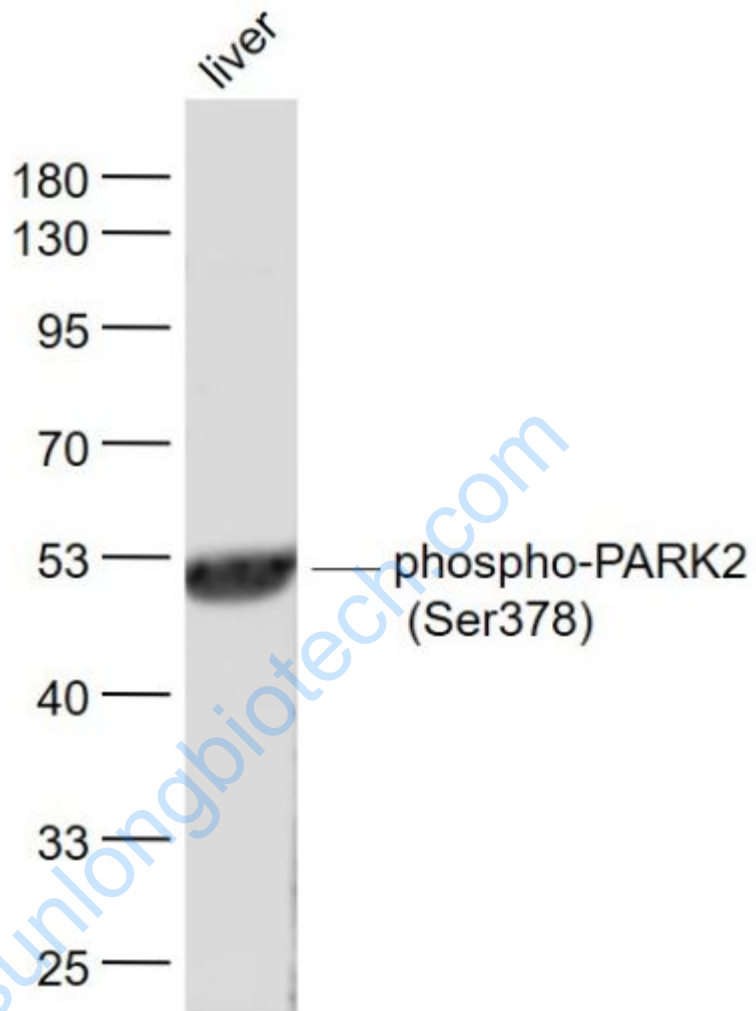
Important Note:

This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

parkin基因的突变, 患者很早就会出现帕金森氏症的症状, Parkin属于RBR蛋白家族, 与Ubiquitin相关蛋白分解途径有关.

Picture:





Sample:

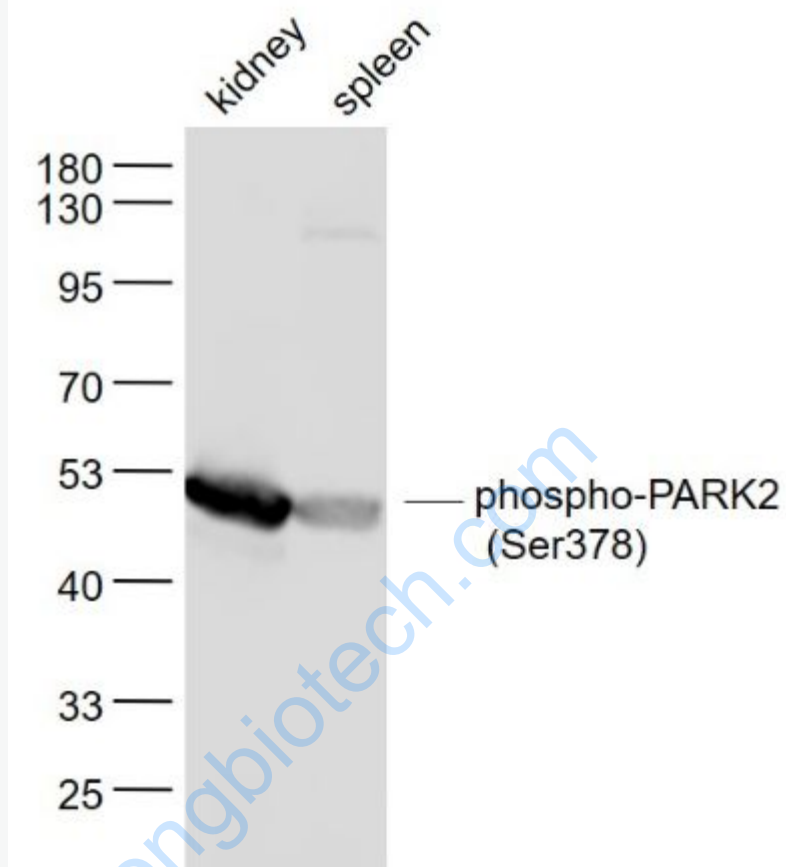
Liver (Mouse) Lysate at 40 ug

Primary: Anti- phospho-PARK2 (Ser378) (SL5553R) at 1/1000 dilution

Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution

Predicted band size: 51 kD

Observed band size: 51 kD



Sample:

Kidney (Mouse) Lysate at 40 ug

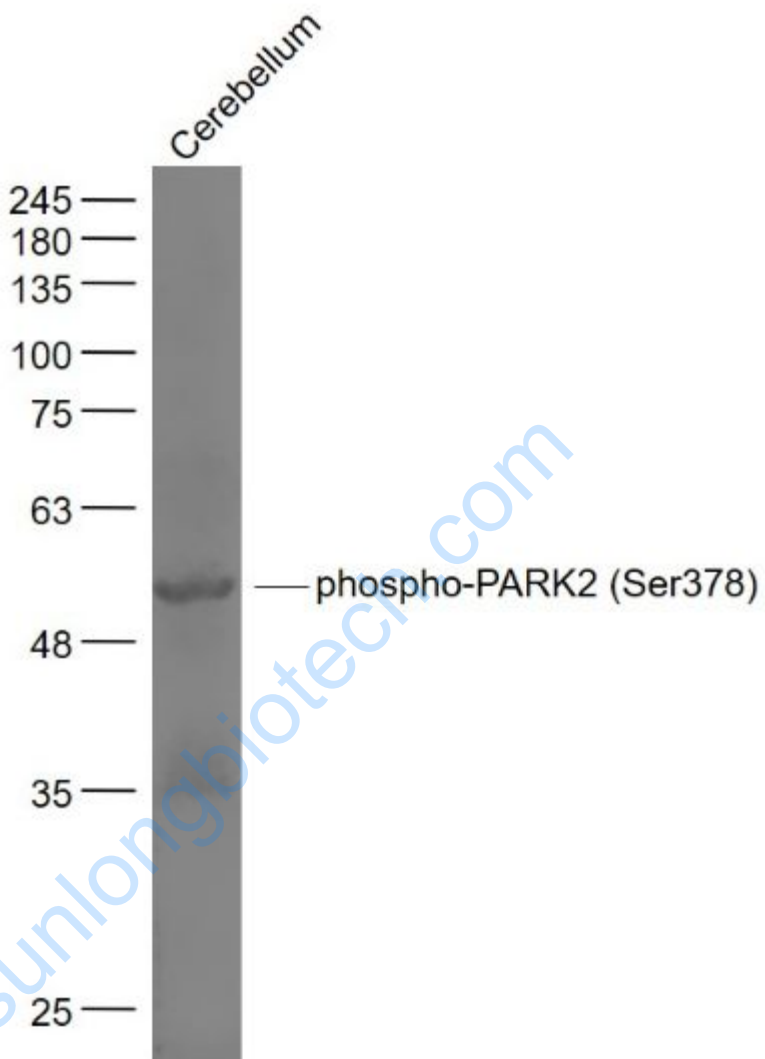
Spleen (Mouse) Lysate at 40 ug

Primary: Anti- phospho-PARK2 (Ser378) (SL5553R) at 1/1000 dilution

Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution

Predicted band size: 51 kD

Observed band size: 51 kD



Sample:

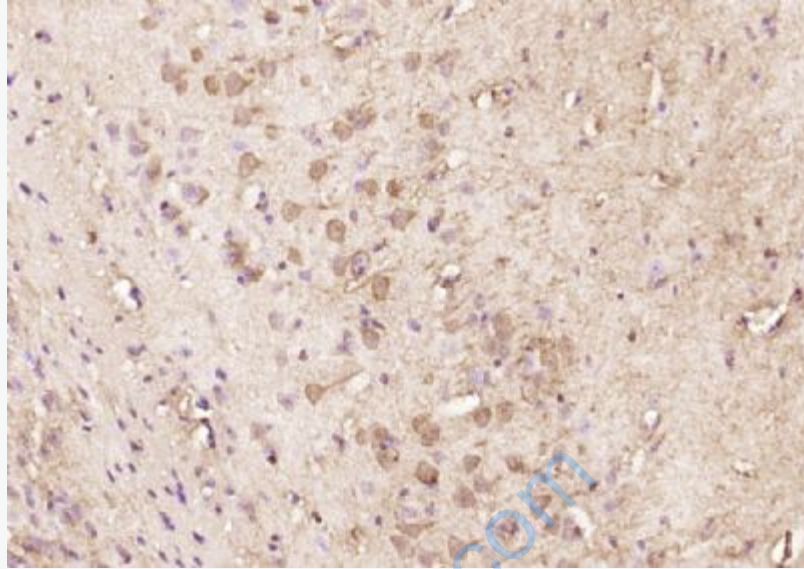
Cerebellum (Mouse) Lysate at 40 ug

Primary: Anti- phospho-PARK2 (Ser378) (SL5553R) at 1/1000 dilution

Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution

Predicted band size: 51 kD

Observed band size: 51 kD



Paraformaldehyde-fixed, paraffin embedded (mouse brain); Antigen retrieval by boiling in sodium citrate buffer (pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 20 minutes; Blocking buffer (normal goat serum) at 37°C for 30min; Antibody incubation with (phospho-PARK2 (Ser378)) Polyclonal Antibody, Unconjugated (SL5553R) at 1:200 overnight at 4°C, followed by operating according to SP Kit(Rabbit) (sp-0023) instructions and DAB staining.