



Parkin Rabbit mAb

Catalog No	YP-rAb-17608
Isotype	IgG
Reactivity	Human,Mouse,Rat
Applications	WB,IF,ELISA
Gene Name	PARK2
Protein Name	E3 ubiquitin-protein ligase parkin
Purification Process	Protein A
Specificity	Endogenous
Formulation	PBS, 50% glycerol, 0.05% Proclin 300, 0.05%BSA
Source	Monoclonal, Rabbit,IgG
Dilution	WB 1:2000-1:10000; IF 1:200-1:1000; ELISA 1:5000-1:20000;
Concentration	0.5 mg/ml
Purity	≥90%
Storage Stability	-15 ° C to -25 ° C/1 year(Do not lower than -25 ° C)
Synonyms	Parkin;E3 ubiquitin-protein ligase parkin;PARK2 ; PRKN ; E3 ubiquitin-protein ligase parkin ; Parkinson juvenile disease protein 2 ; Parkinson disease protein 2;PARK2
Observed Band	52kD
Calculated Molecular Weight	52kD
Cell Pathway	Cytoplasm
Tissue Specificity	Highly expressed in the brain including the substantia nigra (PubMed:9560156, PubMed:19501131). 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:14614460, PubMed:12719539). Overexpression protects dopamine neurons from kainate-mediated apoptosis (PubMed:12628165). Found in serum (at protein level) (PubMed:19501131).
Function	Disease:Defects in PARK2 are a cause of Parkinson disease (PD) [MIM:168600]. PD is a complex, multifactorial disorder that typically manifests after the age of 50 years, although early-onset cases (before 50 years) are known. PD generally arises as a sporadic condition but is occasionally inherited as a simple mendelian trait. Although sporadic and familial PD are very similar, inherited forms of the disease usually begin at earlier ages and are associated with atypical clinical features. PD is characterized by bradykinesia, resting tremor, muscular rigidity



and postural instability, as well as by a clinically significant response to treatment with levodopa. The pathology of PD involves the loss of dopaminergic neurons in the substantia nigra and the presence of Lewy bodies (intraneuronal accumulations of aggregated proteins), in surviving neurons in various areas of the brain. Disease: Defects in PARK2 are the cause of autosomal recessive early onset Parkinson disease 2 (PARK2) [MIM:600116]; also known as early-onset parkinsonism with diurnal fluctuation (EPDF) or autosomal recessive juvenile Parkinson disease (PDJ). PARK2 is symptomatically different in several aspects from idiopathic Parkinson disease, although classic symptoms such as bradykinesia, rigidity and tremor are present. Additional clinical features include early DOPA-induced dyskinesia, diurnal fluctuation of the symptoms, sleep benefit, dystonia and hyper-reflexia. PARK2 is usually characterized by onset before 40, with a mean age at onset of 23.2 years. Pathologically, PARK2 patients show loss of dopaminergic neurons in the substantia nigra, similar to that seen in Parkinson disease; however, Lewy bodies (intraneuronal accumulations of aggregated proteins) are absent. Disease: Defects in PARK2 may be involved in the development and/or progression of ovarian cancer. Domain: The ubiquitin-like domain binds the PSMD4 subunit of 26S proteasomes. Function: Functions within a multiprotein E3 ubiquitin ligase complex, catalyzing the covalent attachment of ubiquitin moieties onto substrate proteins. These substrates include SYT11, CCNE1, GPR37, STUB1, a 22 kDa O-linked glycosylated isoform of SNCAIP and SEPT5. May play a more general role in the ubiquitin proteasomal pathway by participating in the removal and/or detoxification of abnormally folded or damaged protein. 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. miscellaneous: The parkin locus (PRKN), adjacent to the 6q telomere is hyper-recombinable and lies within FRA6E, the third most common fragile site in tumor tissue. pathway: Protein modification; protein ubiquitination. PTM: Auto-ubiquitinates in an E2-dependent manner leading to its own degradation. PTM: 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. similarity: Belongs to the RBR family. Parkin subfamily. similarity: Contains 1 IBR-type zinc finger. similarity: Contains 1 ubiquitin-like domain. similarity: Contains 2 RING-type zinc fingers. subcellular location: Co-localizes with SYT11 in neurites. Co-localizes with SNCAIP in brainstem Lewy bodies. subunit: Forms a n E3 ubiquitin ligase complex with UBE2L3 or UBE2L6. 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. 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.

Background

The precise function of this gene is unknown; however, the encoded protein is a component of a multiprotein E3 ubiquitin ligase complex that mediates the targeting of substrate proteins for proteasomal degradation. Mutations in this gene are known to cause Parkinson disease and autosomal recessive juvenile Parkinson disease. Alternative splicing of this gene produces multiple transcript variants encoding distinct isoforms. Additional splice variants of this gene have been described but currently lack transcript support. [provided by RefSeq, Jul 2008].

matters needing attention

Avoid repeated freezing and thawing!

杭州臻优品生物科技有限公司

热销产品:

蛋白、一抗、抗体对、ELISA试剂盒、生化试剂盒
CCK8试剂盒、QPCR检测试剂盒

检测服务:

ELISA检测及定制服务 | 生化检测 | PCR、QPCR检测 | WB检测
| CO-IP检测 | 切片 | 染色 | 免疫组化 | 免疫荧光 | 透射电镜全套
| 宏基因组、转录组、基因组、蛋白组、代谢组测序



关注官网

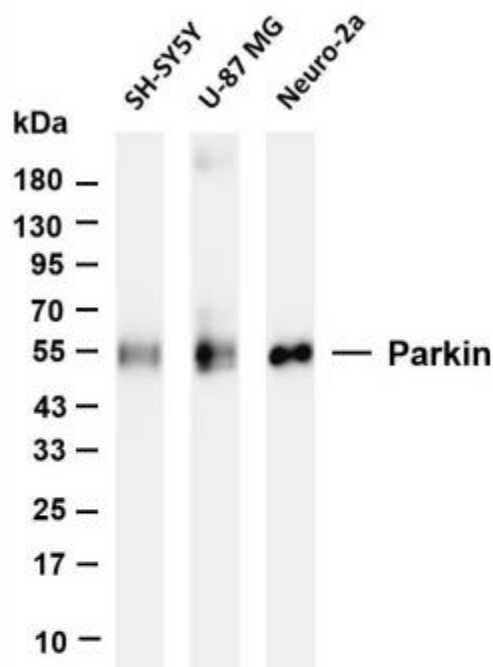


关注客服

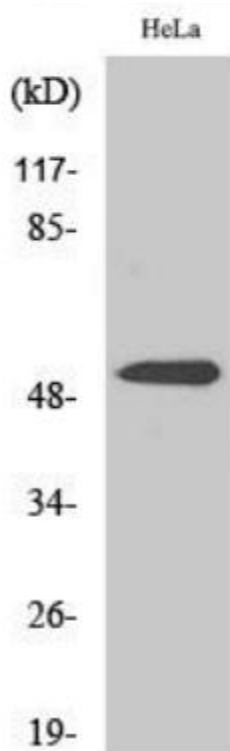


Usage suggestions

This product can be used in immunological reaction related experiments. For more information, please consult technical personnel.

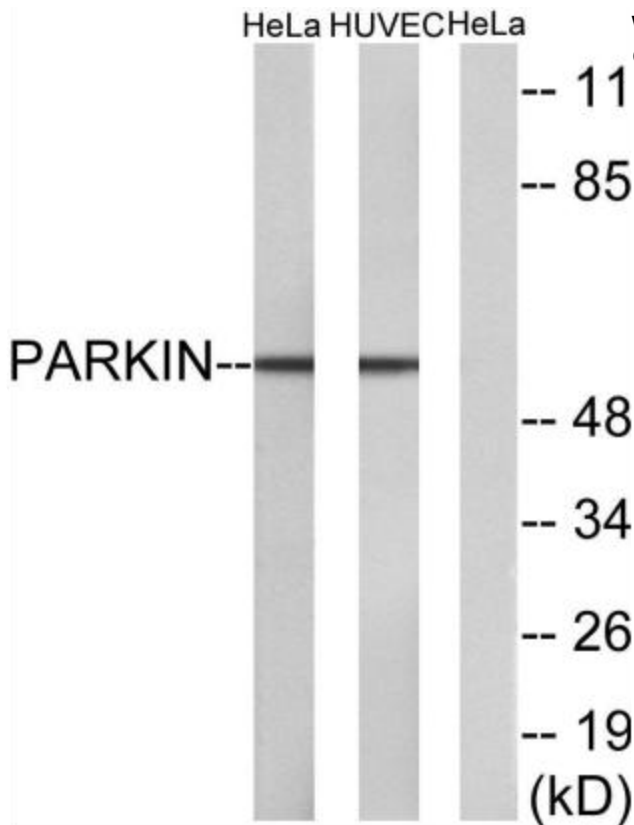


Various whole cell lysates were separated by 4-20% SDS-PAGE, and the membrane was blotted with anti-Parkin antibody. The HRP-conjugated Goat anti-Rabbit IgG(H + L) antibody was used to detect the antibody. Lane 1: SH-SY5Y Lane 2: U-87 MG Lane 3: Neuro-2a Predicted band size: 52kDa Observed band size: 52kDa

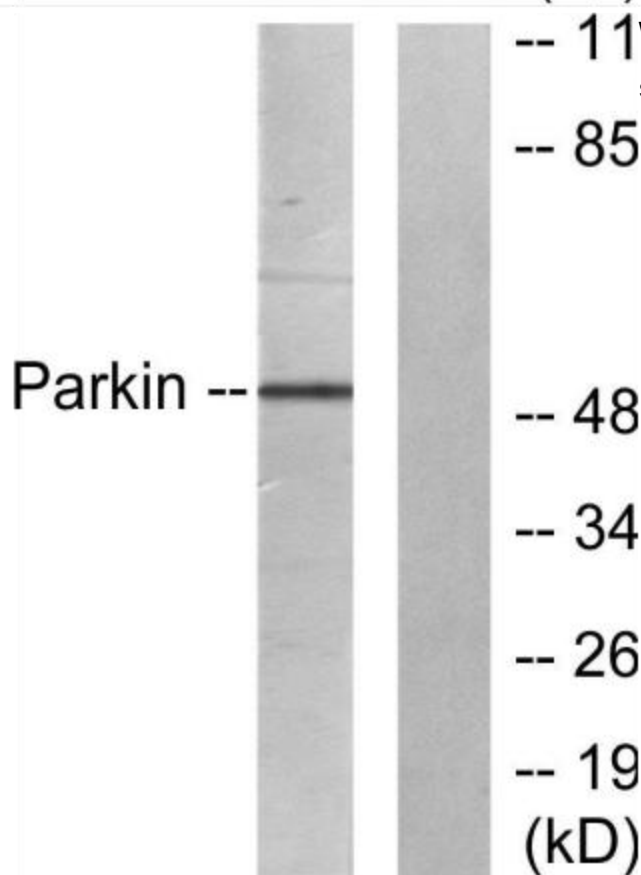


Western Blot analysis of various cells using Parkin Antibody





Western blot analysis of lysates from HeLa and HUVEC cells, using Parkin Antibody. The lane on the right is blocked with the synthesized peptide.



Western blot analysis of lysates from Jurkat cells, using Parkin Antibody. The lane on the right is blocked with the synthesized peptide.

