abcam

Product datasheet

Anti-Parkin (phospho S378) antibody ab73016

1 References 1 Image

Overview

Product name	Anti-Parkin (phospho S378) antibody
Description	Rabbit polyclonal to Parkin (phospho S378)
Host species	Rabbit
Specificity	ab73016 is specific for the ~52k parkin protein phosphorylated at Ser378. Immunolabeling of the parkin band is absent in parkin S378 mutants.
Tested applications	**Suitable for:** WB
Species reactivity	**Reacts with:** Human
Immunogen	Synthetic peptide corresponding to Human Parkin (phospho S378).
Positive control	HEK293 cells transfected with Parkin Wild type (WT).
General notes	Recent evidence suggests that phosphorylation of parkin at Ser378 may have an important regulatory role on its E3 ubiquitin ligase activity (Yamamoto et al., 2005). The Life Science industry has been in the grips of a reproducibility crisis for a number of years. Abcam is leading the way in addressing this with our range of recombinant monoclonal antibodies and knockout edited cell lines for gold-standard validation. Please check that this product meets your needs before purchasing. If you have any questions, special requirements or concerns, please send us an inquiry and/or contact our Support team ahead of purchase. Recommended alternatives for this product can be found below, along with publications, customer reviews and Q&As

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles.
Storage buffer	pH: 7.50 Constituents: 0.01% BSA, 50% Glycerol, 0.87% Sodium chloride, 0.238% HEPES
Purity	Immunogen affinity purified
Purification notes	ab73016 is prepared from rabbit serum by affinity purification via sequential chromatography on phospho- and dephosphopeptide affinity columns.
Primary antibody notes	Recent evidence suggests that phosphorylation of parkin at Ser378 may have an important

regulatory role on its E3 ubiquitin ligase activity (Yamamoto et al., 2005).

Clonality Polyclonal

Isotype IgG

Applications

The Abpromise guarantee Our **Abpromise guarantee** covers the use of ab73016 in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
WB		1/1000. Detects a band of approximately 52 kDa (predicted molecular weight: 52 kDa).

Target

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 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.

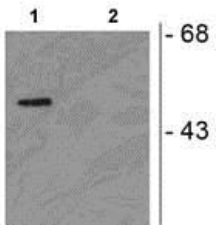
Tissue specificity Highly expressed in the brain including the substantia nigra. Expressed in heart, testis and skeletal muscle. Expression is down-regulated or absent in tumor biopsies, and absent in the brain of PARK2 patients. Overexpression protects dopamine neurons from kainate-mediated apoptosis.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

Pathway Protein modification; protein ubiquitination.

Involvement in 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 (intraneuronal 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 befor 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 (intraneuronal accumulations of aggregated proteins) are absent. Note=Defects in PARK2 may be involved in the development and/or progression of ovarian cancer.
Sequence similarities	Belongs to the RBR family. Parkin subfamily. Contains 1 IBR-type zinc finger. Contains 2 RING-type zinc fingers. Contains 1 ubiquitin-like domain.
Domain	The ubiquitin-like domain binds the PSMD4 subunit of 26S proteasomes.
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.
Cellular localization	Cytoplasm > cytosol. Nucleus. Endoplasmic reticulum. Mitochondrion. Mainly localizes in the cytosol. Co-localizes with SYT11 in neutrites. Co-localizes with SNCAIP in brainstem Lewy bodies. Relocates to dysfunctional mitochondria that have lost the mitochondial membrane potential; recruitement to mitochondria is PINK1-dependent.

Images



Western blot - Anti-Parkin (phospho S378) antibody (ab73016)

All lanes : Anti-Parkin (phospho S378) antibody (ab73016) at 1/1000 dilution

Lane 1 : HEK293 cells transfected with Parkin WT (phospho)
Lane 2 : HEK293 cells transfected with Parkin S378 mutant (non-phospho)

Predicted band size: 52 kDa
Observed band size: 52 kDa

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