


# Anti-Parkin (phospho S101) antibody ab73015

**2 References**   **1 图像**

### 概述

产品名称	Anti-Parkin (phospho S101)抗体
描述	兔多克隆抗体to Parkin (phospho S101)
宿主	Rabbit
特异性	ab73015 is specific for the ~52k parkin protein phosphorylated at Ser101. Immunolabeling of the parkin band is absent in parkin S101 mutants.
经测试应用	<b>适用于:</b> WB
种属反应性	<b>与反应:</b> Human <b>预测可用于:</b> Cow, Non human primates 
免疫原	Synthetic peptide corresponding to Human Parkin (phospho S101).
阳性对照	HEK293 cells transfected with Parkin WT.
常规说明	

Recent evidence suggests that phosphorylation of parkin at Ser101 may have an important regulatory role on its E3 ubiquitin ligase activity (Yamamoto et al., 2005).

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### 性能

形式	Liquid
存放说明	Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles.
存储溶液	pH: 7.50 Constituents: 0.01% BSA, 50% Glycerol, 0.87% Sodium chloride, 0.238% HEPES
纯度	Immunogen affinity purified
纯化说明	ab73015 is prepared from rabbit serum by affinity purification via sequential chromatography on phospho- and dephosphopeptide affinity columns.

Primary antibody说明	Recent evidence suggests that phosphorylation of parkin at Ser101 may have an important regulatory role on its E3 ubiquitin ligase activity (Yamamoto et al., 2005).
克隆	多克隆
同种型	IgG

应用

The Abpromise guarantee      **Abpromise™**承诺保证使用ab73015于以下的经测试应用

“应用说明”部分 下显示的仅为推荐的起始稀释度;实际最佳的稀释度/浓度应由使用者检定。

应用	Ab评论	说明
WB		1/1000. Detects a band of approximately 52 kDa (predicted molecular weight: 52 kDa).

靶标

功能	<p>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.</p>
组织特异性	<p>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).</p>
通路	<p>Protein modification; protein ubiquitination.</p>
疾病相关	<p>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</p>

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 (intraneuronal accumulations of aggregated proteins) are absent.

Note=Defects in PARK2 may be involved in the development and/or progression of ovarian cancer.

#### 序列相似性

Belongs to the RBR family. Parkin subfamily.

Contains 1 IBR-type zinc finger.

Contains 2 RING-type zinc fingers.

Contains 1 ubiquitin-like domain.

#### 结构域

The ubiquitin-like domain binds the PSMD4 subunit of 26S proteasomes.

#### 翻译后修饰

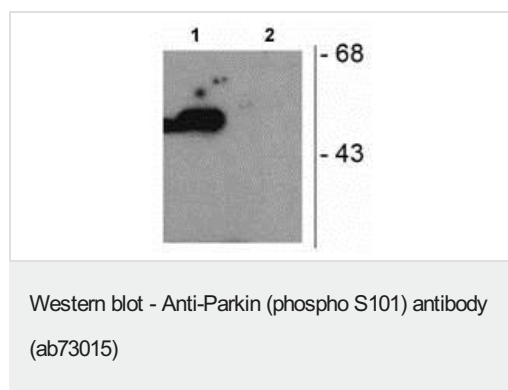
Auto-ubiquitinates in an E2-dependent manner leading to its own degradation. Also polyubiquitinated by RNF41 for proteasomal degradation.

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.

#### 细胞定位

Cytoplasm > cytosol. Nucleus. Endoplasmic reticulum. Mitochondrion. 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.

#### 图片



**All lanes :** Anti-Parkin (phospho S101) antibody (ab73015) at 1/1000 dilution

**Lane 1 :** HEK293 cells transfected with Parkin WT (phospho)

**Lane 2 :** HEK293 cells transfected with Parkin S101 mutant (non-phospho)

**Predicted band size:** 52 kDa

**Observed band size:** 52 kDa

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