# abcam

## Product datasheet

# Anti-Parkin antibody ab15494

5 References 画像数 1

製品の概要

製品名 Anti-Parkin antibody

製品の詳細 Rabbit polyclonal to Parkin

由来種 Rabbit

アプリケーション 適用あり: ICC/IF, IHC-P

種交差性 交差種: Human

交差が予測される動物種: Mouse, Rat 🔷

免疫原 Synthetic peptide within Human Parkin aa 50-150 (N terminal). The exact sequence is proprietary.

ポジティブ・コントロール Recombinant Human Parkin protein (ab114219) can be used as a positive control in WB. IHC-P:

Human brain tissue.

特記事項 This product is FOR RESEARCH USE ONLY. For commercial use, please contact

partnerships@abcam.com.

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.

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found below, along with publications, customer reviews and Q&As

製品の特性

製品の状態 Liquid

保存方法 Shipped at 4°C. Upon delivery aliquot and store at -20°C. Avoid freeze / thaw cycles.

**バッファー** pH: 7.60

Preservative: 0.1% Sodium azide Constituents: PBS. 1% BSA

精製度 Immunogen affinity purified

**ポリ/モノ** ポリクローナル

アイソタイプ IgG

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アプリケーション	Abreviews	特記事項
ICC/IF		Use at an assay dependent concentration.
IHC-P		1/30. Perform heat mediated antigen retrieval before commencing with IHC staining protocol.

#### ターゲット情報

#### 機能

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.

#### 組織特異性

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).

#### パスウェイ

Protein modification; protein ubiquitination.

# 関連疾患

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.

**配列類似性** Belongs to the RBR family. Parkin subfamily.

Contains 1 IBR-type zinc finger. Contains 2 RING-type zinc fingers. Contains 1 ubiquitin-like domain.

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

翻訳後修飾 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  $\,$ 

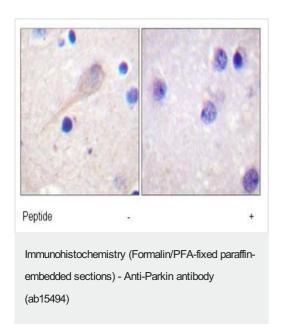
substrates.

**細胞内局在** 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.

#### 画像



ab15494 staining Parkin in Alzheimers brain by Immunohistochemistry (FFPE-sections).

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