Antibody Details:

**Product name:** Anti-Parkin antibody

**Description:** Rabbit polyclonal to Parkin

**Host species:** Rabbit

**Tested applications:** Suitable for: WB, IHC-P

**Species reactivity:** Reacts with: Human, Pig

**Immunogen:** Recombinant fragment (His-T7-tag) corresponding to Human Parkin aa 229-465. (Expressed in E.coli).

**Sequence:**

```
IATNSRNITCTCDVRSPVLFQCNSRHVICLDCFHYLC
VTRLNDRQFV
HDPQLGYSLPCVAGCPNSLIKELHHFRILGEEQYNRYQ
QYGAEETVLQMG
GVLCPRPCAGLLEPEPDQRKVTCCEGGNGLGCGFAF
CRECEAYHEGEC
AVFEASGTTTQAYRVDERAQARWEAASKETIKKT
KPCPRCHVPEK
GGCMHMKPQPCPQCRLEWCWNCGCEWNRCVCMGDHWFDV
```

**Database link:** O60260

**Positive control:**

WB: HEK-293T cell lysate; Pig brain lysate; Recombinant human Parkin protein. IHC-P: Human kidney tissue.

**Form:** Liquid

**Storage instructions:** Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C long term. Avoid freeze / thaw cycle.

**Storage buffer:**

- **pH:** 7.40
- **Preservative:** 0.02% Sodium azide
- **Constituents:** PBS, 50% Glycerol
Purity
Immunogen affinity purified

Purification notes
ab233434 was purified by antigen-specific affinity chromatography followed by Protein A affinity chromatography.

Clonality
Polyclonal

Isotype
IgG

Applications
Our Abpromise guarantee covers the use of ab233434 in the following tested applications.
The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

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<th>Application</th>
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<td>WB</td>
<td></td>
<td>Use a concentration of 0.5 - 2 µg/ml. Predicted molecular weight: 52 kDa.</td>
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<tr>
<td>IHC-P</td>
<td></td>
<td>Use a concentration of 5 - 20 µg/ml.</td>
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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 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 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.

**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-ubiquitinites 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**

**Images**
Formalin-fixed, paraffin-embedded human kidney tissue stained for Parkin using ab233434 at 20 µg/ml in immunohistochemical analysis. DAB staining.

**Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) - Anti-Parkin antibody (ab233434)**
All lanes: Anti-Parkin antibody (ab233434) at 2 µg/ml

Lane 1: HEK-293T (Human epithelial cell line from embryonic kidney transformed with large T antigen) cell lysate

Lane 2: Pig brain lysate

Predicted band size: 52 kDa

Anti-Parkin antibody (ab233434) at 2 µg/ml + Recombinant human Parkin protein

Predicted band size: 52 kDa

Please note: All products are “FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES”

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