abcam

Product datasheet

Recombinant Human Parkin protein (denatured) ab177625

1 Image

Description

Product name	Recombinant Human Parkin protein (denatured)
Purity	> 80 % SDS-PAGE.
Expression system	Escherichia coli
Accession	O60260
Protein length	Full length protein
Animal free	No
Nature	Recombinant
Species	Human
Sequence	MGSSHHHHHHSSGLVPRGSHMIVFVRFNSSHGFPVEVDS DTSIFQLKEVV AKRQGVPADQLRVIFAGKELRNDWTVQNCDLDQQSIVHIV QRPWRKGQEM NATGGDDPRNAAGGCEREPQSLTRVDLSSSVLPGDSVG LAVILHTDSRKD SPPAGSPAGRSIYNSFYVYCKGPCQRVQPGKLRVQCSTC RQATLTLTQGP SCWDDVLIPNRMSGECQSPHCPGTSAEFFFKCGAHPTS DKETSVALHLIA TNSRNITCITCTDVRSPVLVFQCNSRHVICLDCFHLYCVTRL NDRQFVHD PQLGYSLPCVAGCPNSLIKELHHFRILGEEQYNRYQQYGA EECVLQMGGV LCPRPGCGAGLLPEPDQRKVTCEGGNGLGCGFAFCREC KEAYHEGECSAV FEASGTTTQAYRVDERAAEQARWEAASKETIKKTTKPCP RCHVPVEKNGG CMHMKCPQPQCRLEWCWNCGCEWNRVCMGDHWFDV
Predicted molecular weight	54 kDa including tags
Amino acids	1 to 465
Tags	His tag N-Terminus

Additional sequence information	NP_004553.2
Description	Recombinant Human Parkin protein

Specifications

Our **Abpromise guarantee** covers the use of **ab177625** in the following tested applications.

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

Applications	SDS-PAGE
Form	Liquid

Preparation and Storage

Stability and Storage	Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C or -80°C. Avoid freeze / thaw cycle. pH: 8.00 Constituents: 0.32% Tris-HCl buffer, 10% Glycerol (glycerin, glycerine), 2.4% Urea

General Info

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.

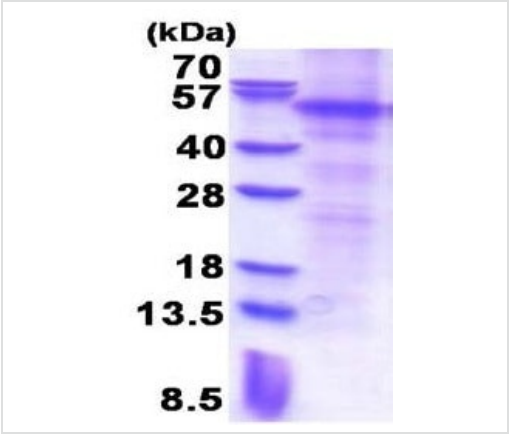
Post-translational modifications

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



SDS-PAGE - Recombinant Human Parkin protein (denatured) (ab177625)

15% SDS-PAGE analysis of ab177625 (3 µg).

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

Our Abpromise to you: Quality guaranteed and expert technical support

- Replacement or refund for products not performing as stated on the datasheet
- Valid for 12 months from date of delivery
- Response to your inquiry within 24 hours
- We provide support in Chinese, English, French, German, Japanese and Spanish
- Extensive multi-media technical resources to help you
- We investigate all quality concerns to ensure our products perform to the highest standards

If the product does not perform as described on this datasheet, we will offer a refund or replacement. For full details of the Abpromise, please visit **https://www.abcam.com/abpromise** or contact our technical team.

Terms and conditions

- Guarantee only valid for products bought direct from Abcam or one of our authorized distributors