abcam

Product datasheet

Parkin overexpression 293T lysate (whole cell) ab94077

画像数 2

医薬用外劇物

製品の概要

製品名	Parkin overexpression 293T lysate (whole cell)
特記事項	ab94077 is a 293T cell transfected lysate in which Human Parkin has been transiently over-expressed using a pCMV-Parkin plasmid. The lysate is provided in 1X Sample Buffer.
アプリケーション	**適用あり:** WB

製品の特性

Mycoplasma free	Yes
製品の状態	Liquid
保存方法	Shipped on dry ice. Upon delivery aliquot and store at -20ºC. Avoid freeze / thaw cycles.
バッファー	Constituents: 0.01% Bromophenol blue, 2.3% Beta mercaptoethanol, 2% Sodium lauryl sulfate, 0.788% Tris HCl, 10% Glycerol (glycerin, glycerine)
背景	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. 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. Similarity: 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. PTM: 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.

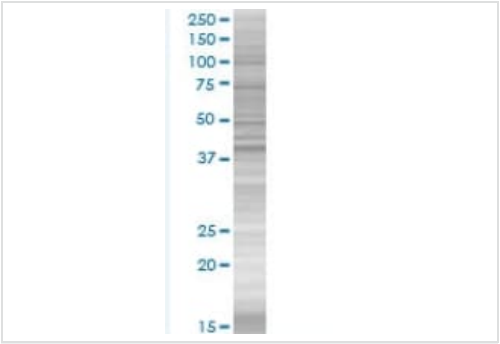
アプリケーション

The Abpromise guarantee **Abpromise保証は、次のテスト済みアプリケーションにおけるab94077の使用に適用されます**

アプリケーションノートには、推奨の開始希釈率がありますが、適切な希釈率につきましてはご検討ください。

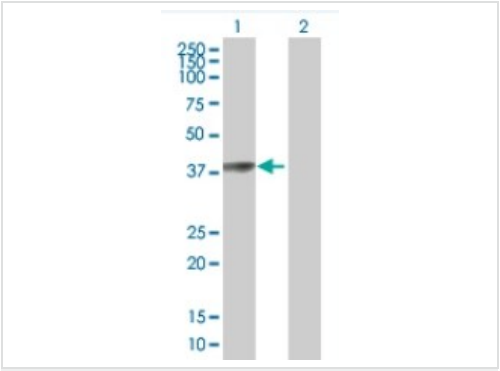
アプリケーション	Abreviews	特記事項
WB		Use at an assay dependent dilution.

画像



SDS-PAGE - Parkin overexpression 293T lysate (whole cell) (ab94077)

ab94077 at 15µg/lane on an SDS-PAGE gel.



Western blot - Parkin overexpression 293T lysate (whole cell) (ab94077)

All lanes : Anti-Parkin antibody (**ab55488**) at 1/500 dilution

Lane 1 : Parkin 293T Transfected Lysate - Positive Control (ab94077)
Lane 2 : 293T non-transfected lysate

Lysates/proteins at 25 µg per lane.

Secondary
All lanes : Goat Anti-mouse IgG (H and L) HRP conjugated at 1/2500 dilution

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