p62 (human) polyclonal antibody

P62, also known as Sequestosome I, is a 62kDa, 440 amino acid protein, initially identified as a ligand of the SH2 domain of p56lck, now known to be expressed in many tissues. In addition to TRAF6, PEST and zinc finger motifs, p62 has a C-terminal ubiquitin binding association (UBA) domain with an affinity for multi-ubiquitin chains, and it is considered to serve as a scaffold protein, capable of binding to multiple signalling molecules and uniting receptor-mediated signalling events with ubiquitinylation. Elevated levels of p62 have been reported in breast tumours and in alcoholic liver disease where p62 has been shown to be involved in the formation of Mallory bodies. Several mutations in the p62 UBA domain have been identified and the etiology of Paget's disease of bone has been linked to one such mutation. Kuusisto and colleagues have demonstrated that p62 is also present in elevated levels in the hallmark inclusions found in various neurodegenerative conditions, including tauopathies (Alzheimer's disease, Picks disease, and frontotemporal dementia) and synucleinopathies (Parkinson's disease, dementia with Lewy body disease and multiple system atrophy). In recent years ubiquitin immunostaining has been used to provide adjunct information for neuropathological diagnosis, but it is becoming evident that p62 may be an even more reliable marker of neurodegenerative disease inclusion detection than tau, alpha-synuclein or ubiquitin immunostaining.

This antibody is covered by our Worry-Free Guarantee.

Citations: 176 View Online »

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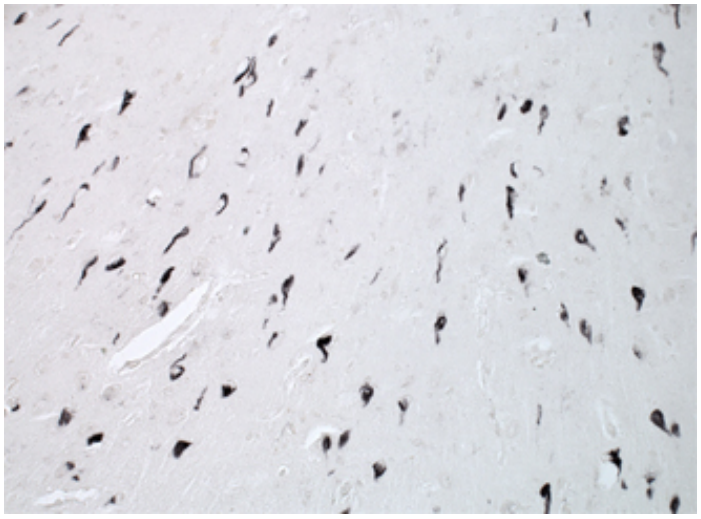
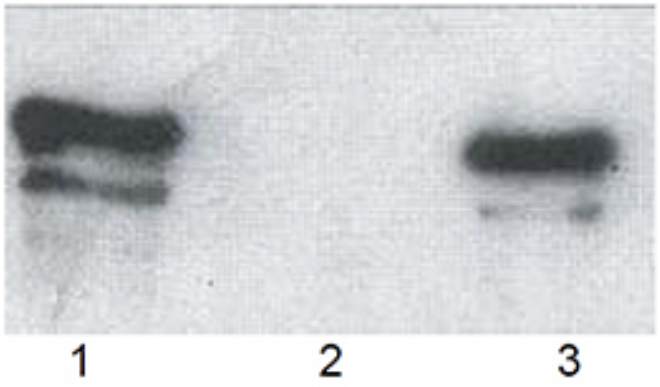
BML-PW9860-0025	25µl
BML-PW9860-0100	100µl

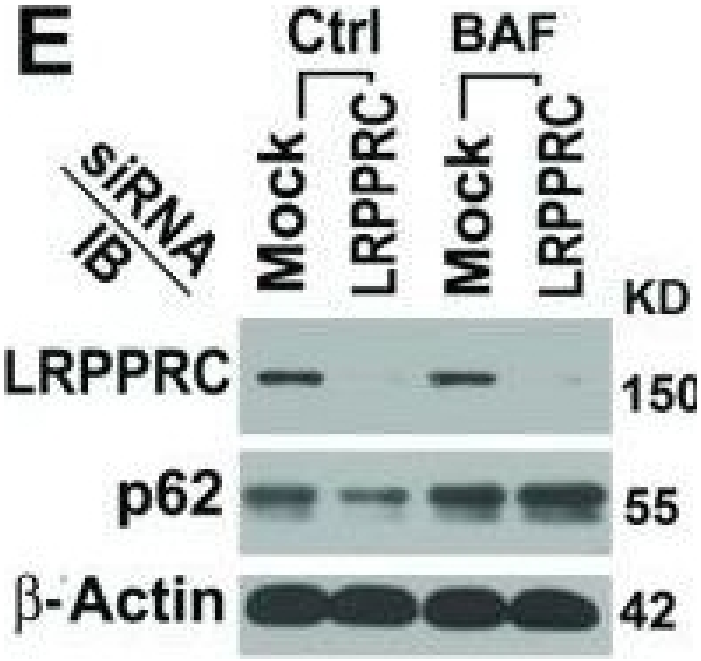
Manuals, SDS & CofA View Online »



Immunohistochemistry analysis of p62 immunoreactivity is present in neurons of the hippocampus of an Alzheimer patient. Note the intense reaction in the neurofibrillary tangles (dilution 1:1000).
Micrograph courtesy of Professor Fred van Leeuwen, University of Maastricht, The Netherlands.

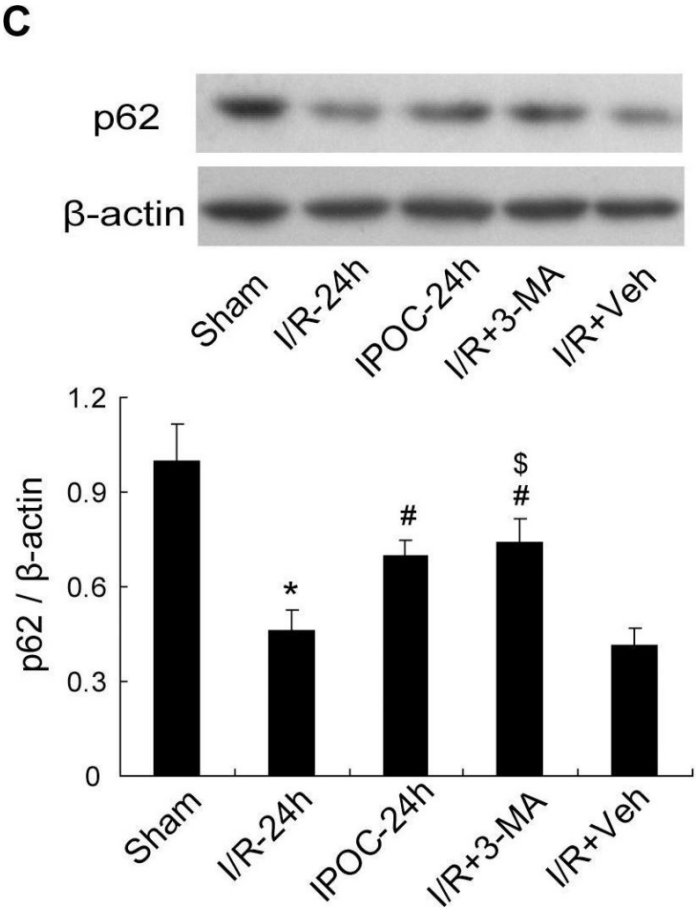


Western blot analysis of wild type p62, GST-tagged (human, recombinant) bound to: (1) glutathione-agarose and (3) ubiquitin-agarose (2: agarose control). Bound species were analysed by PAGE followed by blotting on to PVDF and probing with Prod. No. BML-PW9860.



Suppression of LRPPRC leads to enhancement of basal levels of autophagy in native HeLa cells(A) Immunoblot analyses of LC3 isoforms in lysates from HeLa cells treated with random siRNA (Mock) or LRPPRC-specific siRNA (LRPPRC) in the absence (Ctrl) or presence of lysosomal inhibitor NH4Cl. Molecular masses are indicated in kDa. (B) Plots of relative intensities of LC3-I and LC3-II bands. The LC3-I and LC3-II intensities in samples treated with mock siRNA were set to 1. Results are means±S.D. of at least three repeats and the differences were compared using a paired Student's t test. *P≤0.05. (C) Transmission electron microscopy imaging of HeLa cells treated with random siRNA (Mock) or LRPPRC-specific siRNA (LRPPRC) in the absence (Ctrl) or presence (BAF) of lysosomal inhibitor bafilomycin A1. *, autophagy vacuoles. (D) Plot of percentages of area occupied by autophagy vacuoles in the transmission electron microscopy images. Results are means±S.D. of at least three repeats and the differences were compared using Student's t test. *P≤0.05. (E) Immunoblot (IB) analyses of p62 levels in lysates from HeLa cells treated with random siRNA (Mock) or LRPPRC-specific siRNA (LRPPRC) in the absence (Ctrl) or presence (BAF) of bafilomycin A1. (F) Plots of relative intensities of p62. The p62 intensities in samples treated with mock siRNA were set to 1. Results are means±S.D. of at least three repeats and the differences were compared using a paired Student's t test. *P≤0.05. (G) Immunostaining analysis of p62 levels in HeLa cells treated with random siRNA (Mock) or LRPPRC-specific siRNA (LRPPRC) in the absence (Ctrl) or presence (BAF) of bafilomycin A1.

Image collected and cropped by CiteAb under a CC-BY license from the following publication: Mitochondrion-associated protein LRPPRC suppresses the initiation of



The changes of expressions of LC3, Beclin 1, p62 and Bcl-2 after 3-MA treatment.(A–C) The changes of LC3, Beclin 1 and p62 after the treatment of 3-MA. 3-MA downregulated the ratio of LC3-II/LC3-I and the expression of Beclin 1, and upregulated the expression of p62 as compared with that in I/R-24 h or I/R+Veh group. (D) The expression of Bcl-2 after the treatment of 3-MA. The expression of Bcl-2 decreased at 24 h postischemia, however, IPOC or treatment with 3-MA partially resulted in the recovery of Bcl-2. Levels of β-actin protein were used as the loading control. n = 5 for each group. *p<0.05 vs. the Sham group; #p<0.05 vs. I/R-24 h group; $p<0.05 vs. I/R+Veh group.

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Handling & Storage

Handling	Avoid freeze/thaw cycles. After opening, prepare aliquots and store at -20°C.
Long Term Storage	-20°C
Shipping	Blue Ice

Regulatory Status

RUO - Research Use Only

Product Details

Alternative Name	SQSTM1, Sequestosome I
Application	ELISA, IF, IHC, WB
Formulation	Liquid. In PBS containing 0.09% sodium azide.
Host	Rabbit
Immunogen	Synthetic peptide corresponding to aa 387-436 human p62.
Purity Detail	Peptide affinity purified.
Recommendation Dilutions/Conditions	Immunohistochemistry (1:1,000)Western Blot (1:1,000)Suggested dilutions/conditions may not be available for all applications.Optimal conditions must be determined individually for each application.
Source	Purified from rabbit serum.
Species Reactivity	Human
Technical Info / Product Notes	**Cited samples:** *For an overview on cited samples please click here.*
UniProt ID	Q13501
Worry-free Guarantee	**This antibody is covered by our Worry-Free Guarantee.**

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