

Product Datasheet

p62/SQSTM1 Antibody (2C11) - Azide and BSA Free H00008878-M01

Unit Size: 0.1 mg

Aliquot and store at -20C or -80C. Avoid freeze-thaw cycles.

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H00008878-M01

p62/SQSTM1 Antibody (2C11) - Azide and BSA Free

Product Information	
Unit Size	0.1 mg
Concentration	Concentrations vary lot to lot. See vial label for concentration. If unlisted please contact technical services.
Storage	Aliquot and store at -20C or -80C. Avoid freeze-thaw cycles.
Clonality	Monoclonal
Clone	2C11
Preservative	No Preservative
Isotype	IgG2a Kappa
Purity	IgG purified
Buffer	In 1x PBS, pH 7.4
Target Molecular Weight	47.7 kDa

Product Description	
Description	Quality control test: Antibody Reactive Against Recombinant Protein.
Host	Mouse
Gene ID	8878
Gene Symbol	SQSTM1
Species	Human, Mouse, Rat, Bovine, Hamster, Rabbit
Reactivity Notes	Rat reactivity reported in scientific literature (PMID: 24352657). Human reactivity reported in scientific literature (PMID: 26571504). Rabbit reactivity reported in scientific literature (PMID: 23569437). Please note that this antibody is reactive to Mouse and derived from the same host, Mouse. Mouse-On-Mouse blocking reagent may be needed for IHC and ICC experiments to reduce high background signal. You can find these reagents under catalog numbers PK-2200-NB and MP-2400-NB. Please contact Technical Support if you have any questions.
Immunogen	SQSTM1 (AAH03139.1, 1 a.a. ~ 440 a.a) full-length recombinant protein with GST tag. MW of the GST tag alone is 26 KDa. MASLTVKAYLLGKEDAAREIRRFSCCSPEPEAEAEAAAGPGPCERLLSRVAAL FPALRPGGFQAHYRDEDGDLVAFSSDEELTMAMSYVKDDIFRIYIKEKKECRRD HRPPCAQEAPRNMVHPNVICDGCNGPVVGTRYKCSVCPDYDLCVCEGKGL HRGHTKLAFPSPFGHLSEGFSSRWRKVKHGHFGWPGWEMGPPGNWSPR PPRAGEARPGPTAESASGPSSEDPVSNFLKNVGESVAAALSPLGIEVDIDVEHG GKR SRLTPVSPSSSTEEKSSSQPSSCCSDPSKPGGNVEGATQSLAEQMRKI ALESEGRPEEQMESDNCSSGGDDWTHLSSKEVDPSTGELQSLQMPESGPS SLDPSQEGPTGLKEAALYPHLPPEADPR LIESLSQMLSMGFSDDEGGWLTRLLQ TKNYDIGAALDTIQYSKHPPPL
Notes	This product is produced by and distributed for Abnova, a company based in Taiwan.

Product Application Details	
Applications	Western Blot, ELISA, Immunocytochemistry/ Immunofluorescence, Immunohistochemistry, Immunohistochemistry-Frozen, Immunohistochemistry-Paraffin, Immunoprecipitation
Recommended Dilutions	Western Blot 1:100-1:2000, ELISA, Immunohistochemistry 1:10-1:500, Immunocytochemistry/ Immunofluorescence 10ug/mL, Immunoprecipitation 1:10-1:1000, Immunohistochemistry-Paraffin 1:10-1:500, Immunohistochemistry-Frozen 1:10-1:500



Application Notes

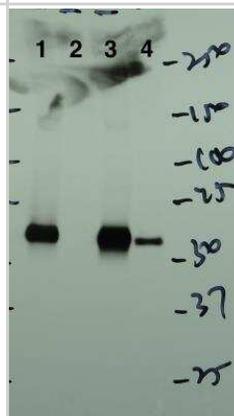
Use in Immunohistochemistry-paraffin reported in scientific literature (PMID: 23569437).

Images

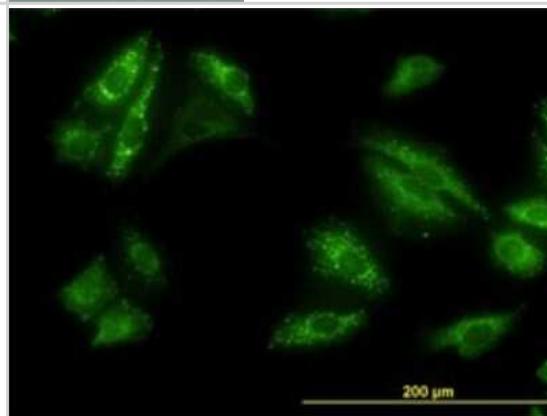
Western Blot: p62/SQSTM1 Antibody (2C11) [H00008878-M01] - WB analysis of recombinant p62 protein (immunogen, 74.51 KDa) using SQSTM1/p62 antibody (clone 2C11).



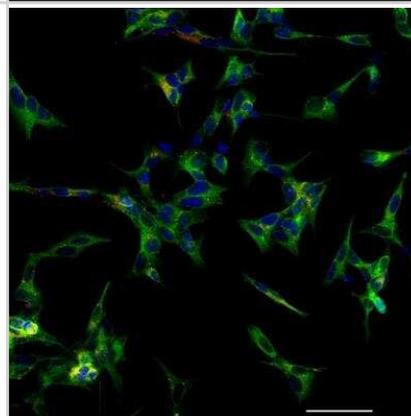
Western Blot: p62/SQSTM1 Antibody (2C11) [H00008878-M01] - WB analysis of p62 in p62 WT and KO mouse hepatocytes and HepG2 cell lysate using anti-p62/SQSTM1 antibody clone 2C11. Sample lane1: p62 +/- mouse hepatocytes, lane 2: p62 -/- mouse hepatocytes, lane3: Wt mouse hepatocytes, lane 4: HepG2 cells. This image is from a verified customer product review.



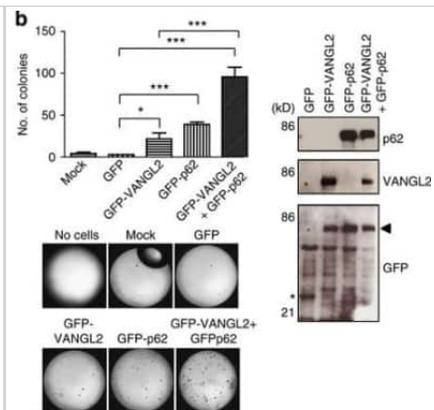
Immunocytochemistry/Immunofluorescence: p62/SQSTM1 Antibody (2C11) [H00008878-M01] - ICC-IF analysis of SQSTM1 protein in HeLa cell using SQSTM1/p62 antibody (clone 2C11) at 10 ug/ml concentration.



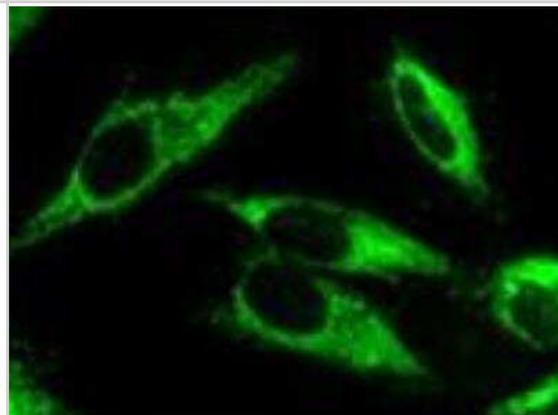
Immunocytochemistry/Immunofluorescence: p62/SQSTM1 Antibody (2C11) [H00008878-M01] - Analysis of p62 in SH-SY5Y cells using anti-p62/SQSTM1 antibody. Red - p62 puncta; Blue - nuclear DAPI; Green - Cytoskeleton. Image from verified customer review.



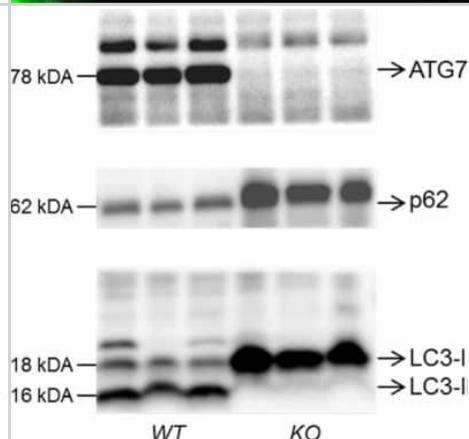
Western Blot: p62/SQSTM1 Antibody (2C11) [H00008878-M01] - Disruption of the VANGL2-p62/SQSTM1 interaction in breast cancer cells. Soft agar colony formation of T47D cells overexpressing GFP, GFP-VANGL2 and GFP-p62/SQSTM1 (right). Protein expression was revealed with anti-p62/SQSTM1, anti-VANGL2 and anti-GFP antibodies by western blot analysis (right). In anti-GFP blot, the arrowhead indicates position of co-migrating GFP-VANGL2 and GFP-p62/SQSTM1 and the asterisk pinpoints GFP alone. Error bars represent mean \pm s.d. (n=3). Image collected and cropped by CiteAb from the following publication (<https://www.nature.com/doi/10.1038/ncomms10318>), licensed under a CC-BY license.



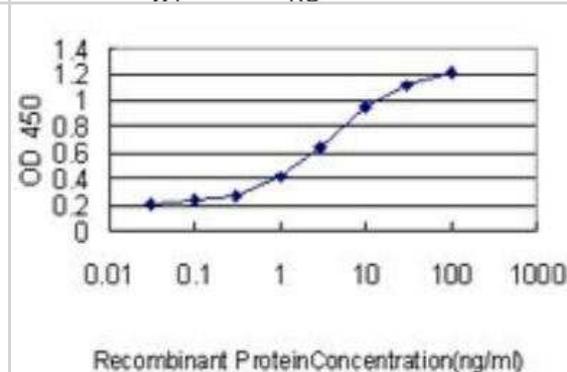
Immunocytochemistry/Immunofluorescence: p62/SQSTM1 Antibody (2C11) [H00008878-M01] - Analysis of p62/SQSTM1 antibody on HeLa cells. Image from verified customer review.



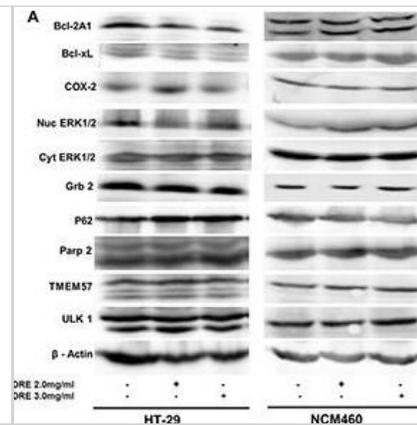
Western Blot: p62/SQSTM1 Antibody (2C11) [H00008878-M01] - Confirmation of loss of hepatic ATG7 protein and a phenotype of hepatic autophagy inactivation. Fourth ug of liver protein were loaded on 4-20% gradient Tris-Glycine gels and subsequently immunoblotted with antibodies against ATG7, p62 and LC3. Blots were visualized with G:BOX Chemi XRQ (SynGene). Representative immunoblots are shown for WT and KO mice. Full-length immunoblots are presented in Supplementary Figure S1. ATG7: autophagy-related protein 7; p62: sequestosome 1; LC3: microtubule-associated protein 1 light chain 3; WT: Mx1-Cre-Atg7F/F mouse; KO: Mx1-Cre+Atg7F/F mouse. Image collected and cropped by CiteAb from the following publication (<https://www.nature.com/articles/s41598-017-14405-w>), licensed under a CC-BY license.



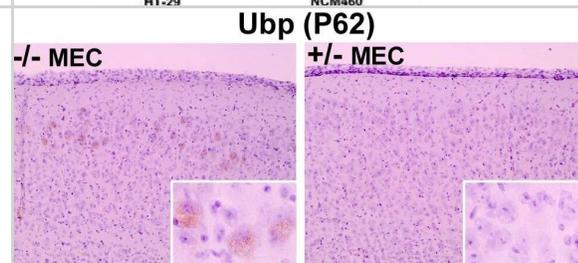
ELISA: p62/SQSTM1 Antibody (2C11) [H00008878-M01] - Detection limit for recombinant GST tagged SQSTM1 is approximately 0.03ng/ml as a capture antibody.



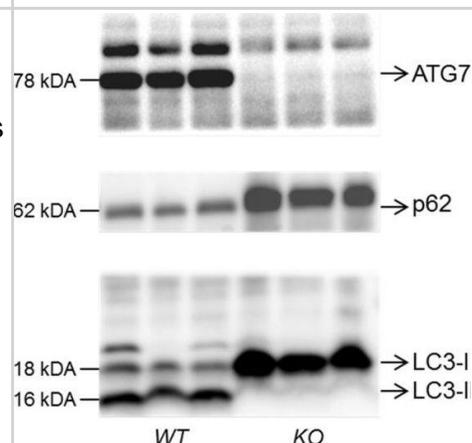
Western Blot: p62/SQSTM1 Antibody (2C11) [H00008878-M01] - Activation of multiple signaling pathways by dandelion root extractA. Western blots of proteins involved in programmed cell death & cell survival & inflammation B. Densitometry quantification of western blot analysis from three independent experiments. *P<0.05, **P< 0.001. Image collected & cropped by CiteAb from the following publication (<https://www.oncotarget.com/lookup/doi/10.18632/oncotarget.11485>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



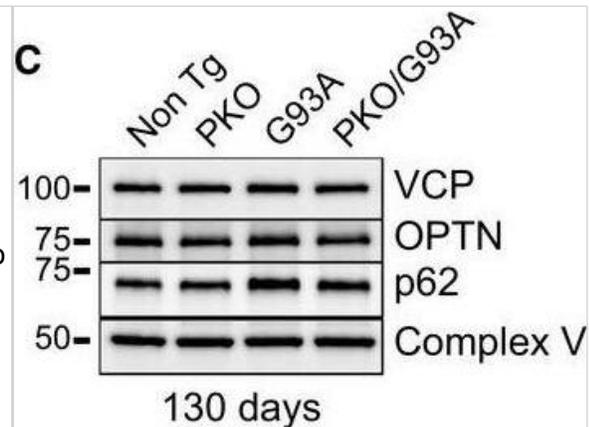
Immunohistochemistry: p62/SQSTM1 Antibody (2C11) [H00008878-M01] - Elevated levels of additional proteins & GM3 ganglioside in the MEC of MPS IIIB brain. Staining performed with antibodies to the indicated substances was observed in the MEC region of 3 month-old MPS IIIB mice (for total ubiquitin & polyubiquitin) & 6 months for all others. Staining was not seen in the MEC region of age-matched control mice (Naglu +/-) nor in the LEC region of MPS IIIB mice (the latter not shown). Image collected & cropped by CiteAb from the following publication (<https://dx.plos.org/10.1371/journal.pone.0027461>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



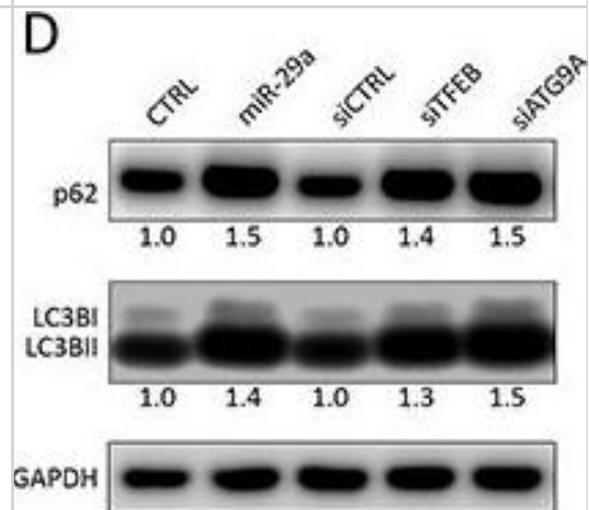
Western Blot: p62/SQSTM1 Antibody (2C11) [H00008878-M01] - Confirmation of loss of hepatic ATG7 protein & a phenotype of hepatic autophagy inactivation. Forty µg of liver protein were loaded on 4–20% gradient Tris-Glycine gels & subsequently immunoblotted with antibodies against ATG7, p62 & LC3. Blots were visualised with G:BOX Chemi XRQ (SynGene). Representative immunoblots are shown for WT & KO mice. Full-length immunoblots are presented in Supplementary Figure S1. ATG7: autophagy-related protein 7; p62: sequestosome 1; LC3: microtubule-associated protein 1 light chain 3; WT: Mx1-Cre-Atg7F/F mouse; KO: Mx1-Cre+Atg7F/F mouse. Image collected & cropped by CiteAb from the following publication (<https://pubmed.ncbi.nlm.nih.gov/29074879>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



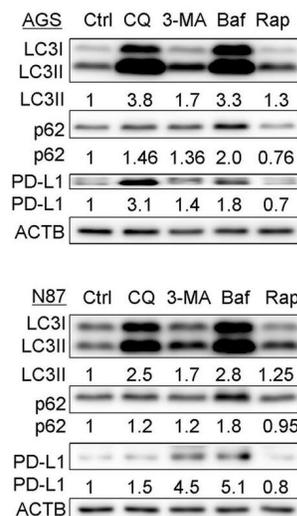
Western Blot: p62/SQSTM1 Antibody (2C11) [H00008878-M01] - The content of mitochondrial p62, OPTN, & VCP in spinal cord of SOD1 \square G93A mice is decreased by Parkin knockout. Immunostaining of anterior horn of lumbar spinal cord for ChAT (in green) & p62 (in red) at 130 days. Scale bar, 10 μ m. Quantification of the percentage of ChAT \square positive motor neurons (MN) containing p62 \square positive inclusions at 130 days. PKO/G93A MN have less SOD1 aggregates than G93A, while Non Tg & PKO MN have none. Data were collected from n = 4 (two males & two females) mice for Non Tg & PKO & n = 6 (three males & three females) for G93A & PKO/G93A. Number of images recorded for each genotype: n = 9 for Non Tg & PKO, n = 16 for G93A, & n = 12 for PKO/G93A. Results are expressed as mean \pm SEM; *P = 0.032, by unpaired one-tailed Student's t-test. Representative Western blots of OPTN, p62, & VCP in spinal cord mitochondrial fractions at 130 days. Protein levels are normalized by Complex V. The quantifications in panels (D–F) show that mitochondria of PKO/G93A mice have less mitophagy adaptor proteins than G93A relative to Complex V. Quantification of OPTN relative to Complex V at 130 days. Results are expressed as mean \pm SEM & as percent of Non Tg; n = 8 (four males & four females); *P = 0.039 by paired Student's t-test. Quantification of p62, relative to Complex V at 130 days. Results are expressed as mean \pm SEM & as percent of Non Tg; n = 8 (four males & four females); **P = 0.0029 by paired Student's t-test. VCP was quantified using Complex V as loading reference at 130 days. Results are expressed as mean \pm SEM & as percent of Non Tg; n = 8 (four males & four females); *P = 0.044 by paired Student's t-test. Source data are available online for this figure. Image collected & cropped by CiteAb from the following publication (<https://pubmed.ncbi.nlm.nih.gov/30126943>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



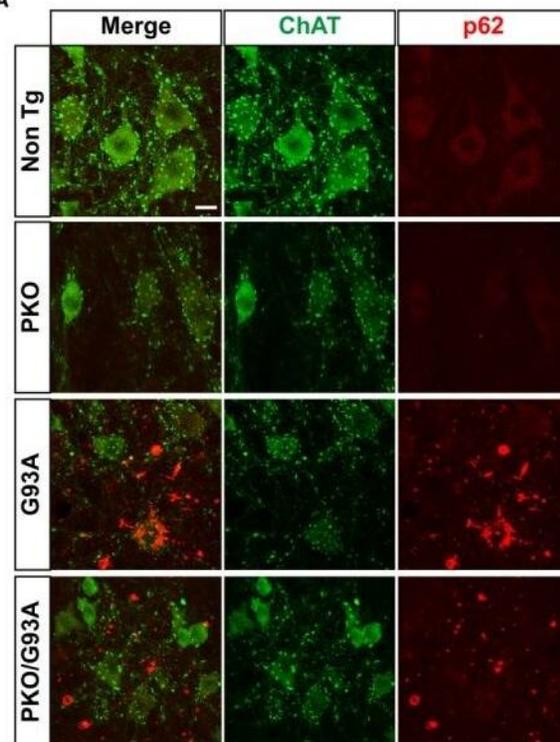
Western Blot: p62/SQSTM1 Antibody (2C11) [H00008878-M01] - miR-29a downregulates TFEB & ATG9A to inhibit autophagy. Schematic representation of the miR-29 family members & 3'-UTR binding sites of miR-29 targets as well as mutated binding sites used in Luciferase Assays: Transcription Factor EB (TFEB) & Autophagy-related protein 9A (ATG9A). All three miR-29 family members (miR-29a, miR-29b, & miR-29c) have identical seed sequences. Conserved miR-29 binding sites in the 3'-UTR of mRNA transcripts encoding ATG9A & TFEB are depicted in bold. B. 10ug of total protein cell lysates from Panc-1 transfected with CTRL or miR-29a mimics were subjected to western blot analysis for TFEB, ATG9A, & GAPDH. Relative quantification of band intensities normalized to GAPDH are shown below respective blots. C. Relative firefly luciferase activity from TFEB & ATG9A 3' UTR wild type (WT) & mutant (mut) reporter constructs following co-transfection into Panc-1 cells with control or miR-29a mimics. All readouts were normalized to renilla luciferase activity for each well. Average relative luminescence normalized to respective controls is presented (n=6) \pm S.E.M. D. 5ug of total protein cell lysates from Panc-1 cells were transfected with CTRL, miR-29a mimics, siCTRL, siTFEB, or siATG9A. 24 hours post-transfection, total protein was harvested & subjected to western blot analysis for p62 & LC3B, & GAPDH was used as loading control. Quantification of band intensities normalized to GAPDH & relative to control are shown below respective blots. All experiments were repeated 3 times & representative data is presented. Image collected & cropped by CiteAb from the following publication (<https://www.oncotarget.com/lookup/doi/10.18632/oncotarget.11928>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



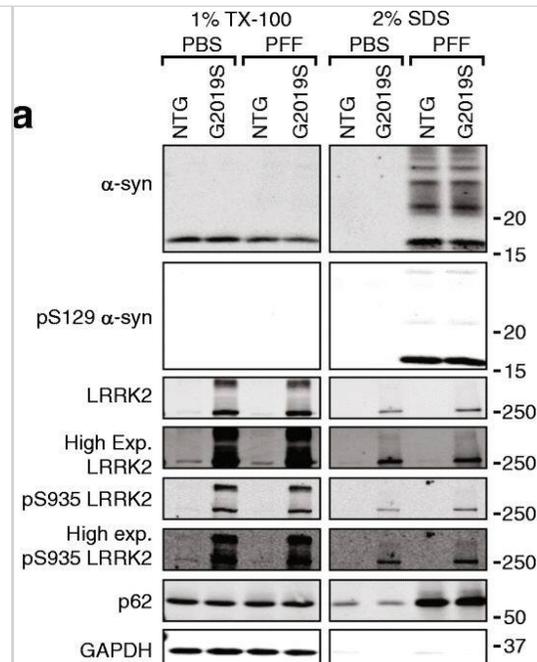
Western Blot: p62/SQSTM1 Antibody (2C11) [H00008878-M01] - Effects of autophagy inhibitors in combination with IFN- γ on expression of PD-L1 in gastric cancer cell lines. a The effect of chloroquine (CQ) or 3-MA on expression of PD-L1 with or without IFN- γ for 24 h was determined by flow cytometry assays. In AGS & NCI-n87 cells, MFI as the indication of PD-L1 expression level can be further increased by the treatment of IFN- γ . b Levels of LC3B-I/II, p62/SQSTM1 & PD-L1 were determined by Western blots in AGS & NCI-n87 cells treated by CQ, 3-MA, bafilomycin A1 (Baf) or rapamycin (Rap) for 24 h. c Positive staining of PD-L1 (red) & LC3 positive puncta (green) was determined by immunofluorescence in AGS & NCI-n87 cells treated by autophagy inhibitors & activator as in (b). d Rapamycin decreased the levels of PD-L1 protein in AGS & NCI-n87 cells as shown by flow cytometry. Results were averaged & blots were representative of 4 independent experiments, * $p < 0.05$, ** $p < 0.01$ Image collected & cropped by CiteAb from the following publication (<https://pubmed.ncbi.nlm.nih.gov/30925913>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.

B

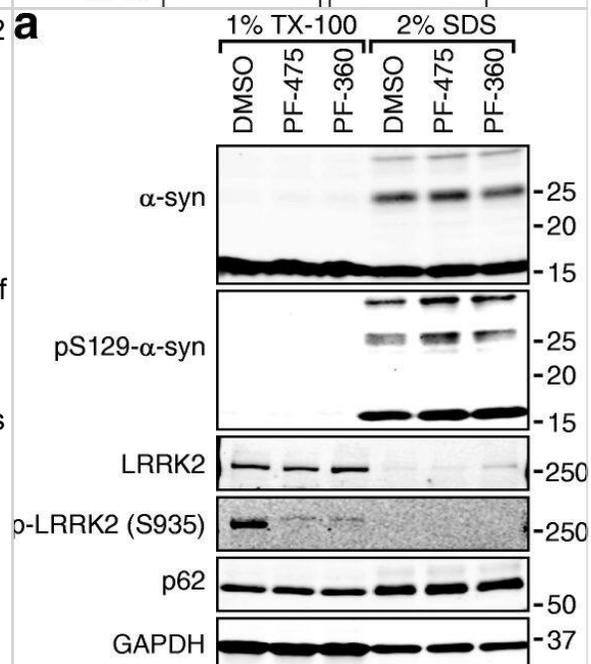
Immunocytochemistry/ Immunofluorescence: p62/SQSTM1 Antibody (2C11) [H00008878-M01] - The content of mitochondrial p62, OPTN, & VCP in spinal cord of SOD1 \square G93A mice is decreased by Parkin knockout. Immunostaining of anterior horn of lumbar spinal cord for ChAT (in green) & p62 (in red) at 130 days. Scale bar, 10 μ m. Quantification of the percentage of ChAT \square positive motor neurons (MN) containing p62 \square positive inclusions at 130 days. PKO/G93A MN have less SOD1 aggregates than G93A, while Non Tg & PKO MN have none. Data were collected from $n = 4$ (two males & two females) mice for Non Tg & PKO & $n = 6$ (three males & three females) for G93A & PKO/G93A. Number of images recorded for each genotype: $n = 9$ for Non Tg & PKO, $n = 16$ for G93A, & $n = 12$ for PKO/G93A. Results are expressed as mean \pm SEM; * $P = 0.032$, by unpaired one-tailed Student's t -test. Representative Western blots of OPTN, p62, & VCP in spinal cord mitochondrial fractions at 130 days. Protein levels are normalized by Complex V. The quantifications in panels (D–F) show that mitochondria of PKO/G93A mice have less mitophagy adaptor proteins than G93A relative to Complex V. Quantification of OPTN relative to Complex V at 130 days. Results are expressed as mean \pm SEM & as percent of Non Tg; $n = 8$ (four males & four females); * $P = 0.039$ by paired Student's t -test. Quantification of p62, relative to Complex V at 130 days. Results are expressed as mean \pm SEM & as percent of Non Tg; $n = 8$ (four males & four females); ** $P = 0.0029$ by paired Student's t -test. VCP was quantified using Complex V as loading reference at 130 days. Results are expressed as mean \pm SEM & as percent of Non Tg; $n = 8$ (four males & four females); * $P = 0.044$ by paired Student's t -test. Source data are available online for this figure. Image collected & cropped by CiteAb from the following publication (<https://pubmed.ncbi.nlm.nih.gov/30126943>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.

A

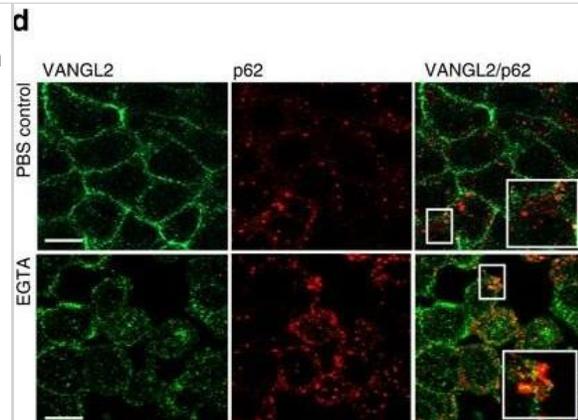
Western Blot: p62/SQSTM1 Antibody (2C11) [H00008878-M01] - G2019S LRRK2 hippocampal neurons do not have elevated α -synuclein pathology 14 days post-transduction. a Primary hippocampal neurons from NTG or G2019S pups were transduced with 2.5 μ g/mL α -synuclein PFFs & allowed to age a further 14 days prior to sequential detergent fractionation. TX-100-insoluble α -synuclein & p62 are similar in both neuron types. b Quantification of soluble proteins shows ~25-fold elevation in the expression of LRRK2 & a commensurate ~50-fold elevation in pS395 LRRK2, indicative of the elevated LRRK2 kinase activity associated with the G2019S mutation. Soluble α -synuclein levels were equivalent between the cultures. c No significant differences were found between the genotypes in insoluble proteins by an unpaired t-test with Welch's correction. (N = 3 biological replicates for each protein). Means + s.e.m.; **P < 0.01; *P < 0.05 by an unpaired t-test with Welch's correction for unequal variances. All values are normalized to NTG neurons treated with α -synuclein PFFs & DMSO Image collected & cropped by CiteAb from the following publication (<https://actaneurocomms.biomedcentral.com/articles/10.1186/s40478-018-0550-0>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



Western Blot: p62/SQSTM1 Antibody (2C11) [H00008878-M01] - LRRK2 inhibition does not reduce insoluble α -synuclein in wildtype hippocampal neurons. a Primary hippocampal neurons from CD1 pups were treated with 30 nM LRRK2 inhibitors PF-475, PF-360 or DMSO as a vehicle control, then transduced with 2.5 μ g/mL α -synuclein PFFs & allowed to age a further 14 days prior to sequential detergent fractionation. TX-100-insoluble α -synuclein & p62 are similar in both LRRK2 inhibitor treated & untreated neurons. b Quantification of soluble proteins show some reduction of LRRK2 protein levels PF-475 treatment & ~75% inhibition of LRRK2 activity (as assayed by pS935 LRRK2) by both PF-475 & PF-360. c Insoluble pS129 α -synuclein was slightly, but significantly elevated by PF-360 treatment, while α -synuclein & p62 were unchanged by one-way ANOVA. (N = 5 biological replicates for each protein). Means + s.e.m.; *P < 0.05, ****p < 0.0001 by one-way ANOVA with Dunnett's multiple comparison test. All values are normalized first to GAPDH, as a loading control, then to DMSO-treated neurons Image collected & cropped by CiteAb from the following publication (<https://actaneurocomms.biomedcentral.com/articles/10.1186/s40478-018-0550-0>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



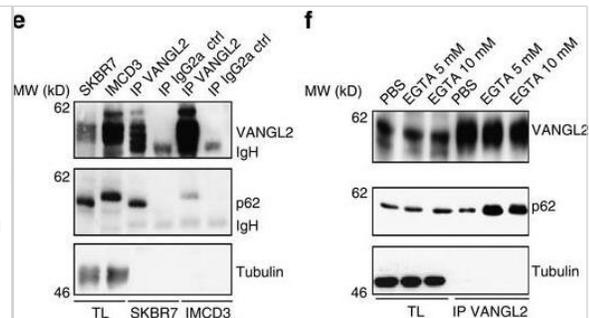
Immunocytochemistry/ Immunofluorescence: p62/SQSTM1 Antibody (2C11) [H00008878-M01] - Colocalization of VANGL2 & p62/SQSTM1 in late endosomes. (a) Immunofluorescence staining of SKBR7 cells showed colocalization of endogenous VANGL2 (green) & p62/SQSTM1 (red) in discrete cytoplasmic puncta. Scale bar, 10 μ m. The mean Pearson correlation for VANGL2 & p62/SQSTM1 is 0.62, calculated using the Image J software for \square 15 cells per field of view & from 10 images. (b) Partial colocalization of VANGL2 & p62/SQSTM1 in late endosomes of SKBR7 cells stained w/ the LAMP1 marker. Scale bar, 20 μ m. (c) SKBR7 cells cultured in a nutrient-deprived medium & treated w/ 100 nM bafilomycin A1 (6 h) before fixation. Double labelling against VANGL2 (arrowheads) & p62/SQSTM1 (arrows), as described in Methods, showed accumulations of both markers in vesicular structures probably resembling endosomes/amphisomes. Scale bar, 200 nm. (d)



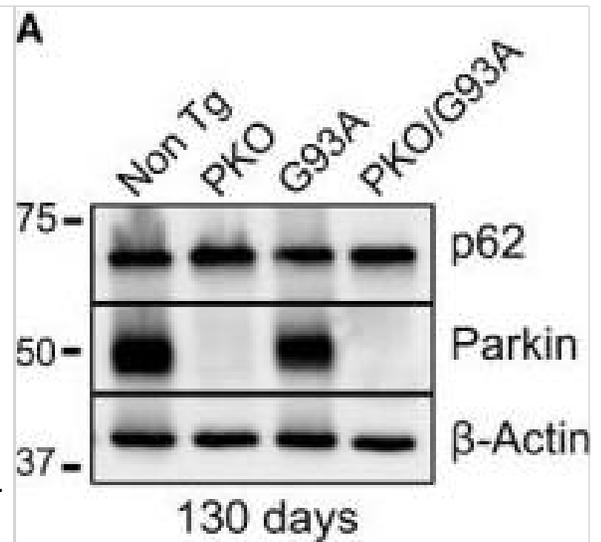
IMCD3 cells treated w/ PBS or EGTA (5 mM) for 30 min. Immunofluorescence & confocal analysis performed using the indicated antibodies. Scale bar, 10 μ m. Inserts show colocalized VANGL2 & p62/SQSTM1. (e) The VANGL2–p62/SQSTM1 complex recovered in confluent SKBR7 or IMCD3 cells w/ 2G4 mAb (IP VANGL2) but not a control antibody (IP IgG2a ctrl) as seen using western blot analysis w/ the indicated antibodies. The complex more abundant in cancer cells (SKBR7) than in polarized cells (IMCD3). Note that human (SKBR7 cells) & murine (IMCD3 cells) p62/SQSTM1 run at different molecular weights. (f) Lysates of confluent IMCD3 cells treated for 30 min w/ PBS or EGTA subjected to 2G4 mAb immunoprecipitation (IP VANGL2).

Immunoprecipitated proteins probed by western blot analysis w/ the indicated antibodies. Increased amounts of VANGL2–p62/SQSTM1 complexes recovered after EGTA treatment. Image collected & cropped by CiteAb from the following publication (<https://www.nature.com/articles/ncomms10318>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.

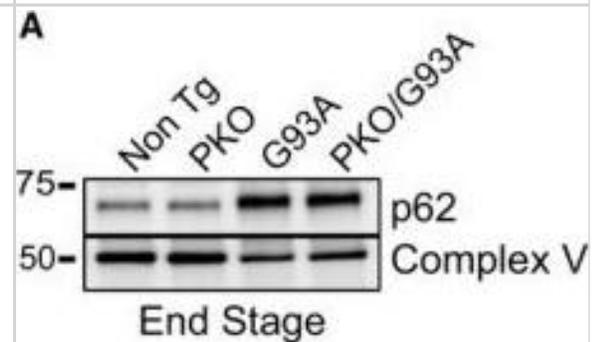
Western Blot: p62/SQSTM1 Antibody (2C11) [H00008878-M01] - Colocalization of VANGL2 & p62/SQSTM1 in late endosomes. (a) Immunofluorescence staining of SKBR7 cells showed colocalization of endogenous VANGL2 (green) & p62/SQSTM1 (red) in discrete cytoplasmic puncta. Scale bar, 10 μ m. The mean Pearson correlation for VANGL2 & p62/SQSTM1 is 0.62, calculated using the Image J software for \square 15 cells per field of view & from 10 images. (b) Partial colocalization of VANGL2 & p62/SQSTM1 in late endosomes of SKBR7 cells stained w/ the LAMP1 marker. Scale bar, 20 μ m. (c) SKBR7 cells cultured in a nutrient-deprived medium & treated w/ 100 nM bafilomycin A1 (6 h) before fixation. Double labelling against VANGL2 (arrowheads) & p62/SQSTM1 (arrows), as described in Methods, showed accumulations of both markers in vesicular structures probably resembling endosomes/amphisomes. Scale bar, 200 nm. (d) IMCD3 cells treated w/ PBS or EGTA (5 mM) for 30 min. Immunofluorescence & confocal analysis performed using the indicated antibodies. Scale bar, 10 μ m. Inserts show colocalized VANGL2 & p62/SQSTM1. (e) The VANGL2–p62/SQSTM1 complex recovered in confluent SKBR7 or IMCD3 cells w/ 2G4 mAb (IP VANGL2) but not a control antibody (IP IgG2a ctrl) as seen using western blot analysis w/ the indicated antibodies. The complex more abundant in cancer cells (SKBR7) than in polarized cells (IMCD3). Note that human (SKBR7 cells) & murine (IMCD3 cells) p62/SQSTM1 run at different molecular weights. (f) Lysates of confluent IMCD3 cells treated for 30 min w/ PBS or EGTA subjected to 2G4 mAb immunoprecipitation (IP VANGL2). Immunoprecipitated proteins probed by western blot analysis w/ the indicated antibodies. Increased amounts of VANGL2–p62/SQSTM1 complexes recovered after EGTA treatment. Image collected & cropped by CiteAb from the following publication (<https://www.nature.com/articles/ncomms10318>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



Western Blot: p62/SQSTM1 Antibody (2C11) [H00008878-M01] - Parkin protein levels are unaffected in liver while a small decrease is measured in G93A cerebellum. Representative Western blot for Parkin & p62 in cerebellum (Cb) homogenates at 130 days. Quantification indicates that Parkin is decreased in G93A mice relative to Non Tg at 130 days. Protein levels were normalized by β -actin. Results are expressed as mean \pm SEM & percent of Non Tg; n = 8 (four males & four females) mice per group. *P = 0.039 by paired Wilcoxon's test. Quantification of p62 in the homogenates indicates that p62 is increased in PKO & PKO/G93A mice, independent of SOD1-G93A expression, at 130 days of age. β -actin was used for normalization. Results are expressed as mean \pm SEM & percent of Non Tg; n = 8 (four males & four females) mice per group. No statistically significant differences were found between Non Tg & G93A by paired Friedman's test with Dunn's correction (P = 0.99). ***P = 0.0001 by paired Student's t-test (G93A vs. PKO/G93A). Representative Western blot of Parkin & p62 in liver homogenates at 130 days. Parkin protein levels were quantified at 130 days. β -actin was used for normalization. Results are expressed as mean \pm SEM & percent of Non Tg; n = 8 (four males & four females) mice per group. No statistically significant differences were found between Non Tg & G93A (P = 0.546 by paired Wilcoxon's test). Quantification of p62 protein levels in liver at 130 days. Results are expressed as mean \pm SEM & percent of Non Tg; n = 8 (four males & four females) mice per group. No statistically significant differences were found between Non Tg & G93A (P = 0.546 by paired Wilcoxon's test). No statistically significant differences were found among the other groups. Source data are available online for this figure. Image collected & cropped by CiteAb from the following publication (<https://pubmed.ncbi.nlm.nih.gov/30126943>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



Western Blot: p62/SQSTM1 Antibody (2C11) [H00008878-M01] - Parkin knockout mitigates the accumulation of mitophagy adaptors in G93A mitochondria at disease end stage. Representative Western blots of p62 (A), OPTN, & VCP (B) in spinal cord mitochondria from end-stage mice. p62 quantification, using Complex V as normalizer, shows a strong accumulation of p62 in SOD1-G93A mitochondria at end stage. Results are expressed as mean \pm SEM & as percent of Non Tg; n = 8 (four males & four females) mice per group. No statistically significant differences were found between G93A & PKO/G93A (P = 0.078 by paired Wilcoxon's test); ***P = 0.0007 (Non Tg vs. G93A) & *P = 0.037 (PKO & PKO/G93A) both by paired Friedman's test with Dunn's correction. Quantification of OPTN accumulation in end-stage mitochondria, with Complex V as protein loading control. Results are expressed as mean \pm SEM & as percent of Non Tg; n = 8 (four males & four females) mice per group. **P = 0.0078 (for G93A & PKO/G93A) by paired Wilcoxon's test; ***P = 0.0003 (Non Tg vs. G93A) by paired Friedman's test with Dunn's correction. No other statistically significant differences were found. Quantification of VCP in mitochondria at disease end stage. Complex V was used as normalizer. Results are expressed as mean \pm SEM & as percent of Non Tg; n = 8 (four males & four females) mice per group. *P = 0.039 (for G93A & PKO/G93A) by paired Wilcoxon's test; ***P = 0.0007 (Non Tg vs. G93A) & *P = 0.037 (PKO & PKO/G93A) both by paired Friedman's test with Dunn's correction. Source data are available online for this figure. Image collected & cropped by CiteAb from the following publication (<https://pubmed.ncbi.nlm.nih.gov/30126943>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



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