Product Datasheet

Otubain-2 Antibody (OTI11B3)
NBP2-03223

Unit Size: 0.1 ml

Store at -20C. Avoid freeze-thaw cycles.

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Publications: 1

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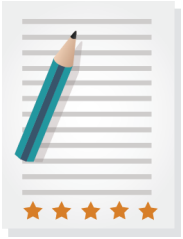
Updated 9/9/2025 v.20.1



NBP2-03223

Otubain-2 Antibody (OTI11B3)

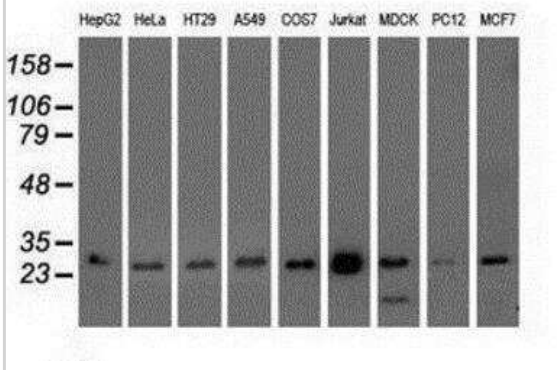
Product Information	
Unit Size	0.1 ml
Concentration	1 mg/ml
Storage	Store at -20C. Avoid freeze-thaw cycles.
Clonality	Monoclonal
Clone	OTI11B3
Preservative	0.02% Sodium Azide
Isotype	IgG1
Purity	Immunogen affinity purified
Buffer	PBS (pH 7.3), 1.0% BSA and 50% Glycerol
Target Molecular Weight	27 kDa

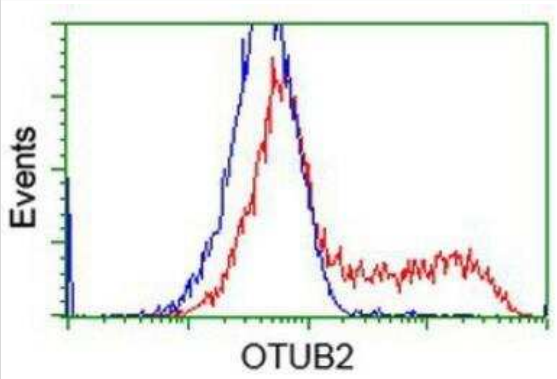
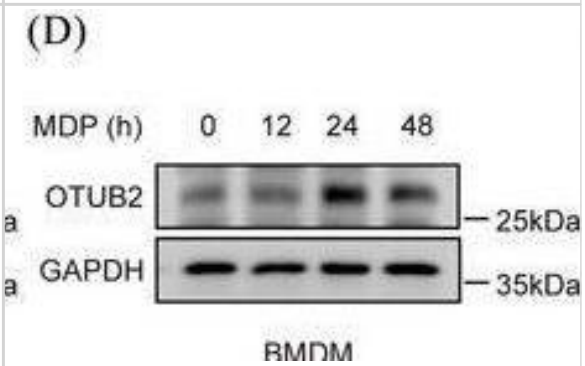
Product Description	
Description	Novus Biologicals Mouse Otubain-2 Antibody (OTI11B3) (NBP2-03223) is a monoclonal antibody validated for use in WB and Flow. Anti-Otubain-2 Antibody: Cited in 1 publication. All Novus Biologicals antibodies are covered by our 100% guarantee.
Host	Mouse
Gene ID	78990
Gene Symbol	OTUB2
Species	Human, Mouse, Rat, Canine, Monkey
Immunogen	Full length human recombinant protein of human OTUB2 (NP_075601) produced in HEK293T cell.

Product Application Details	
Applications	Western Blot, Flow Cytometry
Recommended Dilutions	Western Blot 1:500-2000, Flow Cytometry 1:100

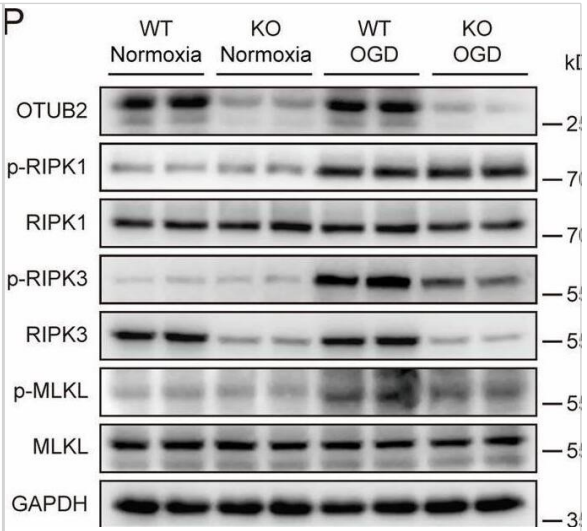
Images

Western Blot: Otubain-2 Antibody (11B3) [NBP2-03223] - Analysis of extracts (35ug) from 9 different cell lines by using anti-Otubain-2 monoclonal antibody.



Flow Cytometry: Otubain-2 Antibody (11B3) [NBP2-03223] - HEK293T cells transfected with either overexpression plasmid (Red) or empty vector control plasmid (Blue) were immunostained by anti-Otubain-2 Antibody, and then analyzed by flow cytometry.	
Western Blot: Otubain-2 Antibody (11B3) [NBP2-03223] - HEK293T cells were transfected with the pCMV6-ENTRY control (Left lane) or pCMV6-ENTRY OTUB2 (Right lane) cDNA for 48 hrs and lysed. Equivalent amounts of cell lysates (5 ug per lane) were separated by SDS-PAGE and immunoblotted with anti-OTUB2.	
OTUB2 enhances MDP□induced cytokine production in macrophages. (A, B) Representative OTUB2 (red) and F4/80 (green) immunofluorescence staining of colon samples from humans (A) and mice (B). Scale bar = 100 um. (C, D) BMDM isolated from C57BL/6 mice were stimulated with 500 ng/mL LPS (C) or 200 ng/mL L18□MDP (D) for indicated periods of time. OTUB2 protein levels were then analysed by Western blot. (E) BMDM isolated from C57BL/6 mice were stimulated with 200 ng/mL L18□MDP for indicated periods of time. The relative mRNA levels of Otub2 were determined by qRT□PCR. Data are presented as the relative increase over untreated control samples. (F) RAW264.7 cells were stimulated with 200 ng/mL L18□MDP for indicated periods of time. OTUB2 protein levels were then analysed by Western blot. (G–K) BMDMs isolated from Otub2+/+ and Otub2–/– mice were stimulated with 200 ng/mL L18□MDP for 3 h or left untreated. The relative expression of Il1b (G), Il6 (H), Tnf (I), Cxcl2 (J) and Cxcl10 (K) mRNA was determined by qRT□PCR. Data are presented as the relative increase over untreated control samples. Data in E and G–K are displayed as mean +/- SEM. *p < .05, **p < .01, ***p < .001. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/39358938), licensed under a CC-BY license. Not internally tested by Novus Biologicals.	

OTUB2 deficiency ameliorates RIPK3-mediated neuronal necroptosis. (A–G) Four hours after MCAO, the ischemic cerebral hemisphere was analyzed by Western blot with indicated antibodies. Representative immunoblots (A) as well as relative quantification of p-RIPK1 (B), p-RIPK3 (C), p-MLKL (D), RIPK1 (E), RIPK3 (F), and MLKL (G) are shown. Two-way ANOVA, n = 3/group, biological replicates. (H, I) Four hours after MCAO, the ischemic penumbra was analyzed by immunofluorescence with indicated antibodies. Representative images (H) and percentages of p-MLKL+ neurons (I) are shown. Scale bar, 100 μm. Unpaired Student's t test, n = 3/group, biological replicates. (J–L) Four hours after MCAO, the relative transcription of Il1b (J), Il6 (K), and Tnf (L) in the ischemic cerebral hemisphere was determined by qRT-PCR. Unpaired Student's t test, n = 3–5/group, biological replicates. (M, N) Representative TTC staining (M) and percentages of cerebral infarct volume (N) on day 1 after MCAO. Unpaired Student's t test, n = 5/group, biological replicates. (O) Otub2+/+ and Otub2−/− HT22 cells were subjected to OGD for 6 h followed by reoxygenation for 12 h in the presence or absence of Nec-1 (50 μM). Cell viability was measured by CCK-8 test. Two-way ANOVA, n = 10/group, biological replicates. (P) Otub2+/+ and Otub2−/− HT22 cells were subjected to OGD for 6 h followed by reoxygenation for 12 h. Whole-cell lysates were analyzed by Western blot with indicated antibodies. (Q) After OGD treatment for 6 h followed by reoxygenation for 12 h, Otub2+/+ and Otub2−/− HT22 cells were analyzed by immunofluorescence with indicated antibodies. Scale bar, 20 μm. Data in (B–G, I, J–L, N, O) show the mean +/- SEM. Source data are available online for this figure. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/40021931), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



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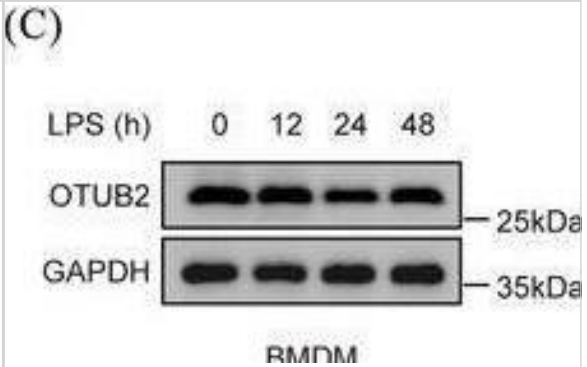
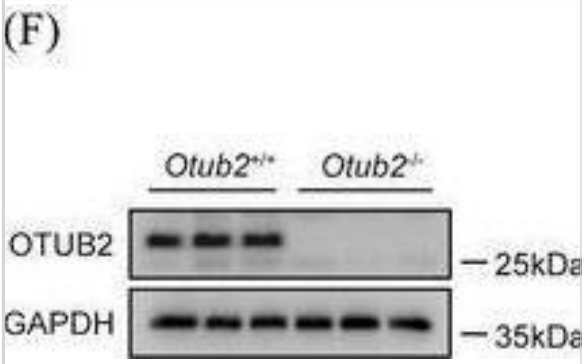


Colonic expression OTUB2 is downregulated in colitis. (A) Representative immunohistochemical staining of OTUB2 in control and UC colon samples. Scale bar = 50 um. (B) Representative immunohistochemical staining of OTUB2 in colon samples from control and DSS□treated C57BL/6 mice. Control mice received regular drinking water. Mice in the DSS group were given 2% DSS for 8 days, followed by regular drinking water for 2 days. Scale bar = 50 um. (C, D) Representative immunoblots (C) and relative quantification (D) of OTUB2 protein in colon samples from control and DSS□treated C57BL/6 mice. (E) Relative mRNA levels of Otub2 in colon samples from control and DSS□treated C57BL/6 mice were analysed by qRT□PCR. (F) Western blot analysis of OTUB2 protein abundance in colon samples from Otub2+/+ and Otub2–/– mice. (G) The representative image (left) and length (right) of colons from Otub2+/+ and Otub2–/– mice. (H, I) Representative H&E (H) and PAS/AB (I) staining of colons from Otub2+/+ and Otub2–/– mice. Scale bar = 50 um. Data in D, E and G are shown as mean +/- SEM. ns, no significant difference. *p < .05, **p < .01. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/39358938), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



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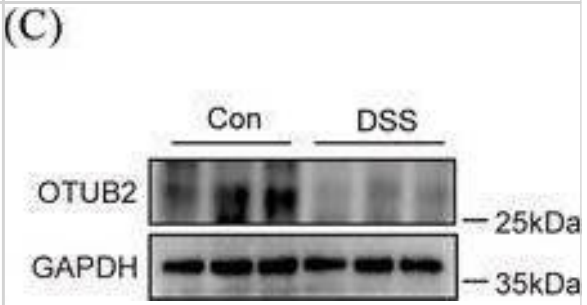

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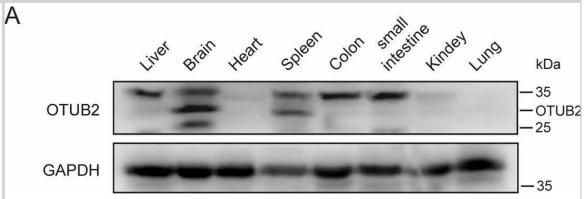

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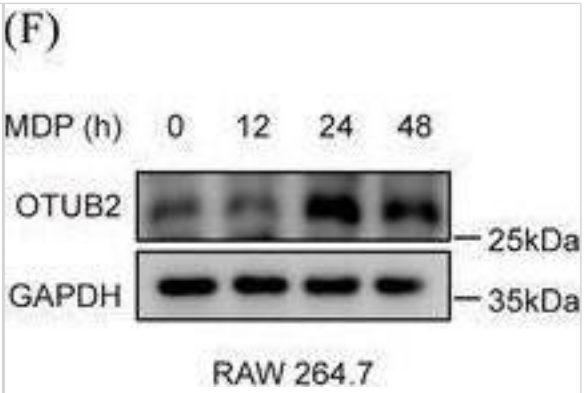

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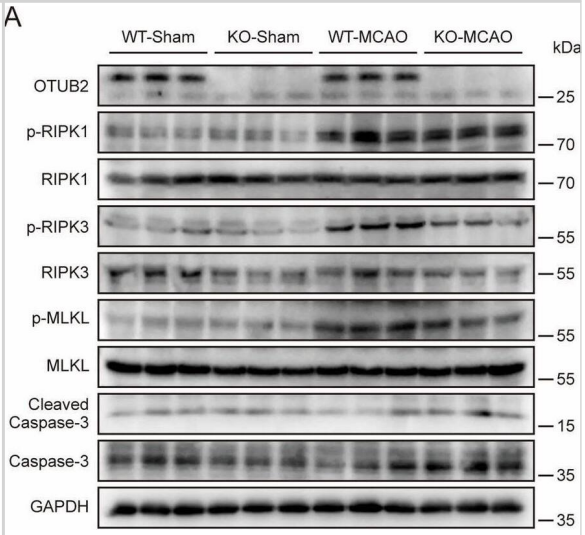
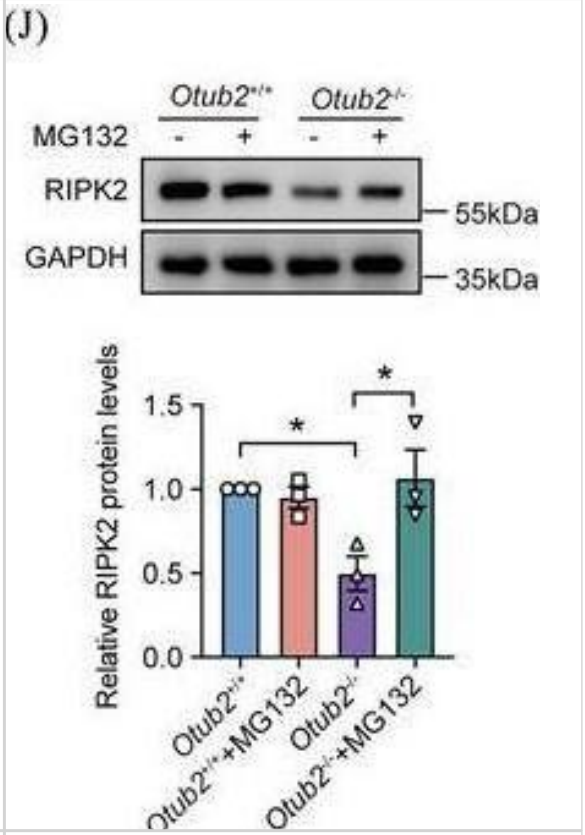


OTUB2 enhances MDP□induced signalling by inhibiting the proteasomal degradation of RIPK2. (A) After stimulation with 200 ng/mL L18□MDP for indicated periods of time, BMDMs derived from Otub2+/+ and Otub2–/– mice were lysed and analysed by Western blot with indicated antibodies. Densitometric quantification is shown in the figure. (B) Schematic diagram of MDP□induced signalling. (C) The protein abundance of NOD2, RIPK2 and XIAP in Otub2+/+ and Otub2–/– BMDMs was determined by Western blot. (D–F) The relative protein levels of NOD2 (D), XIAP (E) and RIPK2 (F) in Otub2+/+ and Otub2–/– BMDMs. (G) The relative mRNA levels of Ripk2 in Otub2+/+ and Otub2–/– BMDMs were determined by qRT□PCR. (H) RAW264.7 cells were transfected with FLAG□Vector or FLAG□OTUB2 plasmids for 24 h. Thereafter, cells were lysed and analysed by Western blot with indicated antibodies. (I) After treatment with 20 ng/mL CHX for indicated periods of time, BMDMs isolated from Otub2+/+ and Otub2–/– mice were lysed and analysed by Western blot with indicated antibodies. (J, K) BMDMs isolated from Otub2+/+ and Otub2–/– mice were treated with 20 uM MG132 (J) or 50 uM CQ (K) for 6 h or left untreated. Whole Cell lysates were analysed by Western blot with indicated antibodies. Representative immunoblots (upper panel) and quantification (lower panel) are shown. Data in D–G, J and K are shown as mean +/- SEM. ns, no significant difference. *p < .05, **p < .01. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/39358938), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



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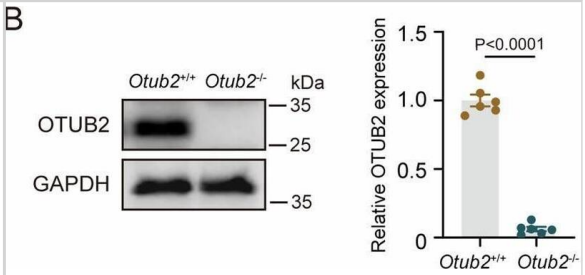
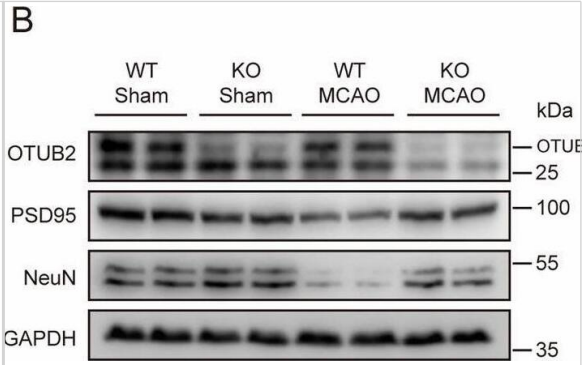
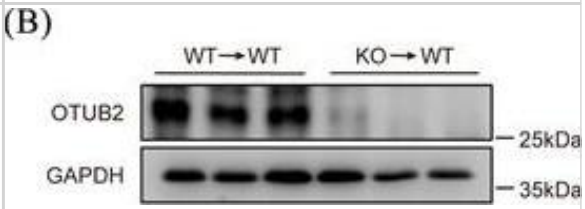
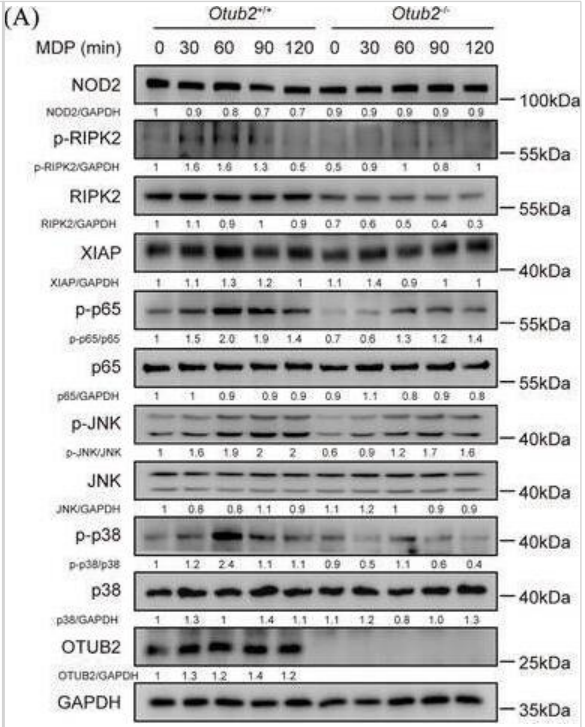
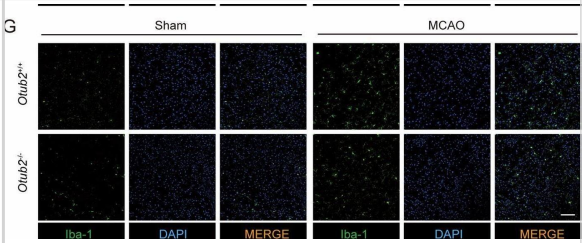


OTUB2 deficiency diminishes MCAO-induced neuronal loss and neuroinflammation.(A) Representative immunofluorescence staining of OTUB2 (red) and NeuN (green) in the brain of C57BL/6 mice. Scale bar, 20 μm. (B) On day 2 after MCAO, the ischemic cerebral hemisphere of Otub2+/+ and Otub2−/− mice was analyzed by Western blot with indicated antibodies. (C, D) The relative expression of NeuN (C) and PSD95 (D) was quantified after normalization to GAPDH. Unpaired Student's t test, n = 4/group, biological replicates. (E, F) Representative immunofluorescence staining (E) and quantification (F) of NeuN+ cells in the ischemic penumbra on day 2 after MCAO. Scale bar, 100 μm. Unpaired Student's t test, n = 5/group, biological replicates. (G) Representative immunofluorescence staining of Iba1+ cells in the ischemic penumbra on day 2 after MCAO. Scale bar, 100 μm. (H) Representative z-stack images of Iba1+ cells in the ischemic penumbra on day 2 after MCAO. Scale bar, 5 μm. (I–M) Forty-eight hours after MCAO, the relative transcription of Il1b (I), Il6 (J), Tnf (K), Ccl2 (L), and Cxcl10 (M) in the ischemic cerebral hemisphere was determined by qRT-PCR. Mann–Whitney U test (I) and Unpaired Student's t test (J–M), n = 5–7/group, biological replicates. Data in (C, D, I–M) show the mean +/- SEM. Source data are available online for this figure. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/40021931), licensed under a CC-BY license. Not internally tested by Novus Biologicals.	
Deficiency of OTUB2 in haematopoietic cells exacerbates DSS□induced colitis. (A) Experimental flowchart for bone marrow transplantation. (B) Eight weeks after bone marrow transplantation, OTUB2 expression in splenocytes of Otub2+/+ mice receiving bone marrow from Otub2+/+ (WT→WT) and Otub2–/– (KO→WT) mice was analysed by Western blot. (C, D) The chimeric mice were fed drinking water containing 2% DSS for 8 days, and then given normal drinking water for 2 days. Body weight (C) and disease activity index (D) were recorded daily (n = 7/group). (E, F) The representative image (E) and length (F) of colons from chimeric mice on day 10 after DSS treatment. (G and H) Histology score (G) as well as representative H&E and PAS/AB staining (H) of colons from chimeric mice on day 10 after DSS treatment. Scale bar = 50 um. Data in C, D, F and G are shown as mean +/- SEM. *p < .05, **p < .01, ***p < .001. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/39358938), licensed under a CC-BY license. Not internally tested by Novus Biologicals.	

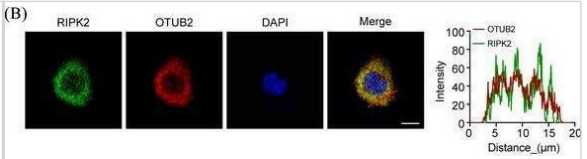
OTUB2 enhances MDP□induced signalling by inhibiting the proteasomal degradation of RIPK2. (A) After stimulation with 200 ng/mL L18□MDP for indicated periods of time, BMDMs derived from Otub2+/+ and Otub2–/– mice were lysed and analysed by Western blot with indicated antibodies. Densitometric quantification is shown in the figure. (B) Schematic diagram of MDP□induced signalling. (C) The protein abundance of NOD2, RIPK2 and XIAP in Otub2+/+ and Otub2–/– BMDMs was determined by Western blot. (D–F) The relative protein levels of NOD2 (D), XIAP (E) and RIPK2 (F) in Otub2+/+ and Otub2–/– BMDMs. (G) The relative mRNA levels of Ripk2 in Otub2+/+ and Otub2–/– BMDMs were determined by qRT□PCR. (H) RAW264.7 cells were transfected with FLAG□Vector or FLAG□OTUB2 plasmids for 24 h. Thereafter, cells were lysed and analysed by Western blot with indicated antibodies. (I) After treatment with 20 ng/mL CHX for indicated periods of time, BMDMs isolated from Otub2+/+ and Otub2–/– mice were lysed and analysed by Western blot with indicated antibodies. (J, K) BMDMs isolated from Otub2+/+ and Otub2–/– mice were treated with 20 uM MG132 (J) or 50 uM CQ (K) for 6 h or left untreated. Whole Cell lysates were analysed by Western blot with indicated antibodies. Representative immunoblots (upper panel) and quantification (lower panel) are shown. Data in D–G, J and K are shown as mean +/- SEM. ns, no significant difference. *p < .05, **p < .01. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/39358938), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



OTUB2 deficiency diminishes MCAO-induced neuronal loss and neuroinflammation.(A) Representative immunofluorescence staining of OTUB2 (red) and NeuN (green) in the brain of C57BL/6 mice. Scale bar, 20 μm. (B) On day 2 after MCAO, the ischemic cerebral hemisphere of Otub2+/+ and Otub2−/− mice was analyzed by Western blot with indicated antibodies. (C, D) The relative expression of NeuN (C) and PSD95 (D) was quantified after normalization to GAPDH. Unpaired Student's t test, n = 4/group, biological replicates. (E, F) Representative immunofluorescence staining (E) and quantification (F) of NeuN+ cells in the ischemic penumbra on day 2 after MCAO. Scale bar, 100 μm. Unpaired Student's t test, n = 5/group, biological replicates. (G) Representative immunofluorescence staining of Iba1+ cells in the ischemic penumbra on day 2 after MCAO. Scale bar, 100 μm. (H) Representative z-stack images of Iba1+ cells in the ischemic penumbra on day 2 after MCAO. Scale bar, 5 μm. (I–M) Forty-eight hours after MCAO, the relative transcription of Il1b (I), Il6 (J), Tnf (K), Ccl2 (L), and Cxcl10 (M) in the ischemic cerebral hemisphere was determined by qRT-PCR. Mann–Whitney U test (I) and Unpaired Student's t test (J–M), n = 5–7/group, biological replicates. Data in (C, D, I–M) show the mean +/- SEM. Source data are available online for this figure. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/40021931), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



OTUB2 interacts with RIPK2 to mediate K48 deubiquitination. (A) BMDM lysates were immunoprecipitated with anti□OTUB2 antibody. Immunoprecipitated proteins and cell lysates were analysed by Western blot with indicated antibodies. (B) Subcellular distribution of OTUB2 (red) and RIPK2 (green) in BMDMs was examined by immunofluorescence. Scale bar = 5 um. (C, D) NIH/3T3 cells were co□transfected with FLAG□OTUB2 and HIS□MYC□RIPK2 plasmids for 24 h. Whole□cell lysates were immunoprecipitated with anti□FLAG (C) or anti□MYC (D) antibodies. Immunoprecipitated proteins and cell lysates were analysed by Western blot with the indicated antibodies. (E) BMDMs were treated with 20 uM MG132 for 6 h before lysis. Proteins were immunoprecipitated from whole□cell lysates with anti□RIPK2 antibody and analysed by Western blot. (F, G) Schematic diagram (F) and representative immunoblots (G) of the in vitro deubiquitination assay. (H) Schematic diagram of the OTUB2 active site and the C51S mutant construct. (I, J) NIH/3T3 cells were transfected with the indicated plasmids for 24 h, followed by treatment with 20 uM MG132 for 6 h. Proteins were immunoprecipitated from whole□cell lysates with anti□MYC antibody and analysed by Western blot. (K) NIH/3T3 cells were transfected with the indicated plasmids for 24 h. Whole□cell lysates were analysed by Western blot with the indicated antibodies. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/39358938), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



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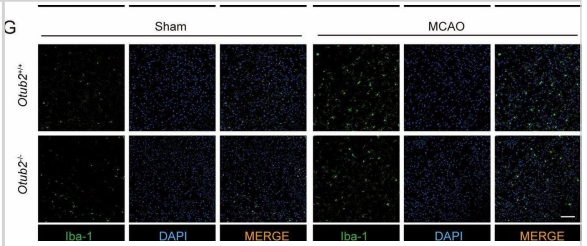

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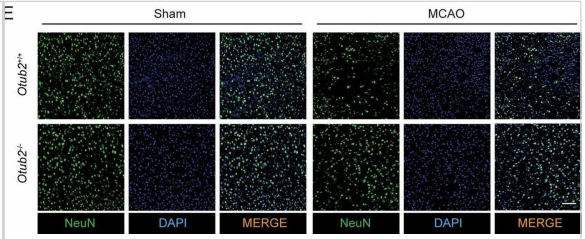

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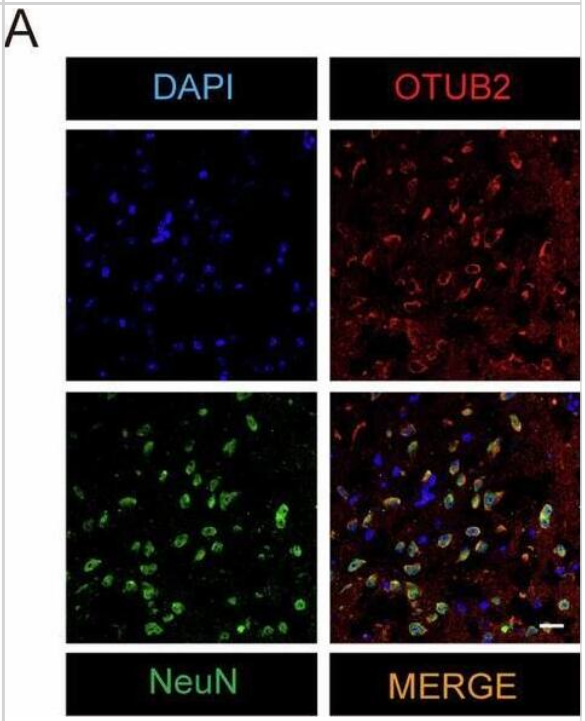
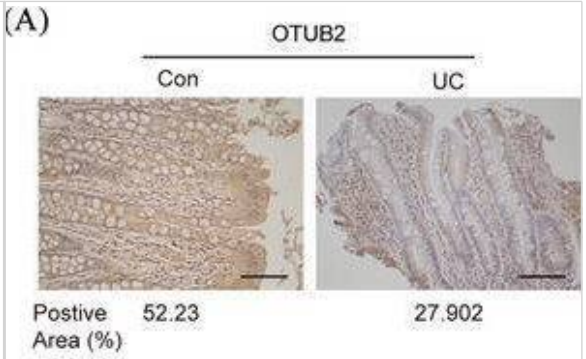
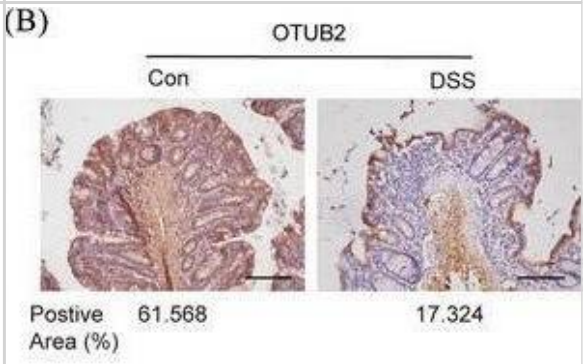
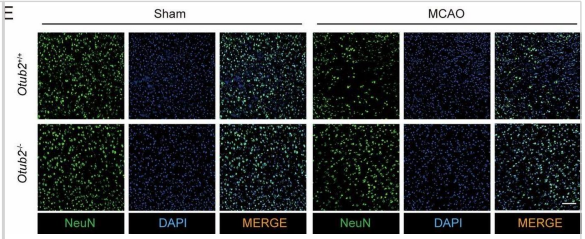
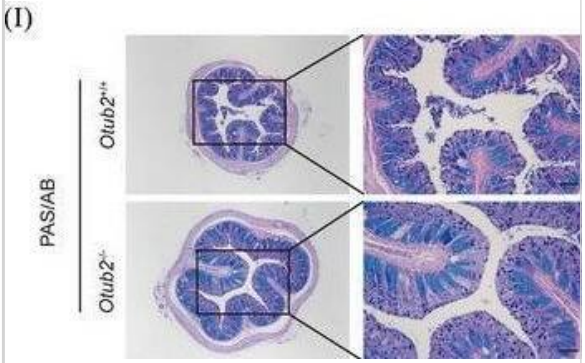


Colonic expression OTUB2 is downregulated in colitis. (A) Representative immunohistochemical staining of OTUB2 in control and UC colon samples. Scale bar = 50 um. (B) Representative immunohistochemical staining of OTUB2 in colon samples from control and DSS□treated C57BL/6 mice. Control mice received regular drinking water. Mice in the DSS group were given 2% DSS for 8 days, followed by regular drinking water for 2 days. Scale bar = 50 um. (C, D) Representative immunoblots (C) and relative quantification (D) of OTUB2 protein in colon samples from control and DSS□treated C57BL/6 mice. (E) Relative mRNA levels of Otub2 in colon samples from control and DSS□treated C57BL/6 mice were analysed by qRT□PCR. (F) Western blot analysis of OTUB2 protein abundance in colon samples from Otub2+/+ and Otub2–/– mice. (G) The representative image (left) and length (right) of colons from Otub2+/+ and Otub2–/– mice. (H, I) Representative H&E (H) and PAS/AB (I) staining of colons from Otub2+/+ and Otub2–/– mice. Scale bar = 50 um. Data in D, E and G are shown as mean +/- SEM. ns, no significant difference. *p < .05, **p < .01. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/39358938), licensed under a CC-BY license. Not internally tested by Novus Biologicals.	
Colonic expression OTUB2 is downregulated in colitis. (A) Representative immunohistochemical staining of OTUB2 in control and UC colon samples. Scale bar = 50 um. (B) Representative immunohistochemical staining of OTUB2 in colon samples from control and DSS□treated C57BL/6 mice. Control mice received regular drinking water. Mice in the DSS group were given 2% DSS for 8 days, followed by regular drinking water for 2 days. Scale bar = 50 um. (C, D) Representative immunoblots (C) and relative quantification (D) of OTUB2 protein in colon samples from control and DSS□treated C57BL/6 mice. (E) Relative mRNA levels of Otub2 in colon samples from control and DSS□treated C57BL/6 mice were analysed by qRT□PCR. (F) Western blot analysis of OTUB2 protein abundance in colon samples from Otub2+/+ and Otub2–/– mice. (G) The representative image (left) and length (right) of colons from Otub2+/+ and Otub2–/– mice. (H, I) Representative H&E (H) and PAS/AB (I) staining of colons from Otub2+/+ and Otub2–/– mice. Scale bar = 50 um. Data in D, E and G are shown as mean +/- SEM. ns, no significant difference. *p < .05, **p < .01. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/39358938), licensed under a CC-BY license. Not internally tested by Novus Biologicals.	

OTUB2 deficiency diminishes MCAO-induced neuronal loss and neuroinflammation.(A) Representative immunofluorescence staining of OTUB2 (red) and NeuN (green) in the brain of C57BL/6 mice. Scale bar, 20 μm. (B) On day 2 after MCAO, the ischemic cerebral hemisphere of Otub2+/+ and Otub2−/− mice was analyzed by Western blot with indicated antibodies. (C, D) The relative expression of NeuN (C) and PSD95 (D) was quantified after normalization to GAPDH. Unpaired Student's t test, n = 4/group, biological replicates. (E, F) Representative immunofluorescence staining (E) and quantification (F) of NeuN+ cells in the ischemic penumbra on day 2 after MCAO. Scale bar, 100 μm. Unpaired Student's t test, n = 5/group, biological replicates. (G) Representative immunofluorescence staining of Iba1+ cells in the ischemic penumbra on day 2 after MCAO. Scale bar, 100 μm. (H) Representative z-stack images of Iba1+ cells in the ischemic penumbra on day 2 after MCAO. Scale bar, 5 μm. (I–M) Forty-eight hours after MCAO, the relative transcription of Il1b (I), Il6 (J), Tnf (K), Ccl2 (L), and Cxcl10 (M) in the ischemic cerebral hemisphere was determined by qRT-PCR. Mann–Whitney U test (I) and Unpaired Student's t test (J–M), n = 5–7/group, biological replicates. Data in (C, D, I–M) show the mean +/- SEM. Source data are available online for this figure. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/40021931), licensed under a CC-BY license. Not internally tested by Novus Biologicals.	
Colonic expression OTUB2 is downregulated in colitis. (A) Representative immunohistochemical staining of OTUB2 in control and UC colon samples. Scale bar = 50 um. (B) Representative immunohistochemical staining of OTUB2 in colon samples from control and DSS□treated C57BL/6 mice. Control mice received regular drinking water. Mice in the DSS group were given 2% DSS for 8 days, followed by regular drinking water for 2 days. Scale bar = 50 um. (C, D) Representative immunoblots (C) and relative quantification (D) of OTUB2 protein in colon samples from control and DSS□treated C57BL/6 mice. (E) Relative mRNA levels of Otub2 in colon samples from control and DSS□treated C57BL/6 mice were analysed by qRT□PCR. (F) Western blot analysis of OTUB2 protein abundance in colon samples from Otub2+/+ and Otub2–/– mice. (G) The representative image (left) and length (right) of colons from Otub2+/+ and Otub2–/– mice. (H, I) Representative H&E (H) and PAS/AB (I) staining of colons from Otub2+/+ and Otub2–/– mice. Scale bar = 50 um. Data in D, E and G are shown as mean +/- SEM. ns, no significant difference. *p < .05, **p < .01. Image collected and cropped by CiteAb from the following open publication (https://pubmed.ncbi.nlm.nih.gov/39358938), licensed under a CC-BY license. Not internally tested by Novus Biologicals.	

Publications

Du X, Xu J, Mei F et al. Deubiquitination of RIPK2 by OTUB2 augments NOD2 signalling and protective effects in intestinal inflammation. Clinical and translational medicine 2024-10-03 [PMID: 39358938]

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