NLRP3/NALP3 (human) monoclonal antibody (Nalpy3-b)

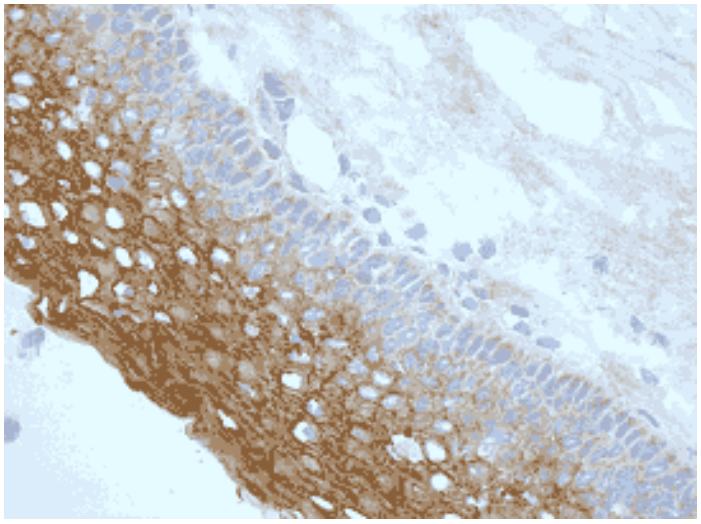
This antibody is covered by our Worry-Free Guarantee.

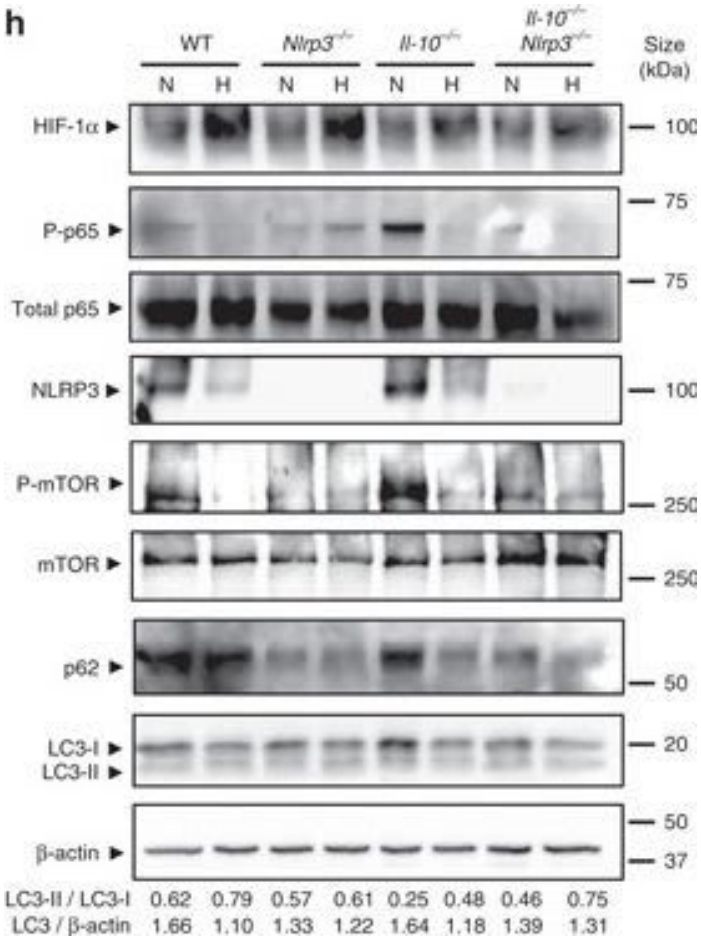
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ALX-804-819-C100	100µg

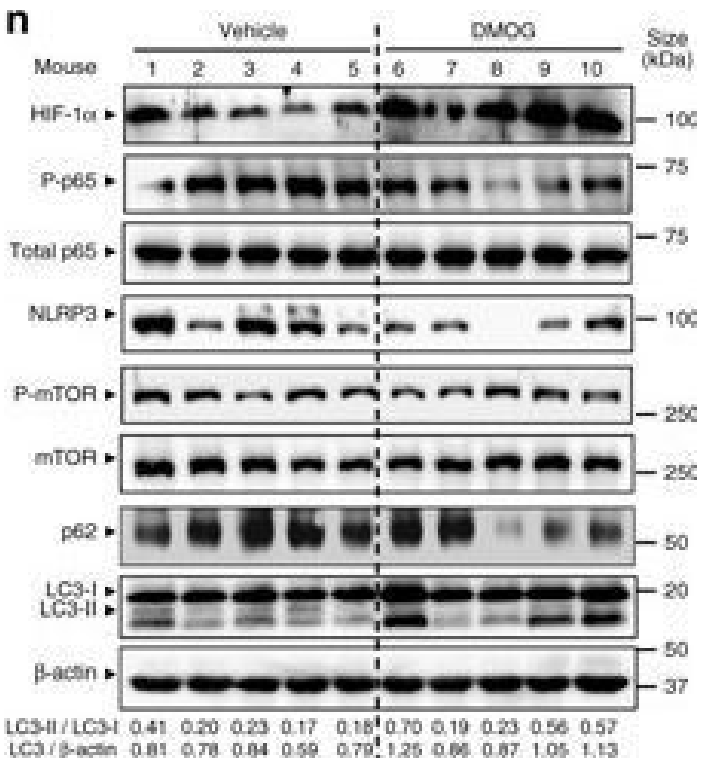
Manuals, SDS & CofA View Online »





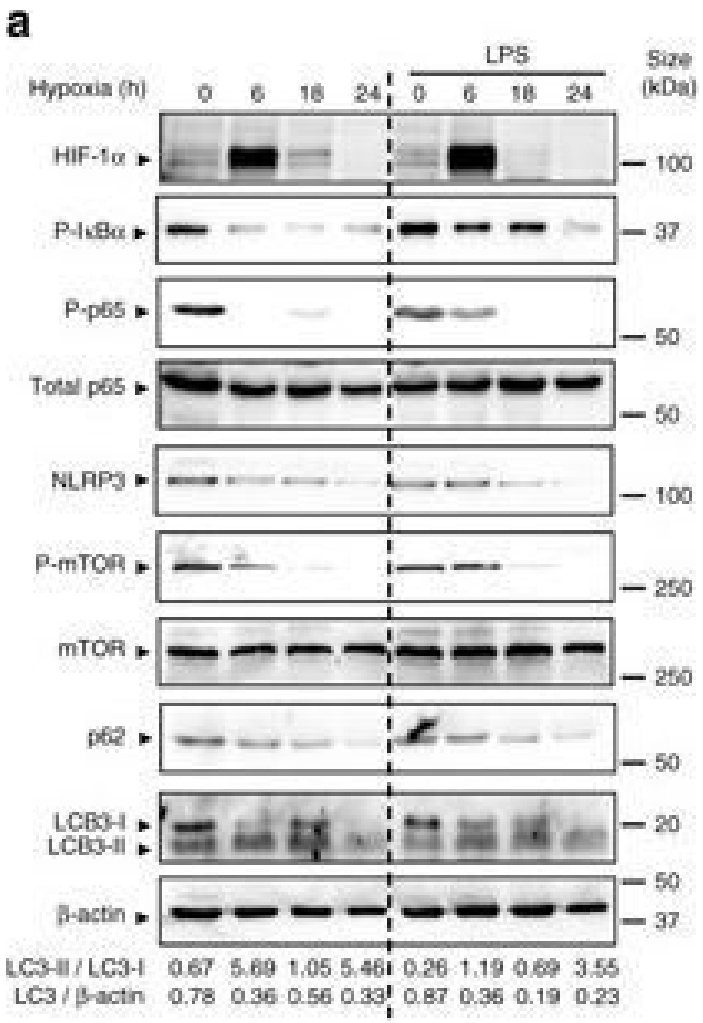
Hypoxia reduces inflammatory processes and induces autophagy in Il-10−/− mice. a, b, c, d, e, f and g WT, Nrlp3−/−,Il-10−/−, and Il-10−/−Nrlp3−/−double knockout mice were subjected to normoxia (N, 21% O2) or hypoxia (H, 8% O2). After 18 h, mice were killed and colon biopsies were collected. Statistical analysis was performed using one-way ANOVA followed by Tukey's post-test. Results represent mean + s.e.m., WT mice under normoxia: n = 5, WT mice under hypoxia: n = 5; Nrlp3−/− mice under normoxia: n = 4; Nrlp3−/− mice under hypoxia: n = 5; Il-10−/− mice under hypoxia: n = 4; Il-10−/− mice under hypoxia: n = 4; Il-10−/−Nrlp3−/− mice under normoxia: n = 7; Il-10−/−Nrlp3−/− mice under hypoxia: n = 9, *P < 0.05; **P < 0.01; ***P < 0.001. h Total protein was isolated and western blot performed. LC3-I and LC3-II bands were quantified, and autophagy was measured by variations in the ratio of LC3-II/LC3-I and the total amount of LC3 (LC3-I plus LC3-II) relative to β-actin

Image collected and cropped by CiteAb under a CC-BY license from the following publication: Hypoxia ameliorates intestinal inflammation through NLRP3/mTOR downregulation and autophagy activation. *Nat Commun* (2017)

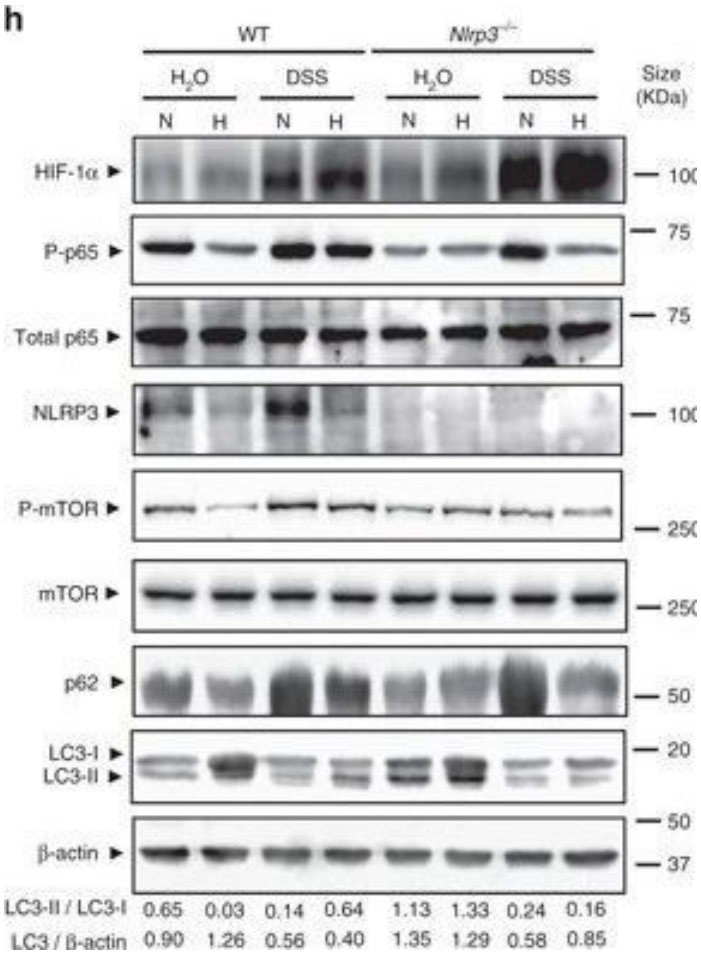


DMOG reduces inflammation and induces autophagy in DSS-treated mice. a, b, c, d, e and f WT mice were administered with 2% DSS for 5 days and co-administered with 8 mg DMOG injected intraperitoneally every second day. Weight loss was monitored during the course of treatment a. After a recovery period, mice were killed at day 9 colon biopsies were collected and colon length was measured b. Mucosal damage was assessed by colonoscopy c and murine endoscopic score of colitis severity (MEICS) was calculated d. H&E staining of distal colon sections displayed a significant decrease in barrier breakdown and inflammatory infiltrate in DMOG-treated mice e. Scale bar, 100 μm. Total histological score at day 9 was calculated as the sum of epithelial damage and infiltration score f. g, h, i, j, k, l and m distal colon biopsies were collected and transcript analysis was performed. Statistical analysis was performed using Student t-test.Results represent mean + s.e.m., DSS-treated mice administered with PBS: n = 5; DSS-treated mice administered with DMOG: n = 5; *P < 0.05; **P < 0.01. n Total protein was isolated and Western blot performed. LC3-I and LC3-II bands were quantified, and autophagy was measured by variations in the ratio of LC3-II/LC3-I and the total amount of LC3 (LC3-I plus LC3-II) relative to β-actin

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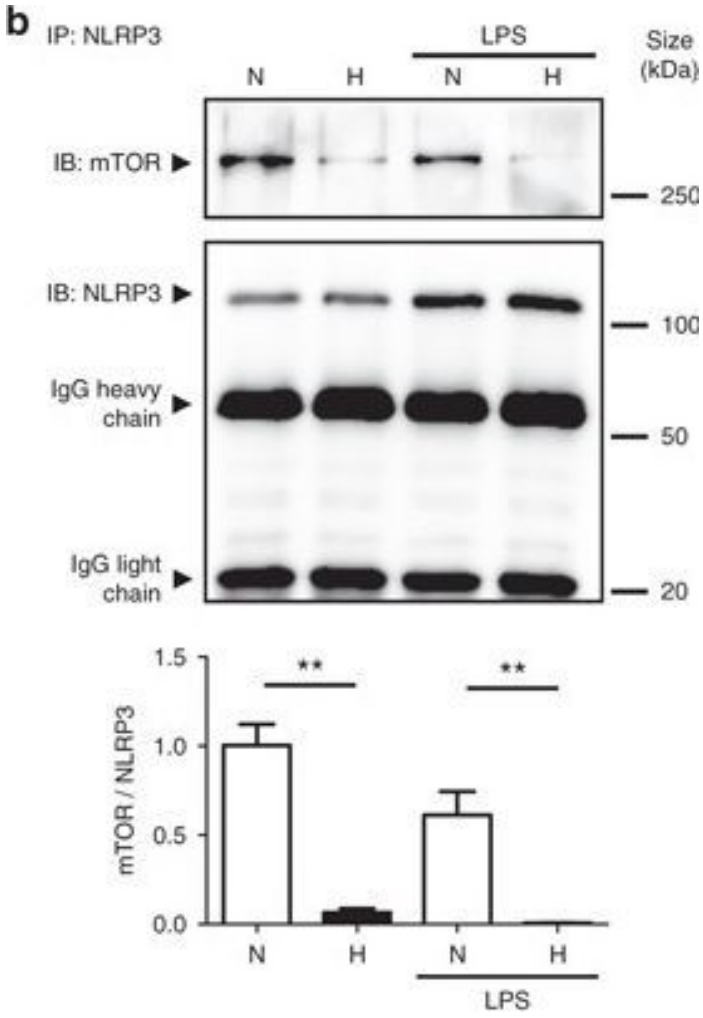


Hypoxia reduces inflammatory signaling pathways and NLRP3 expression and induces autophagy in IECs. a HT-29 cells were subjected to normoxia and hypoxia at the indicated times in the absence or presence of 10 μg/ml LPS. Autophagy was measured by variations in the ratio of LC3-II/LC3-I and the total amount of LC3 (LC3-I plus LC3-II) relative to β-actin. Results are representative of two independent experiments. b, c and d HT-29 cells were subjected to normoxia and hypoxia for the indicated periods in the absence or presence of 10 μg/ml LPS, followed by transcript analysis. Statistical analysis was performed using one-way ANOVA followed by Tukey's post-test. Results represent mean + s.e.m. of two independent experiments done in triplicate, *P < 0.05; **P < 0.01; ***P < 0.001; ns not significant. e HT-29 cells were subjected to normoxia or hypoxia in the presence and absence of 10 μg/ml LPS and 20 μM of MG132. Results are representative of two independent experiments. f and g Putative binding sites for HIF-1α and NF-κB were found in the p62 f and NLRP3 g promoters using Genomatix software tools. Numbers under the boxes indicate the distance from the transcription start site. HT-29 cells were subjected to normoxia (21% O2) or hypoxia (0.2% O2) for 6 h and 24 h. ChIP analysis was performed using antibodies against HIF-1α and NF-κB for immunoprecipitation. PCR was performed using the promoter-specific primers



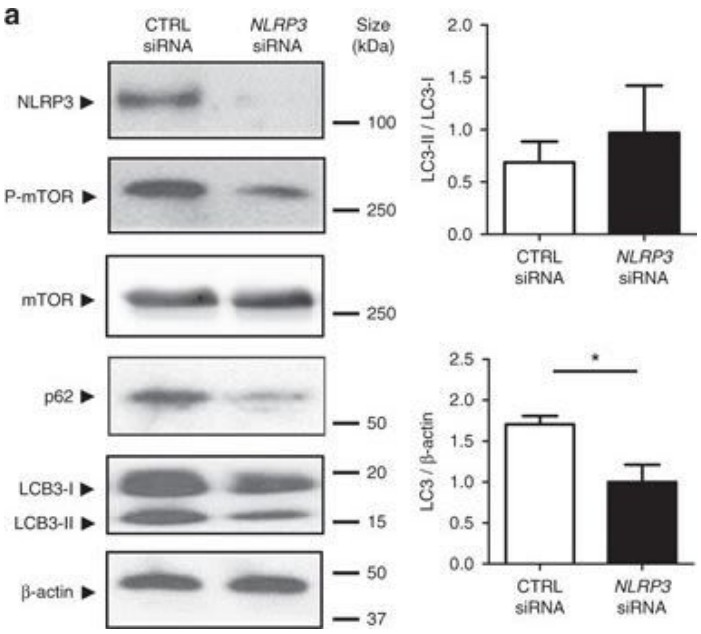
Hypoxia reduces inflammatory processes and induces autophagy in the DSS mouse model of colitis. a, b, c, d, e, f and g WT and Nrlp3−/− mice were administered with DSS or DSS-free water and subjected to normoxia (N, 21% O2) or hypoxia (H, 8% O2). After 18 h, mice were killed and colon biopsies were collected. Statistical analysis was performed using one-way ANOVA followed by Tukey's post-test. Results represent mean + s.e.m., WT mice under normoxia: n = 5, WT mice under normoxia: n = 5, DSS-treated WT mice under normoxia: n = 6; Nrlp3−/− mice under normoxia: n = 5; DSS-treated Nrlp3−/− mice under normoxia: n = 6; WT mice under hypoxia: n = 4, DSS-treated WT mice under hypoxia: n = 5; Nrlp3−/− mice under hypoxia: n = 3; DSS-treated Nrlp3−/− mice under hypoxia: n = 3, *P < 0.05; **P < 0.01; ***P < 0.001. h Total protein was isolated and western blot performed. LC3-I and LC3-II bands were quantified, and autophagy was measured by variations in the ratio of LC3-II/LC3-I and the total amount of LC3 (LC3-I plus LC3-II) relative to β-actin

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NLRP3 regulates autophagy through an inflammasome-independent mechanism via direct binding to mTOR. a HT-29 cells were transfected with NLRP3-specific siRNA or negative control siRNA using Lipofectamine RNAiMAX. Forty-eight hours after transfection, total protein was isolated and western blot performed. Quantification of the ratio of LC3-II/LC3-I and the total amount of LC3 (LC3-I plus LC3-II) relative to β-actin is presented. Statistical analysis was performed using Student t-test. Results represent mean + s.e.m. of three independent experiments, *P < 0.05. b HT-29 cells were subjected to normoxia (21% O2) or hypoxia (0.2% O2) for 24 h in the presence and absence of LPS. Co-IP using NLRP3 antibody was performed, followed by anti-mTOR immunoblotting. Quantification relative to cells subjected to normoxia in the absence of LPS after normalization to NLRP3 is presented. Statistical analysis was performed using one-way ANOVA followed by Tukey's post-test. Results represent mean of three independent experiments + s.e.m., **P < 0.01

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NLRP3 regulates autophagy through an inflammasome-independent mechanism via direct binding to mTOR. a HT-29 cells were transfected with NLRP3-specific siRNA or negative control siRNA using Lipofectamine RNAiMAX. Forty-eight hours after transfection, total protein was isolated and western blot performed. Quantification of the ratio of LC3-II/LC3-I and the total amount of LC3 (LC3-I plus LC3-II) relative to β-actin is presented. Statistical analysis was performed using Student t-test. Results represent mean + s.e.m. of three independent experiments, *P < 0.05. b HT-29 cells were subjected to normoxia (21% O2) or hypoxia (0.2% O2) for 24 h in the presence and absence of LPS. Co-IP using NLRP3 antibody was performed, followed by anti-mTOR immunoblotting. Quantification relative to cells subjected to normoxia in the absence of LPS after normalization to NLRP3 is presented. Statistical analysis was performed using one-way ANOVA followed by Tukey's post-test. Results represent mean of three independent experiments + s.e.m., **P < 0.01

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Handling & Storage

Handling	Avoid freeze/thaw cycles.
Short Term Storage	+4°C
Long Term Storage	-20°C
Shipping	Blue Ice

Regulatory Status

RUO - Research Use Only

Product Details

Alternative Name	NACHT-, LRR- and PYD-containing protein 3, CIAS1, Cryopyrin, Cold autoinflammatory syndrome 1 protein, PYPAF1, PYRIN-containing APAF1-like protein 1
Application	IHC (FS), IP, WB
Clone	Nalpy3-b
Formulation	Liquid. In PBS containing 0.02% sodium azide.
Host	Mouse
Immunogen	Recombinant human NLRP3/NALP3 (NACHT-, LRR- and PYD-containing protein 3) (pyrin domain).
Isotype	IgG1
Recommendation Dilutions/Conditions	Immunoprecipitation (1:50)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 concentrated hybridoma tissue culture supernatant.
Species Reactivity	Human
Specificity	Detects endogenous protein by IP and WB.
UniProt ID	Q96P20

Worry-free Guarantee

This antibody is covered by our Worry-Free Guarantee .

Last modified: May 29, 2024

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