

Product Datasheet

NALP4 Antibody - BSA Free NB100-56156

Unit Size: 0.05 ml

Store at 4C short term. Aliquot and store at -20C long term. Avoid freeze-thaw cycles.

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NB100-56156

NALP4 Antibody - BSA Free

Product Information	
Unit Size	0.05 ml
Concentration	This product is unpurified. The exact concentration of antibody is not quantifiable.
Storage	Store at 4C short term. Aliquot and store at -20C long term. Avoid freeze-thaw cycles.
Clonality	Polyclonal
Preservative	0.02% Sodium Azide
Isotype	IgG
Purity	Unpurified
Buffer	Whole antisera

Product Description	
Description	Novus Biologicals Rabbit NALP4 Antibody - BSA Free (NB100-56156) is a polyclonal antibody validated for use in IHC and WB. Anti-NALP4 Antibody: Cited in 6 publications. All Novus Biologicals antibodies are covered by our 100% guarantee.
Host	Rabbit
Gene ID	147945
Gene Symbol	NLRP4
Species	Human
Specificity/Sensitivity	NALP4
Immunogen	A synthetic peptide corresponding to amino acids 139-157 (FAPKETGKQPRTVIIQGPQ) of human NALP4/PAN2/PYPAF4 was used as immunogen; GenBank no. gi 17064172 gb AAL35293.1 AF442488_1

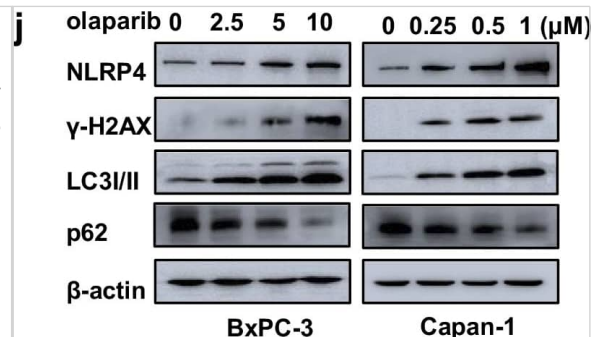
Product Application Details	
Applications	Western Blot, Immunohistochemistry-Paraffin, Immunohistochemistry
Recommended Dilutions	Western Blot 1:1000-1:2000, Immunohistochemistry, Immunohistochemistry-Paraffin 1:1000-1:5000

Images

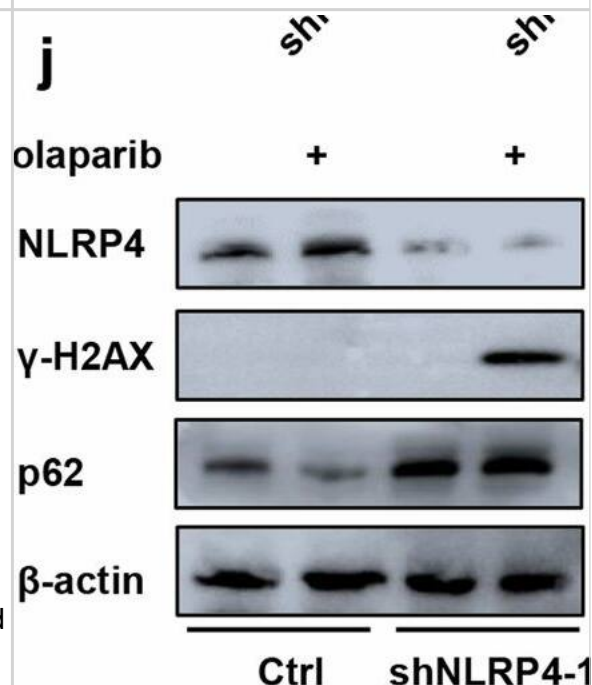
Immunohistochemistry: NALP4 Antibody [NB100-56156] - Formalin-fixed, paraffin-embedded tissue cores from a human breast cancer microarray stained for Smac/Diablo using NB100-56156 at 1:2000. Hematoxylin-eosin counterstain. Differential Smac/DIABLO staining is seen between patient samples.



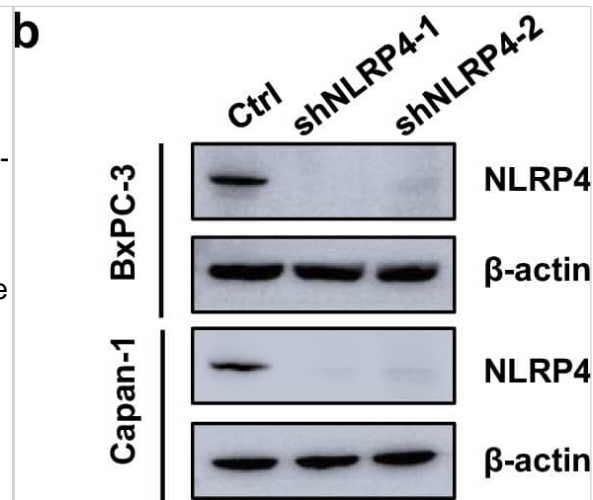
NLRP4 expression induced olaparib resistance in pancreatic cancer cells. a, b MTS assays were performed to calculate the IC₅₀ of olaparib in the indicated cells. c, d The indicated cells were treated with DMSO or olaparib (5 μ M olaparib for BxPC-3 cells and 500 nM olaparib for Capan-1 cells). MTS assays were performed to investigate the effect of NLRP4 on olaparib resistance. The analysis of significant differences was performed with one-way ANOVA. *** $p < 0.001$. e The indicated cells were treated with DMSO or olaparib (5 μ M olaparib for BxPC-3 cells and 500 nM olaparib for Capan-1 cells). Colony formation assays were performed to investigate the effect of NLRP4 on olaparib resistance. The analysis of significant differences was performed with Student's t test. *** $p < 0.001$. ** $p < 0.01$. f, g Flow cytometry results for annexin V/7-ADD staining in the indicated cell lines after exposure to DMSO or olaparib (5 μ M olaparib for BxPC-3 cells and 500 nM olaparib for Capan-1 cells). The analysis of significant differences was performed with Student's t test. *** $p < 0.001$. h Control or NLRP4-knockdown Capan-1 cells were treated with DMSO or olaparib (500 nM) for RNA sequencing and were subjected to GO pathway enrichment analysis. i Co-IP experiments were conducted in NLRP4 OE cells, followed by LC-MS/MS analysis. j Control or NLRP4-knockdown BxPC-3 and Capan-1 cells were treated with the indicated dose of olaparib for 48 h. Western blotting was performed with the indicated antibodies. Image collected and cropped by CiteAb from the following open publication (<https://www.nature.com/articles/s41419-024-06984-0>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



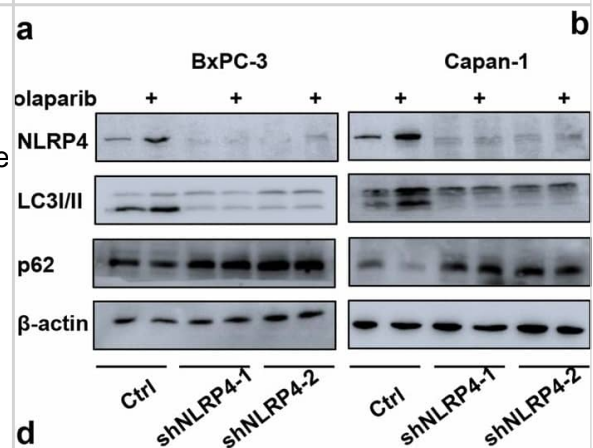
Pancreatic tumors with higher NLRP4 expression are resistant to olaparib *in vivo*. a BxPC-3 xenografts obtained from mice in different groups treated with DMSO or olaparib (50 mg/kg per day). b Quantification of the weight of the tumors in different groups treated with DMSO or olaparib (50 mg/kg per day). The analysis of significant differences was performed with Student's t test. ** $p < 0.01$. c Quantification of tumor volume on the last day in different groups treated with DMSO or olaparib (50 mg/kg per day). The analysis of significant differences was performed with Student's t test. $p < 0.05$, ** $p < 0.01$. d Growth curves of cells treated with DMSO or olaparib. The analysis of significant differences was performed with one-way ANOVA. ** $p < 0.01$, *** $p < 0.001$. e Tumor doubling time in different groups treated with DMSO or olaparib (50 mg/kg per day). The analysis of significant differences was performed with Student's t test. * $p < 0.05$. f–h Tumors were subjected to immunological staining to detect the indicated marker. f Representative IHC micrographs. Scale bar, 100 μ m. g–i The histological score (H score) of the indicated markers was quantified. The analysis of significant differences was performed with Student's t test. * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$. j Western blotting analysis was performed for the indicated antibodies. k Kaplan–Meier survival curves for the specified groups. The p value was calculated by the log rank test. *** $p < 0.001$. Image collected and cropped by CiteAb from the following open publication (<https://www.nature.com/articles/s41419-024-06984-0>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



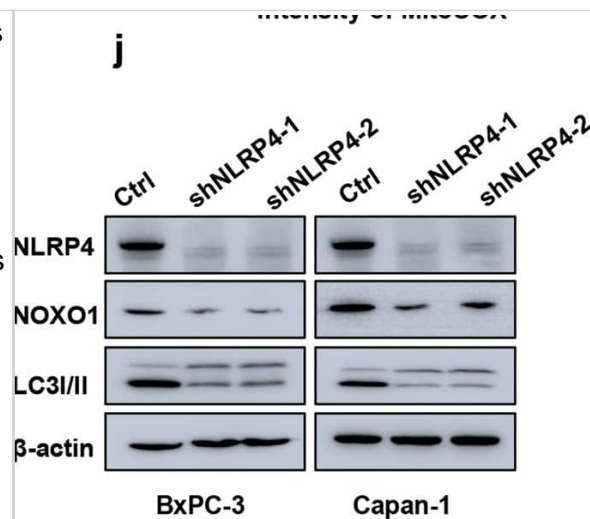
NLRP4 expression promotes pancreatic cancer cell proliferation. a Volcano plot showing the suppression of NLRP4 led to a greater susceptibility to olaparib in pancreatic cancer cell lines. b Immunoblot of NLRP4 in control and NLRP4-knockdown BxPC-3 and Capan-1 cells. BxPC-3 and Capan-1 cells were stably transfected with shNC, shNLRP4-1 and shNLRP4-2, and then MTS assays (c, d) and colony formation assays (e, f) were performed. *** $p < 0.001$. ** $p < 0.01$. The analysis of significant differences was performed with Student's t test. g–j Control and NLRP4-knockdown BxPC-3 and Capan-1 cells were injected into the left flank of nude mice. Tumor volumes were measured every 3 days. Tumors were harvested on day 21, photographed and weighed. Tumor weights are shown in (h), BxPC-3 tumor volumes are shown in (i), and Capan-1 tumor volumes are shown in (j). Data are shown as the mean \pm SD ($n = 5$). *** $p < 0.001$. The analysis of significant differences was performed with Student's t test. *** $p < 0.001$. ** $p < 0.01$. k, l Flow cytometry analysis of annexin V/7-ADD staining in the indicated cell lines. Data are presented as the mean \pm SD from three independent experiments. The analysis of significant differences was performed with Student's t test. Image collected and cropped by CiteAb from the following open publication (<https://www.nature.com/articles/s41419-024-06984-0>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



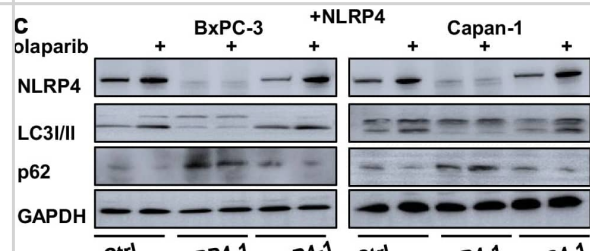
Pancreatic cancer cells with high NLRP4 expression exhibit enhanced levels of autophagy in response to olaparib exposure. a–c Control or NLRP4-knockdown BxPC-3 and Capan-1 cells were treated with DMSO or olaparib (5 μ M olaparib for BxPC-3 cells and 500 nM olaparib for Capan-1 cells) for 48 h. Western blotting analysis and quantification were performed. The analysis of significant differences was performed with Student's t test. *** $p < 0.01$. d–f Effect of NLRP4 on the levels of autophagic flux. The mRFP-GFP-LC3 plasmid was transfected into the indicated cells for 24 h, and then the cells were treated with olaparib (5 μ M olaparib for BxPC-3 cells and 500 nM olaparib for Capan-1 cells) for 48 h. Representative images were obtained by laser scanning confocal microscopy. The average fluorescence intensity of autophagic lysosomes (yellow dots in the merged images) and autophagic lysosomes (red in the merged images) in individual cells was quantified. Scale bar, 5 μ m. The analysis of significant differences was performed with Student's t test. *** $p < 0.001$. g–i TEM-based ultrastructure analysis (autophagosomes) in the indicated cells. The analysis of significant differences was performed with Student's t test. *** $p < 0.001$. Image collected and cropped by CiteAb from the following open publication (<https://www.nature.com/articles/s41419-024-06984-0>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



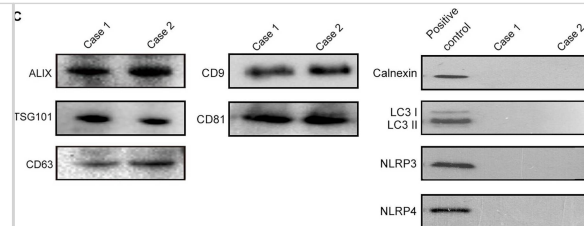
NLRP4 induces mitochondrial ROS generation in pancreatic cancer cells in response to olaparib exposure. **a** Cells treated with DMSO or olaparib (5 μ M olaparib for BxPC-3 cells and 500 nM olaparib for Capan-1 cells) for 48 h were incubated with an ROS indicator. Representative images were obtained. **b, c** Cells treated with DMSO or olaparib (5 μ M olaparib for BxPC-3 cells and 500 nM olaparib for Capan-1 cells) for 48 h were incubated with an ROS indicator, and fluorescence intensity was assessed with flow cytometry. The analysis of significant differences was performed with Student's t test. *** $p < 0.001$. **d, e** Cells treated with DMSO or olaparib (5 μ M olaparib for BxPC-3 cells and 500 nM olaparib for Capan-1 cells) for 48 h were incubated with JC-1 working solution, and fluorescence intensity was assessed with flow cytometry. The analysis of significant differences was performed with Student's t test. *** $p < 0.001$. **f–h** Cells treated with olaparib (5 μ M olaparib for BxPC-3 cells and 500 nM olaparib for Capan-1 cells) plus DMSO or MitoQ for 48 h were incubated with MitoSOX. Representative images were obtained, and the fluorescence intensity was assessed with flow cytometry. The analysis of significant differences was performed with Student's t test. *** $p < 0.001$. **i** Changes in ROS-related genes in control and NLRP4-knockdown Capan-1 cells after olaparib treatment (500 nM olaparib for Capan-1 cells). Scale bar, 5 μ m. **j** Western blotting analysis was performed for the indicated antibodies. Image collected and cropped by CiteAb from the following open publication (<https://www.nature.com/articles/s41419-024-06984-0>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



Complementation of NLRP4 rescues DNA repair defects and autophagy levels in NLRP4-knockdown pancreatic cancer cells and results in olaparib resistance. **a** Representative immunofluorescence micrographs of γ -H2AX foci in the indicated cells with or without olaparib treatment (5 μ M olaparib for BxPC-3 cells and 500 nM olaparib for Capan-1 cells). **b** TEM-based ultrastructure analysis (autophagosomes) in the indicated cells. **c** Cells were treated with DMSO or olaparib (5 μ M olaparib for BxPC-3 cells and 500 nM olaparib for Capan-1 cells) for 48 h. Western blotting analysis was performed for the indicated antibodies. **d, e** Cells treated with DMSO or olaparib (5 μ M olaparib for BxPC-3 cells and 500 nM olaparib for Capan-1 cells) for 48 h were incubated with an ROS indicator, and fluorescence intensity was assessed with flow cytometry. The analysis of significant differences was performed with one-way ANOVA. *** $p < 0.001$. **f, g** The indicated cells were treated with DMSO or olaparib (5 μ M olaparib for BxPC-3 cells and 500 nM olaparib for Capan-1 cells). MTS assays were performed to investigate the effect of NLRP4 on olaparib resistance. The analysis of significant differences was performed with one-way ANOVA. *** $p < 0.001$. **h** Flow cytometry results for annexin V/7-ADD staining in the indicated cell lines after exposure to DMSO or olaparib (5 μ M olaparib for BxPC-3 cells and 500 nM olaparib for Capan-1 cells). The analysis of significant differences was performed with Student's t test. *** $p < 0.001$. **i** Schematic model showing that NLRP4 renders pancreatic cancer resistant to PARPi through the promotion of the DNA damage response and ROS-induced autophagy. Image collected and cropped by CiteAb from the following open publication (<https://www.nature.com/articles/s41419-024-06984-0>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



Identification of salivary exosomes in periodontitis patients and normal subjects. (A) Transmission electron microscopy of exosomes isolated from human saliva. Scale bar: 100 nm. (B) Exosomes concentration and size distribution by NanoSight analysis of human saliva. (C) Immunoblotting showed the exosomal membrane markers (ALIX, TSG101 CD63, CD9 and CD81), the intracellular protein Calnexin, the marker of autophagosome LC3 and markers of inflammasome (NLRP3 and NLRP4) in exosomes isolated from the saliva of one normal subject (case 01) and one periodontitis patient (case 02). Positive control for Calnexin was TE1 cells, and positive control for LC3 was TE1 cells after starvation for 6 h. Positive control for NLRP3 and NLRP4 was THP-1 cells. Image collected and cropped by CiteAb from the following open publication (<https://pubmed.ncbi.nlm.nih.gov/30923536>), licensed under a CC-BY license. Not internally tested by Novus Biologicals.



Publications

Xiao M, Yang J, Dong M et al. NLRP4 renders pancreatic cancer resistant to olaparib through promotion of the DNA damage response and ROS-induced autophagy *Cell Death Dis* 2024-08-26 [PMID: 39187531]

Wu YL, Pan LH, Yi ZJ et al. c-Myb Dominates TBK1-Mediated Endotoxin Tolerance in Kupffer Cells by Negatively Regulating DTX4 *Journal of immunology research* 2023-03-31 [PMID: 37032653] (WB, Human)

Ruscitti P, Berardicurti O, Di Cola I et al. The hyper-expression of NLRP4 characterises the occurrence of macrophage activation syndrome assessing STING pathway in adult onset Still's disease *Clinical and Experimental Immunology* 2022-02-02 [PMID: 35467709]

Yu J, Lin Y, Xiong X et al. Detection of Exosomal PD-L1 RNA in Saliva of Patients With Periodontitis *Front Genet.* 2019-03-13 [PMID: 30923536] (WB, Human)

Yamamoto Mayuko, Kondo Eisaku, Takeuchi Makoto et al. miR-155, a Modulator of FOXO3a Protein Expression, Is Underexpressed and Cannot Be Upregulated by Stimulation of HOZOT, a Line of Multifunctional Treg. *PLoS One.* 2011-01-01 [PMID: 21304824] (WB, Human)

Jounai N, Kobiyama K, Shiina M et al. NLRP4 negatively regulates autophagic processes through an association with beclin1. *J Immunol.* 2011-02-01 [PMID: 21209283] (WB)

Details:

NALP4/PAN2/PYPAF4.1. WB: Endogenous Beclin1 protein was immunoprecipitated from lysed HeLa cells then subject to WB with the NLRP4 (IMG-5743) antibody, Fig 1C.



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NB7160	Goat anti-Rabbit IgG (H+L) Secondary Antibody [HRP]
NBP2-24891	Rabbit IgG Isotype Control

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