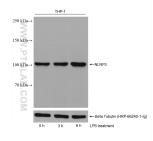
For Research Use Only

NLRP3 Monoclonal antibody Catalog Number:68102-1-Ig Featured Product 111 Publications

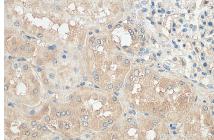
proteintech® Antibodies | ELISA kits | Proteins www.ptglab.com

Basic Information	Catalog Number: 68102-1-lg	GenBank Accession Number: NM_001079821	Purification Method: Protein A purification	
	Size: 150ul , Concentration: 2000 ug/ml by	GenelD (NCBI):	CloneNo.: 3H1A7	
	Nanodrop; Source: Mouse	Full Name: NLR family, pyrin domain containing 3	Recommended Dilutions: WB 1:2000-1:10000 IHC 1:50-1:500	
	Isotype: IgG2a	Calculated MW: 118 kDa		
	Immunogen Catalog Number: AG26289	Observed MW: 110 kDa		
Applications	Tested Applications: WB, IHC, ELISA		Positive Controls:	
	Cited Applications: WB, IHC, IF, CoIP		WB : THP-1 cells, LPS treated THP-1 cells IHC : human kidney tissue,	
	Species Specificity: human			
	Cited Species: human, chicken, bovine			
	Note-IHC: suggested antigen r TE buffer pH 9.0; (*) Alternativ retrieval may be performed w buffer pH 6.0	vely, antigen		
Background Information	NALP3, also named as C1orf7, CIAS1 component of the NLRP3 inflammaso function as an inducer of apoptosis. It activator of NF-kappa-B signaling.NA RELA/NF-KB p65. Also inhibits transce triggers including bacterial or viral ir NLRP3 are the cause of familial cold a urticaria. Defects in NLRP3 are the c	LP3 inhibits TNF-alpha induced activa riptional activity of RELA. NALP3 activa nfection which leads to processing and autoinflammatory syndrome type 1 (F e of Muckle-Wells syndrome (MWS) wh ause of chronic infantile neurologic cu multisystem inflammatory disease (N	unity and inflammation. NALP3 may is complex may function as an upstream tion and nuclear translocation of ates caspase-1 in response to a number o release of IL1B and IL18. Defects in CAS1) which also known as familial cold nich is urticaria-deafness-amyloidosis taneous and articular syndrome (CINCA)	
Background Information	NALP3, also named as C1orf7, CIAS1 component of the NLRP3 inflammaso function as an inducer of apoptosis. If activator of NF-kappa-B signaling.NA RELA/NF-KB p65. Also inhibits transci triggers including bacterial or viral ir NLRP3 are the cause of familial cold a utticaria. Defects in NLRP3 are a caus syndrome. Defects in NLRP3 are the c which also known as neonatal onset the MW of 106-118 kDa and 75-83 kD	me, plays a crucial role in innate imm t interacts selectively with ASC and thi LP3 inhibits TNF-alpha induced activa riptional activity of RELA. NALP3 activa fection which leads to processing and autoinflammatory syndrome type 1 (Fr e of Muckle-Wells syndrome (MWS) wh ause of chronic infantile neurologic cu multisystem inflammatory disease (N	unity and inflammation. NALP3 may is complex may function as an upstream tion and nuclear translocation of ates caspase-1 in response to a number o release of IL1B and IL18. Defects in CAS1) which also known as familial cold nich is urticaria-deafness-amyloidosis taneous and articular syndrome (CINCA)	
	NALP3, also named as C1orf7, CIAS1 component of the NLRP3 inflammaso function as an inducer of apoptosis. In activator of NF-kappa-B signaling.NA RELA/NF-KB p65. Also inhibits transce triggers including bacterial or viral ir NLRP3 are the cause of familial cold urticaria. Defects in NLRP3 are a caus syndrome. Defects in NLRP3 are the co which also known as neonatal onset the MW of 106-118 kDa and 75-83 kD	me, plays a crucial role in innate imm t interacts selectively with ASC and thi LP3 inhibits TNF-alpha induced activa riptional activity of RELA. NALP3 activa fection which leads to processing and autoinflammatory syndrome type 1 (Fr e of Muckle-Wells syndrome (MWS) wi ause of chronic infantile neurologic cu multisystem inflammatory disease (N a(PMID: 17164409, 34680443).	unity and inflammation. NALP3 may is complex may function as an upstream tion and nuclear translocation of ates caspase-1 in response to a number o release of IL1B and IL18. Defects in CAS1) which also known as familial cold nich is urticaria-deafness-amyloidosis taneous and articular syndrome (CINCA) OMID). NLRP3 has some isoforms with	
	NALP3, also named as C1orf7, CIAS1 component of the NLRP3 inflammaso function as an inducer of apoptosis. In activator of NF-kappa-B signaling.NA RELA/NF-KB p65. Also inhibits transci triggers including bacterial or viral in NLRP3 are the cause of familial cold a urticaria. Defects in NLRP3 are a caus syndrome. Defects in NLRP3 are the c which also known as neonatal onset the MW of 106-118 kDa and 75-83 kD Author Pub Qiuyuan Liu 359	me, plays a crucial role in innate imm t interacts selectively with ASC and thi LP3 inhibits TNF-alpha induced activa riptional activity of RELA. NALP3 activa fection which leads to processing and autoinflammatory syndrome type 1 (Fi e of Muckle-Wells syndrome (MWS) wi ause of chronic infantile neurologic cu multisystem inflammatory disease (N a(PMID: 17164409, 34680443). med ID Journal	unity and inflammation. NALP3 may is complex may function as an upstream tion and nuclear translocation of ates caspase-1 in response to a number o release of IL1B and IL18. Defects in CAS1) which also known as familial cold nich is urticaria-deafness-amyloidosis taneous and articular syndrome (CINCA) OMID). NLRP3 has some isoforms with Application WB	
	NALP3, also named as C1orf7, CIAS1 component of the NLRP3 inflammaso function as an inducer of apoptosis. If activator of NF-kappa-B signaling.NA RELA/NF-KB p65. Also inhibits transci triggers including bacterial or viral ir NLRP3 are the cause of familial cold a utticaria. Defects in NLRP3 are ta causs syndrome. Defects in NLRP3 are the c which also known as neonatal onset the MW of 106-118 kDa and 75-83 kD Author Pub Qiuyuan Liu 359 Lu Bai 359	me, plays a crucial role in innate imm t interacts selectively with ASC and thi LP3 inhibits TNF-alpha induced activa riptional activity of RELA. NALP3 active fection which leads to processing and autoinflammatory syndrome type 1 (Fri e of Muckle-Wells syndrome (MWS) with ause of chronic infantile neurologic cu multisystem inflammatory disease (N a(PMID: 17164409, 34680443). med ID Journal 07203 Inflamm Bowel Dis	unity and inflammation. NALP3 may is complex may function as an upstream tion and nuclear translocation of ates caspase-1 in response to a number of release of IL18 and IL18. Defects in CAS1) which also known as familial colo ich is urticaria-deafness-amyloidosis taneous and articular syndrome (CINCA) OMID). NLRP3 has some isoforms with Application WB	
Notable Publications	NALP3, also named as C1orf7, CIAS1 component of the NLRP3 inflammaso function as an inducer of apoptosis. It activator of NF-kappa-B signaling.NA RELA/NF-KB p65. Also inhibits transci triggers including bacterial or viral ir NLRP3 are the cause of familial cold a utticaria. Defects in NLRP3 are ta causs syndrome. Defects in NLRP3 are the c which also known as neonatal onset the MW of 106-118 kDa and 75-83 kD Author Pub Qiuyuan Liu 359 Lu Bai 359	me, plays a crucial role in innate imm t interacts selectively with ASC and thi LP3 inhibits TNF-alpha induced activa riptional activity of RELA. NALP3 activa fection which leads to processing and autoinflammatory syndrome type 1 (Fri e of Muckle-Wells syndrome (MWS) wh ause of chronic infantile neurologic cu multisystem inflammatory disease (N a(PMID: 17164409, 34680443). med ID Journal 07203 Inflamm Bowel Dis 10846 Oxid Med Cell Longev 87632 Int Immunopharmacol er shipment.	unity and inflammation. NALP3 may is complex may function as an upstream tion and nuclear translocation of ates caspase-1 in response to a number o release of IL1B and IL18. Defects in CAS1) which also known as familial cold ich is urticaria-deafness-amyloidosis taneous and articular syndrome (CINCA) OMID). NLRP3 has some isoforms with Application WB	
Notable Publications	NALP3, also named as C1orf7, CIAS1 component of the NLRP3 inflammaso function as an inducer of apoptosis. If activator of NF-kappa-B signaling.NA RELA/NF-KB p65. Also inhibits transci triggers including bacterial or viral ir NLRP3 are the cause of familial cold a urticaria. Defects in NLRP3 are a cause syndrome. Defects in NLRP3 are the co which also known as neonatal onset the MW of 106-118 kDa and 75-83 kD Author Pub Qiuyuan Liu 359 Lu Bai 359 Zhengyuan Huang 399 Storage: Storage Storage Buffer: PBS with 0.02% sodium azide and 50	me, plays a crucial role in innate imm t interacts selectively with ASC and thi LP3 inhibits TNF-alpha induced activa riptional activity of RELA. NALP3 activa fection which leads to processing and autoinflammatory syndrome type 1 (Fri e of Muckle-Wells syndrome (MWS) wh ause of chronic infantile neurologic cu multisystem inflammatory disease (N a(PMID: 17164409, 34680443). med ID Journal 07203 Inflamm Bowel Dis 10846 Oxid Med Cell Longev 87632 Int Immunopharmacol er shipment.	unity and inflammation. NALP3 may is complex may function as an upstream tion and nuclear translocation of ates caspase-1 in response to a number o release of IL1B and IL18. Defects in CAS1) which also known as familial cold ich is urticaria-deafness-amyloidosis taneous and articular syndrome (CINCA) OMID). NLRP3 has some isoforms with Application WB	

Selected Validation Data







Non-treated THP-1 cells and LPS treated THP-1 cells were subjected to SDS PAGE followed by western blot with 68102-1-1g (NLRP3 antibody) at dilution of 1:5000 incubated at room temperature for 1.5 hours. The membrane was stripped and re-blotted with Beta Tubulin antibody as loading control.

Immunohistochemical analysis of paraffinembedded human kidney tissue slide using 68102-1-lg (NLRP3 antibody) at dilution of 1:200 (under 10x lens). Heat mediated antigen retrieval with Tris-EDTA buffer (pH 9.0). Immunohistochemical analysis of paraffinembedded human kidney tissue slide using 68102-1-Ig (NLRP3 antibody) at dilution of 1:200 (under 40x lens). Heat mediated antigen retrieval with Tris-EDTA buffer (pH 9.0).