

**NALP3 / NLRP3 Antibody (N-Terminus)**  
**Rabbit Polyclonal Antibody**  
**Catalog # ALS13309**

**Specification**

---

**NALP3 / NLRP3 Antibody (N-Terminus) - Product Information**

Application	IF
Primary Accession	<a href="#">Q96P20</a>
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Calculated MW	118kDa KDa

**NALP3 / NLRP3 Antibody (N-Terminus) - Additional Information**

**Gene ID** 114548

**Other Names**

NACHT, LRR and PYD domains-containing protein 3, Angiotensin/vasopressin receptor AII/AVP-like, Caterpillar protein 1.1, CLR1.1, Cold autoinflammatory syndrome 1 protein, Cryopyrin, PYRIN-containing APAF1-like protein 1, NLRP3, C1orf7, CIAS1, NALP3, PYPAF1

**Target/Specificity**

Human NLRP3. Immunogenic peptide shares 75% homology with the mouse sequence. The antibody has not been specifically tested with mouse samples at this time. Cross-reactivity or lack thereof therefore cannot be guaranteed.

**Reconstitution & Storage**

Short term 4°C, long term aliquot and store at -20°C, avoid freeze thaw cycles. Store undiluted.

**Precautions**

NALP3 / NLRP3 Antibody (N-Terminus) is for research use only and not for use in diagnostic or therapeutic procedures.

**NALP3 / NLRP3 Antibody (N-Terminus) - Protein Information**

**Name** NLRP3 {ECO:0000303|PubMed:17907925, ECO:0000312|HGNC:HGNC:16400}

**Function**

Sensor component of the NLRP3 inflammasome, which mediates inflammasome activation in response to defects in membrane integrity, leading to secretion of inflammatory cytokines IL1B and IL18 and pyroptosis (PubMed: [16407889](http://www.uniprot.org/citations/16407889), PubMed: [18403674](http://www.uniprot.org/citations/18403674), PubMed: [18604214](http://www.uniprot.org/citations/18604214), PubMed: [23582325](http://www.uniprot.org/citations/23582325), PubMed: [25686105](http://www.uniprot.org/citations/25686105), PubMed: [27929086](http://www.uniprot.org/citations/27929086), PubMed: [28656979](http://www.uniprot.org/citations/28656979))

target="\_blank">28656979</a>, PubMed:<a href="http://www.uniprot.org/citations/28847925" target="\_blank">28847925</a>, PubMed:<a href="http://www.uniprot.org/citations/30487600" target="\_blank">30487600</a>, PubMed:<a href="http://www.uniprot.org/citations/30612879" target="\_blank">30612879</a>, PubMed:<a href="http://www.uniprot.org/citations/31086327" target="\_blank">31086327</a>, PubMed:<a href="http://www.uniprot.org/citations/31086329" target="\_blank">31086329</a>, PubMed:<a href="http://www.uniprot.org/citations/31189953" target="\_blank">31189953</a>, PubMed:<a href="http://www.uniprot.org/citations/33231615" target="\_blank">33231615</a>, PubMed:<a href="http://www.uniprot.org/citations/34133077" target="\_blank">34133077</a>, PubMed:<a href="http://www.uniprot.org/citations/34341353" target="\_blank">34341353</a>, PubMed:<a href="http://www.uniprot.org/citations/34512673" target="\_blank">34512673</a>, PubMed:<a href="http://www.uniprot.org/citations/36442502" target="\_blank">36442502</a>). In response to pathogens and other damage-associated signals that affect the integrity of membranes, initiates the formation of the inflammasome polymeric complex composed of NLRP3, CASP1 and PYCARD/ASC (PubMed:<a href="http://www.uniprot.org/citations/16407889" target="\_blank">16407889</a>, PubMed:<a href="http://www.uniprot.org/citations/18403674" target="\_blank">18403674</a>, PubMed:<a href="http://www.uniprot.org/citations/27432880" target="\_blank">27432880</a>, PubMed:<a href="http://www.uniprot.org/citations/28847925" target="\_blank">28847925</a>, PubMed:<a href="http://www.uniprot.org/citations/31189953" target="\_blank">31189953</a>, PubMed:<a href="http://www.uniprot.org/citations/33231615" target="\_blank">33231615</a>, PubMed:<a href="http://www.uniprot.org/citations/34133077" target="\_blank">34133077</a>, PubMed:<a href="http://www.uniprot.org/citations/34341353" target="\_blank">34341353</a>, PubMed:<a href="http://www.uniprot.org/citations/36142182" target="\_blank">36142182</a>, PubMed:<a href="http://www.uniprot.org/citations/36442502" target="\_blank">36442502</a>). Recruitment of pro-caspase-1 (proCASP1) to the NLRP3 inflammasome promotes caspase-1 (CASP1) activation, which subsequently cleaves and activates inflammatory cytokines IL1B and IL18 and gasdermin-D (GSDMD), promoting cytokine secretion and pyroptosis (PubMed:<a href="http://www.uniprot.org/citations/23582325" target="\_blank">23582325</a>, PubMed:<a href="http://www.uniprot.org/citations/28847925" target="\_blank">28847925</a>, PubMed:<a href="http://www.uniprot.org/citations/31189953" target="\_blank">31189953</a>, PubMed:<a href="http://www.uniprot.org/citations/33231615" target="\_blank">33231615</a>, PubMed:<a href="http://www.uniprot.org/citations/34133077" target="\_blank">34133077</a>, PubMed:<a href="http://www.uniprot.org/citations/34341353" target="\_blank">34341353</a>). Activation of NLRP3 inflammasome is also required for HMGB1 secretion; stimulating inflammatory responses (PubMed:<a href="http://www.uniprot.org/citations/22801494" target="\_blank">22801494</a>). Under resting conditions, ADP-bound NLRP3 is autoinhibited (PubMed:<a href="http://www.uniprot.org/citations/35114687" target="\_blank">35114687</a>). NLRP3 activation stimuli include extracellular ATP, nigericin, reactive oxygen species, crystals of monosodium urate or cholesterol, amyloid-beta fibers, environmental or industrial particles and nanoparticles, such as asbestos, silica, aluminum salts, cytosolic dsRNA, etc (PubMed:<a href="http://www.uniprot.org/citations/16407889" target="\_blank">16407889</a>, PubMed:<a href="http://www.uniprot.org/citations/18403674" target="\_blank">18403674</a>, PubMed:<a href="http://www.uniprot.org/citations/18604214" target="\_blank">18604214</a>, PubMed:<a href="http://www.uniprot.org/citations/19414800" target="\_blank">19414800</a>, PubMed:<a href="http://www.uniprot.org/citations/23871209" target="\_blank">23871209</a>). Almost all stimuli trigger intracellular K(+) efflux (By similarity). These stimuli lead to membrane perturbation and activation of NLRP3 (By similarity). Upon activation, NLRP3 is transported to microtubule organizing center (MTOC), where it is unlocked by NEK7, leading to its relocalization to dispersed trans-Golgi network (dTGN) vesicle membranes and formation of an active inflammasome complex (PubMed:<a href="http://www.uniprot.org/citations/36442502" target="\_blank">36442502</a>). Associates with dTGN vesicle membranes by binding to phosphatidylinositol 4-phosphate (PtdIns4P) (PubMed:<a href="http://www.uniprot.org/citations/30487600" target="\_blank">30487600</a>, PubMed:<a href="http://www.uniprot.org/citations/34554188" target="\_blank">34554188</a>). Shows ATPase activity (PubMed:<a href="http://www.uniprot.org/citations/17483456" target="\_blank">17483456</a>).

## Cellular Location

Cytoplasm, cytosol. Inflammasome. Cytoplasm, cytoskeleton, microtubule organizing center. Golgi apparatus membrane. Endoplasmic reticulum {ECO:0000250|UniProtKB:Q8R4B8}. Mitochondrion. Secreted. Nucleus {ECO:0000250|UniProtKB:Q8R4B8} Note=In macrophages, under resting conditions, mainly located in the cytosol and on membranes of various organelles, such as endoplasmic reticulum, mitochondria and Golgi: forms an inactive double-ring cage that is primarily localized on membranes (By similarity). Upon activation, NLRP3 is transported to microtubule organizing center (MTOC), where it is unlocked by NEK7, leading to its relocation to dispersed trans-Golgi network (dTGN) vesicle membranes for the formation of an active inflammasome complex (By similarity). Recruited to dTGN vesicle membranes by binding to phosphatidylinositol 4- phosphate (PtdIns4P) (PubMed:30487600). After the induction of pyroptosis, inflammasome specks are released into the extracellular space where they can further promote IL1B processing and where they can be engulfed by macrophages (PubMed:24952504). Phagocytosis induces lysosomal damage and inflammasome activation in the recipient cells (PubMed:24952504). In the Th2 subset of CD4(+) helper T-cells, mainly located in the nucleus (By similarity). Nuclear localization depends upon KPNA2 (By similarity). In the Th1 subset of CD4(+) helper T-cells, mainly cytoplasmic (By similarity). {ECO:0000250|UniProtKB:Q8R4B8, ECO:0000269|PubMed:24952504, ECO:0000269|PubMed:30487600}

### **Tissue Location**

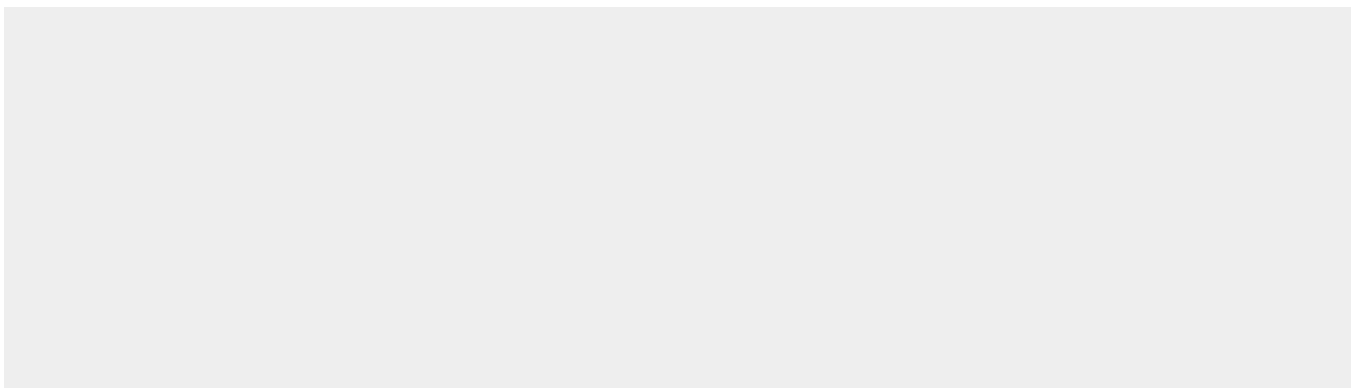
Predominantly expressed in macrophages (PubMed:33231615, PubMed:34133077). Also expressed in dendritic cells, B- and T-cells (at protein level) (PubMed:11786556, PubMed:17164409) Expressed in LPS-treated granulocytes, but not in resting cells (at protein level) (PubMed:17164409). Expression in monocytes is very weak (at protein level) (PubMed:17164409). Expressed in stratified non-keratinizing squamous epithelium, including oral, esophageal and ectocervical mucosa and in the Hassall's corpuscles in the thymus Also, detected in the stratified epithelium covering the bladder and ureter (transitional mucosa) (at protein level) (PubMed:17164409) Expressed in lung epithelial cells (at protein level) (PubMed:23229815). Expressed in chondrocytes (PubMed:12032915) Expressed at low levels in resting osteoblasts (PubMed:17907925)

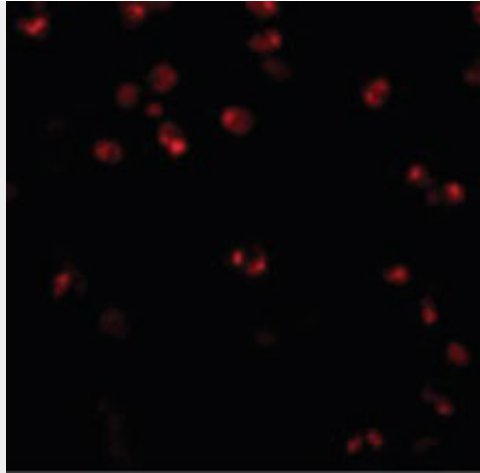
### **NALP3 / NLRP3 Antibody (N-Terminus) - Protocols**

Provided below are standard protocols that you may find useful for product applications.

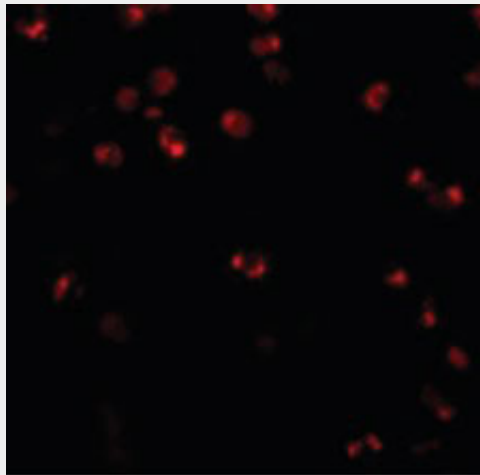
- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

### **NALP3 / NLRP3 Antibody (N-Terminus) - Images**





Immunofluorescence of NALP3 in K562 cells with NALP3 antibody at 20 ug/ml.



Immunofluorescence of NALP3 in K562 cells with NALP3 antibody at 20 ug/ml.

### **NALP3 / NLRP3 Antibody (N-Terminus) - Background**

May function as an inducer of apoptosis. Interacts selectively with ASC and this complex may function as an upstream activator of NF-kappa-B signaling. Inhibits TNF-alpha induced activation and nuclear translocation of RELA/NF-KB p65. Also inhibits transcriptional activity of RELA. Activates caspase-1 in response to a number of triggers including bacterial or viral infection which leads to processing and release of IL1B and IL18.

### **NALP3 / NLRP3 Antibody (N-Terminus) - References**

Hoffman H.M., et al. Nat. Genet. 29:301-305(2001).  
Aganna E., et al. Arthritis Rheum. 46:2445-2452(2002).  
Manji G.A., et al. J. Biol. Chem. 277:11570-11575(2002).  
O'Connor W. Jr., et al. J. Immunol. 171:6329-6333(2003).  
Ota T., et al. Nat. Genet. 36:40-45(2004).