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NALP3 Antibody
Catalog # ASC10999

Specification

NALP3 Antibody - Product Information

Application	**IF**
Primary Accession	Q96P20
Other Accession	NP_899632, 114548
Reactivity	**Human, Mouse**
Host	**Rabbit**
Clonality	**Polyclonal**
Isotype	**IgG**
Application Notes	**NALP3 antibody can be used for detection of NALP3 by Western blot at 1 µg/mL. Antibody can also be used for Immunocytochemistry starting at 2 µg/mL. For immunofluorescence start at 20 µg/mL.**

NALP3 Antibody - Additional Information

Gene ID **114548**

Target/Specificity
NALP3 antibody was raised against a 16 amino acid synthetic peptide from near the amino terminus of human NALP3.

The immunogen is located within amino acids 120 - 170 of NALP3.

Reconstitution & Storage
NALP3 antibody can be stored at 4℃ for three months and -20℃, stable for up to one year. As with all antibodies care should be taken to avoid repeated freeze thaw cycles. Antibodies should not be exposed to prolonged high temperatures.

Precautions
NALP3 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

NALP3 Antibody - 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, PubMed:18403674, PubMed:18604214, PubMed:23582325, PubMed:25686105, PubMed:<a href="http://www.uniprot.org/citations/27929086"

target="_blank">27929086, PubMed:28656979, PubMed:28847925, PubMed:30487600, PubMed:30612879, PubMed:31086327, PubMed:31086329, PubMed:31189953, PubMed:33231615, PubMed:34133077, PubMed:34341353, PubMed:34512673, PubMed:36442502). 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:16407889, PubMed:18403674, PubMed:27432880, PubMed:28847925, PubMed:31189953, PubMed:33231615, PubMed:34133077, PubMed:34341353, PubMed:36142182, PubMed:36442502). 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:23582325, PubMed:28847925, PubMed:31189953, PubMed:33231615, PubMed:34133077, PubMed:34341353). Activation of NLRP3 inflammasome is also required for HMGB1 secretion; stimulating inflammatory responses (PubMed:22801494). Under resting conditions, ADP-bound NLRP3 is autoinhibited (PubMed:35114687). 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:16407889, PubMed:18403674, PubMed:18604214, PubMed:19414800, PubMed:23871209). 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:36442502, PubMed:39173637). Associates with dTGN vesicle membranes by binding to phosphatidylinositol 4-phosphate (PtdIns4P) (PubMed:30487600, PubMed:34554188). Shows ATPase activity (PubMed:17483456).

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 relocalization to dispersed trans-Golgi network (dTGN) vesicle membranes for the formation of an active inflammasome complex (PubMed:39173637) 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, ECO:0000269|PubMed:39173637}

Tissue Location

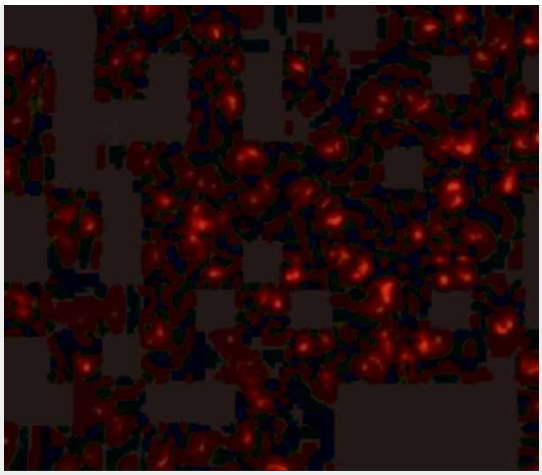
Predominantly expressed in macrophages (PubMed:33231615, PubMed:34133077). Also expressed in dendritic cells, B- and T-cells (at protein level) (PubMed:11786556, PubMed:17164409) Expressed in LPS-treated granulocytes, but not in resting cells (at protein level) (PubMed:17164409). Expression in monocytes 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 Antibody - Protocols

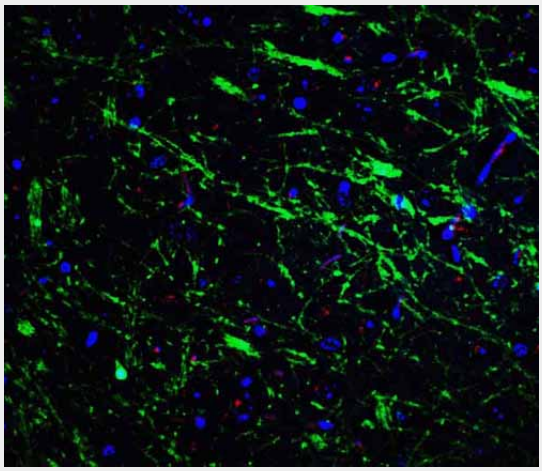
Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

NALP3 Antibody - Images



Immunofluorescence of IKK alpha in Jurkat cells with IKK alpha antibody at 10 µg/mL.



Immunofluorescence of PINK1 in mouse brain tissue with PINK1 Antibodyat 20 µg/mL.

NALP3 Antibody - Background

NALP3 Antibody: NALP3, a member of Nod-like receptors, has a crucial role in inflammation and immunity and may be a proximal sensor of cellular stress and danger signals. NALP3 forms a caspase-1 activating molecular complex termed the inflammasome. The inflammasome allows activation of IL-1β, the key player of the inflammation and fever. NALP3 gene encodes a pyrin like protein which contains a pyrin domain, a nucleotide binding site (NBS) domain, and a leucine rich repeat (LRR) motif. NALP3 protein interacts with apoptosis associated speck like protein containing a CARD and may function as an inducer of apoptosis and an activator of NF-κB signaling. Defects in NALP3 have been associated with familial cold autoinflammatory syndrome (FCAS), Muckle-Wells syndrome (MWS), chronic infantile neurological cutaneous and articular (CINCA) syndrome, and neonatal-onset multisystem inflammatory disease (NOMID).

NALP3 Antibody - References

Chamaillard M, Girardin SE, Viala J, et al. Nods, Nalps and Naip: intracellular regulators of bacterial-induced inflammation. Cell. Microbiol.2003; 5:581-92.
O'Connor W Jr., Hartin JA, Zhu X, et al. Cutting edge: CIAS1/cryopyrin/PYPAF1/NALP3/CATERPILLER 1.1 is an inducible inflammatory mediator with NF-kappa B suppressive properties. J. Immunol.2003; 171:6329-33.
Kanneganti TD, Ozoren N, Body-Malapal M, et al. Bacterial RNA and small antiviral compounds activate caspase-1 through cryopyrin/Nalp. Nature2006; 440:233-6.
Agostini L, Martinon F, Burns K, et al. NALP3 forms an IL-1beta-processing inflammasome with

increased activity in Muckle-Wells autoinflammatory disorder. Immunity 2004; 20:319-25.