NLRP3 Polyclonal Antibody

catalog number: E-AB-93354

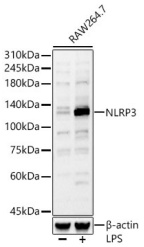
Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

Description

Reactivity	Human;Mouse
Immunogen	Recombinant fusion protein of mouse NLRP3
Host	Rabbit
Isotype	IgG
Purification	Affinity purification
Buffer	Phosphate buffered solution, pH 7.4, containing 0.05% stabilizer and 50% glycerol.

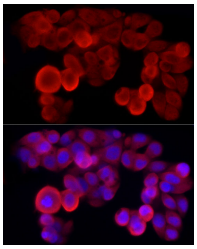
Applications	Recommended Dilution
WB	1:500-1:2000
IF	1:50-1:200

Data

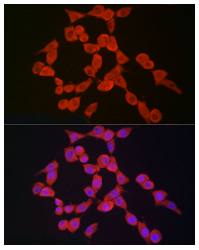


Western blot analysis of RAW264.7 using NLRP3 Polyclonal Antibody at 1:500 dilution.Raw264.7 cells were treated by LPS (1 μg/ml) at 37℃ for 8 hours.

Observed-MW:110 kDa



Immunofluorescence analysis of HepG2 cells using NLRP3 Polyclonal Antibody at dilution of 1:50 (40x lens). Blue: DAPI for nuclear staining.



Immunofluorescence analysis of NIH/3T3 cells using NLRP3 Polyclonal Antibody at dilution of 1:50 (40x lens). Blue: DAPI for nuclear staining.

Preparation & Storage

Storage	Store at -20°C Valid for 12 months. Avoid freeze / thaw cycles.
Shipping	The product is shipped with ice pack,upon receipt,store it immediately at the temperature recommended.

Background

Toll-free: 1-888-852-8623
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This gene encodes a pyrin-like protein containing a pyrin domain, a nucleotide-binding site (NBS) domain, and a leucin e-rich repeat (LRR) motif. This protein interacts with the apoptosis-associated speck-like protein PYCARD/ASC, which contains a caspase recruitment domain, and is a member of the NLRP3 inflammasome complex. This complex functions as an upstream activator of NF-kappaB signaling, and it plays a role in the regulation of inflammation, the immune response, and apoptosis. The SARS-CoV 3a protein, a transmembrane pore-forming viroporin, has been shown to activate the NLRP3 inflammasome via the formation of ion channels in macrophages. Mutations in this gene are associated with familial cold autoinflammatory syndrome (FCAS), Muckle-Wells syndrome (MWS), chronic infantile neurological cutaneous and articular (CINCA) syndrome, neonatal-onset multisystem inflammatory disease (NOMID), keratoendotheliitis fugax hereditarian, and deafness, autosomal dominant 34, with or without inflammation. Multiple alternatively spliced transcript variants encoding distinct isoforms have been identified for this gene. Alternative 5' UTR structures are suggested by available data; however, insufficient evidence is available to determine if all of the represented 5' UTR splice patterns are biologically valid.

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