

# NLRP3 Polyclonal Antibody

Catalog Number:E-AB-93354

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

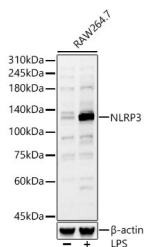
## Description

<b>Reactivity</b>	Human,Mouse
<b>Immunogen</b>	Recombinant fusion protein of mouse NLRP3
<b>Host</b>	Rabbit
<b>Isotype</b>	IgG
<b>Purification</b>	Affinity purification
<b>Conjugation</b>	Unconjugated
<b>Formulation</b>	PBS with 0.05% proclin300,50% glycerol,pH7.3.

## Applications Recommended Dilution

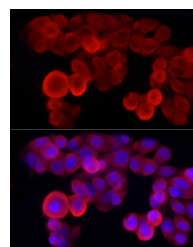
<b>WB</b>	1:500-1:2000
<b>IF</b>	1:50-1:200

## Data

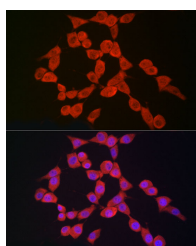


Western blot analysis of RAW264.7 using NLRP3 Polyclonal Antibody at 1:500 dilution. Raw264.7 cells were treated by LPS (1  $\mu$ g/ml) at 37°C for 8 hours.

**Observed Mw:110KDa**



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. Avoid freeze/thaw cycles.

## Background

This gene encodes a pyrin-like protein containing a pyrin domain, a nucleotide-binding site (NBS) domain, and a leucine-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

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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 hereditaria, 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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