NLRP1 Protein, Human (His)

Cat. No.:	HY-P702189
Synonyms:	NLRP1; NACHT; LRR and PYD domains-containing protein 1; Caspase recruitment domain-containing protein 7; Death effector filament-forming ced-4-like apoptosis protein; Nucleotide-binding domain and caspase recruitment domain
Species:	Human
Source:	E. coli
Accession:	Q9C000 (L1379-K1462)
Gene ID:	/
Molecular Weight:	

PROPERTIES

Appearance	Solution.
Formulation	Supplied as a 0.22 μm filtered solution of 50 mM Tris-HCl, pH7.5, 200 mM NaCl, 20% glycerol.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	Please use rapid thawing with running water to thaw the protein.
Storage & Stability	Stored at -80°C for 1 year. It is stable at -20°C for 3 months after opening. It is recommended to freeze aliquots at -80°C for extended storage. Avoid repeated freeze-thaw cycles.
Shipping	Shipping with dry ice.

DESCRIPTION

Background

The NLRP1 protein serves as the pivotal sensor component within the NLRP1 inflammasome, orchestrating inflammasome activation in response to diverse pathogen-associated signals. This activation culminates in pyroptosis, a critical cellular defense mechanism. Inflammasomes, supramolecular complexes that assemble in the cytosol, play indispensable roles in innate immunity and inflammation. Functioning as a recognition receptor (PRR), NLRP1 discerns specific pathogens and damage-associated signals, including cleavage by certain human enteroviruses, double-stranded RNA, UV-B irradiation, and Val-boroPro inhibitor. Upon detection, NLRP1 initiates the formation of the inflammasome polymeric complex, consisting of NLRP1, CASP1, and PYCARD/ASC. In response to pathogen-associated cues, the N-terminal portion of NLRP1 undergoes proteasomal degradation, liberating the cleaved C-terminal segment. This C-terminal fragment polymerizes and associates with PYCARD/ASC, instigating the inflammasome complex formation. The NLRP1 inflammasome recruits pro-caspase-1, fostering CASP1 activation, subsequently cleaving and activating inflammatory cytokines IL1B and IL18, along with gasdermin-D (GSDMD), ultimately inducing pyroptosis. In the absence of GSDMD expression, the NLRP1 inflammasome recruits and activates CASP8, leading to the activation of gasdermin-E (GSDME). Activation of the NLRP1 inflammasome is also crucial for HMGB1 secretion, amplifying inflammatory responses. NLRP1 additionally binds ATP, displaying ATPase activity, playing a pivotal role in antiviral immunity and inflammation in the human airway epithelium. It recognizes various pathogen-associated signals, such as HRV-14 and HRV-16 infection, positive-strand RNA viruses, and long dsRNA, acting as a

direct sensor for RNA virus infection. Furthermore, NLRP1 may be activated by muramyl dipeptide (MDP) in a NOD2-dependent manner. UV-B irradiation causing ribosome collisions activates the NLRP1 inflammasome, and it functions as the precursor of the inflammasome, mediating autoproteolytic processing within the FIIND domain to generate N-terminal and C-terminal segments, which associate non-covalently in the absence of pathogens and other damage-associated signals.

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