Data Sheet (Cat.No.TMPH-01738)

TargetMol

NLRP1 Protein, Human, Recombinant (GST)

General Information

Synonyms:	Caspase recruitment domain-containing protein 7;Nucleotide-binding domain and caspase recruitment domain;Death effector filament-forming ced-4-like apoptosis protein;NACHT, LRR and PYD domains-containing protein 1;NLRP1
Protein Construction:	1-146 aa
Species:	Human
Expression Host:	E. coli
Accession:	Q9C000
Molecular Weight:	42.9 kDa (predicted)
AA Sequence:	MPLDPYHSVTWGHAQGSHHFVFGELRVRPILESLRDEPDPDPRPSREGPAGRVGALARGGPEPCDAASPPG GASCAPELARPREDKSAQQAKLEGGTRLCCRCPEESRLVPGGAVSPGDHVLEVSGTRGTCGCRPRRHAGPEL AHS

QC Testing

Biological Activity:	Activity has not been tested. It is theoretically active, but we cannot guarantee it. If you require protein activity, we recommend choosing the eukaryotic expression version first.
Purity:	> 85% as determined by SDS-PAGE.
Endotoxin:	< 1.0 EU/μg of the protein as determined by the LAL method.
Formulation:	If the delivery form is liquid, the default storage buffer is Tris/PBS-based buffer, 5%-50% glycerol. If the delivery form is lyophilized powder, the buffer before lyophilization is Tris/PBS-based buffer, 6% Trehalose, pH 8.0.

Preparation and Storage

Reconstitution:

Reconstitute the lyophilized protein in sterile deionized water. The product concentration should not be less than 100 μg/mL. Before opening, centrifuge the tube to collect powder at the bottom. After adding the reconstitution buffer, avoid vortexing or pipetting for mixing.

Stability & Storage:

Lyophilized powders can be stably stored for over 12 months, while liquid products can be stored for 6-12 months at -80°C. For reconstituted protein solutions, the solution can be stored at -20°C to -80°C for at least 3 months. Please avoid multiple freeze-thaw cycles and store products in aliquots.

Actual storage temperature shall be subject to the COA.

Shipping:

In general, lyophilized powders are shipped with blue ice, while solutions are shipped with dry ice.

Protein Background

Acts as the sensor component of the NLRP1 inflammasome, which mediates inflammasome activation in response to various pathogen-associated signals, leading to subsequent pyroptosis. Inflammasomes are supramolecular complexes that assemble in the cytosol in response to pathogens and other damage-associated signals and play critical roles in innate immunity and inflammation. Acts as a recognition receptor (PRR): recognizes specific pathogens and other damage-associated signals, such as cleavage by human rhinoviruses 14 and 16 (HRV-14 and HRV-16), double-stranded RNA or Val-boroPro inhibitor, and mediates the formation of the inflammasome polymeric complex composed of NLRP1, CASP1 and PYCARD/ASC. In response to pathogen-associated signals, the N-terminal part of NLRP1 is degraded by the proteasome, releasing the cleaved C-terminal part of the protein (NACHT, LRR and PYD domains-containing protein 1, C-terminus), which polymerizes and associates with PYCARD/ASC to initiate the formation of the inflammasome complex: the NLRP1 inflammasome recruits pro-caspase-1 (proCASP1) and promotes caspase-1 (CASP1) activation, which subsequently cleaves and activates inflammatory cytokines IL1B and IL18 and gasdermin-D (GSDMD), leading to pyroptosis. Activation of NLRP1 inflammasome is also required for HMGB1 secretion; the active cytokines and HMGB1 stimulate inflammatory responses. Binds ATP and shows ATPase activity. Plays an important role in antiviral immunity and inflammation in the human airway epithelium. Specifically recognizes a number of pathogen-associated signals: upon infection by human rhinoviruses 14 and 16 (HRV-14 and HRV-16), NLRP1 is cleaved and activated which triggers NLRP1-dependent inflammasome activation and IL18 secretion. Positive-strand RNA viruses such as. Semliki forest virus and long dsRNA activate the NLRP1 inflammasome, triggering IL1B release in a NLRP1-dependent fashion. Acts as a direct sensor for long dsRNA and thus RNA virus infection. May also be activated by muramyl dipeptide (MDP), a fragment of bacterial peptidoglycan, in a NOD2-dependent manner.; Constitutes the precusor of the NLRP1 inflammasome, which mediates autoproteolytic processing within the FIIND domain to generate the N-terminal and C-terminal parts, which are associated non-covalently in absence of pathogens and other damage-associated signals.; Regulatory part that prevents formation of the NLRP1 inflammasome: in absence of pathogens and other damage-associated signals, interacts with the C-terminal part of NLRP1 (NACHT, LRR and PYD domains-containing protein 1, C-terminus), preventing activation of the NLRP1 inflammasome. In response to pathogen-associated signals, this part is ubiquitinated and degraded by the proteasome, releasing the cleaved C-terminal part of the protein, which polymerizes and forms the NLRP1 inflammasome.; Constitutes the active part of the NLRP1 inflammasome. In absence of pathogens and other damage-associated signals, interacts with the N-terminal part of NLRP1 (NACHT, LRR and PYD domains-containing protein 1, N-terminus), preventing activation of the NLRP1 inflammasome. In response to pathogen-associated signals, the N-terminal part of NLRP1 is degraded by the proteasome, releasing this form, which polymerizes and associates with PYCARD/ASC to form of the NLRP1 inflammasome complex: the NLRP1 inflammasome complex then directly recruits pro-caspase-1 (proCASP1) and promotes caspase-1 (CASP1) activation, leading to gasdermin-D (GSDMD) cleavage and subsequent pyroptosis.; It is unclear whether is involved in inflammasome formation. It is not cleaved within the FIIND domain, does not assemble into specks, nor promote IL1B release. However, in an vitro cell-free system, it has been shown to be activated by MDP.

Tel:781-999-4286 E_mail:info@targetmol.com Address:34 Washington Street,Wellesley Hills,MA 02481