Datasheet for ABIN2170333

anti-STS antibody

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Overview

Quantity:	100 µL
Target:	STS
Reactivity:	Human, Mouse, Rat
Host:	Rabbit
Clonality:	Polyclonal
Conjugate:	This STS antibody is un-conjugated
Application:	Western Blotting (WB), ELISA, Flow Cytometry (FACS), Immunofluorescence (Paraffin-embedded Sections) (IF (p)), Immunohistochemistry (Paraffin-embedded Sections) (IHC (p)), Immunohistochemistry (Frozen Sections) (IHC (fro)), Immunofluorescence (Cultured Cells) (IF (cc))

Product Details

Immunogen:	KLH conjugated synthetic peptide derived from human TMS1/ASC
Isotype:	IgG
Cross-Reactivity:	Bacteria, Human, Mouse, Pig, Rat
Predicted Reactivity:	Dog,Cow,Horse,Rabbit
Purification:	Purified by Protein A.

Target Details

Target:	STS
Alternative Name:	Asc (STS Products)

Target Details

Background:	Synonyms: ASC, TMS, TMS1, CARD5, TMS-1, Apoptosis-associated speck-like protein containing a CARD, hASC, Caspase recruitment domain-containing protein 5, PYD and CARD domain-containing protein, Target of methylation-induced silencing 1, PYCARD Background: Functions as key mediator in apoptosis and inflammation. Promotes caspase-mediated apoptosis involving predominantly caspase-8 and also caspase-9 in a probable cell type-specific manner. Involved in activation of the mitochondrial apoptotic pathway, promotes caspase-8-dependent proteolytic maturation of BID independently of FADD in certain cell types and also mediates mitochondrial translocation of BAX and activates BAX-dependent apoptosis coupled to activation of caspase-9, -2 and -3. Involved in macrophage pyroptosis, a caspase-1-dependent inflammatory form of cell death and is the major constituent of the ASC pyroptosome which forms upon potassium depletion and rapidly recruits and activates caspase-1. In innate immune response believed to act as an integral adapter in the assembly of the inflammasome which activates caspase-1 leading to processing and secretion of proinflammatory cytokines. The function as activating adapter in different types of inflammasomes is mediated by the DAPIN and CARD domains and their homotypic interactions. Required for recruitment of caspase-1 to inflammasomes containing certain pattern recognition receptors, such as NLRP2, NLRP3, AIM2 and probably IFI16. In the NLRP1 and NLRC4 inflammasomes seems not be required but facilitates the processing of procaspase-1. In cooperation with NOD2 involved in an inflammasome activated by bacterial muramyl dipeptide leading to caspase-1 activation. May be involved in DDX58-triggered proinflammatory responses and inflammasome activation. Isoform 2 may have a regulating effect on the function as inflammasome adapter. Isoform 3 seems to inhibit inflammasome-mediated maturation of interleukin-1 beta. In collaboration with AIM2 which detects cytosolic double-stranded DNA may also be involved in a caspase-1-independent cell death that involves caspase-8.
Gene ID:	29108
UniProt:	Q9ULZ3
Pathways:	Steroid Hormone Biosynthesis, Activation of Innate immune Response, Cellular Response to Molecule of Bacterial Origin, Positive Regulation of Endopeptidase Activity, Activated T Cell Proliferation

Application Details

Application Notes:	WB: 1:100-1000, FCM: 1:20-100, IHC-P: 1:100-500, IF(IHC-P): 1:50-200 Optimal working dilution should be determined by the investigator.

Application Details

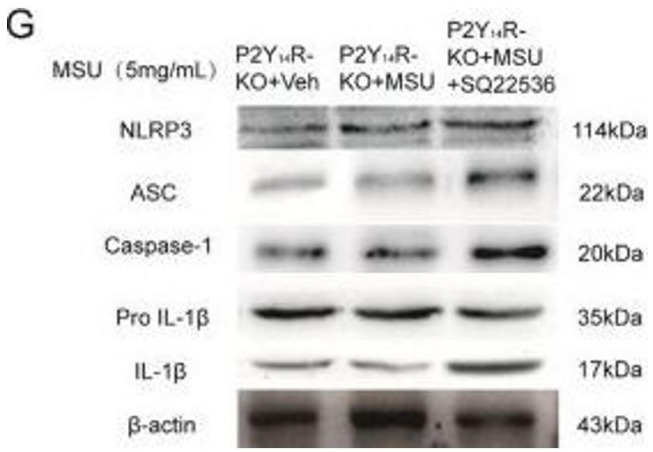
Restrictions:	For Research Use only

Handling

Format:	Liquid
Concentration:	1 μg/μL
Buffer:	0.01M TBS(pH 7.4) with 1 % BSA, 0.02 % Proclin300 and 50 % Glycerol.
Preservative:	Sodium azide
Precaution of Use:	This product contains Sodium azide: a POISONOUS AND HAZARDOUS SUBSTANCE which should be handled by trained staff only.
Storage:	4 °C,-20 °C
Storage Comment:	Shipped at 4°C. Store at -20°C for one year. Avoid repeated freeze/thaw cycles.
Expiry Date:	12 months

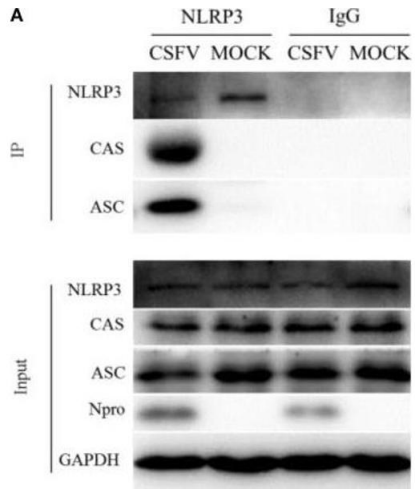
Publications

Product cited in:	Fan, Yuan, Deng, Chen, Xie, Wu, Zhu, Xu, Huang, Yang, Zhang, Chen, Zhao: "Activation of Interleukin-1β Release by the Classical Swine Fever Virus Is Dependent on the NLRP3 Inflammasome, Which Affects Virus Growth in Monocytes." in: **Frontiers in cellular and infection microbiology**, Vol. 8, pp. 225, (2019) (PubMed). Zhang, Liu, Sui, Cai, Liu, Zhu, Yin: "Cortistatin inhibits NLRP3 inflammasome activation of cardiac fibroblasts during sepsis." in: **Journal of cardiac failure**, (2015) (PubMed).



Western Blotting

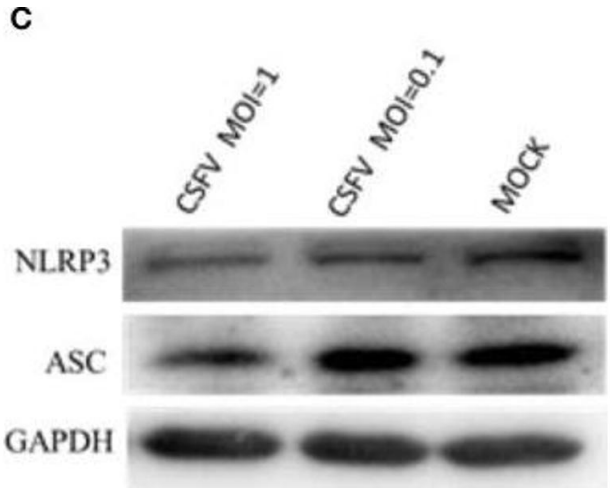
Image 1. Decreased cAMP exaggerated acute gouty arthritis in P2Y14R-KO rats.SQ22536, an adenylate cyclase (AC) inhibitor, was used in our study to reduce cAMP levels in P2Y14R-KO rats. Intra-articular administration of SQ22536 was given to P2Y14R-KO rats prior to MSU model. a The intracellular cAMP level decreased significantly in P2Y14R-KO synovial tissue after SQ22536 treatment by cAMP assay kit (n=6). b cAMP reduction caused by SQ22536 abolished the effective effect of P2Y14R-KO on the joint swelling. Representative photographs to show the swelling of joints are presented. c SQ22536-induced cAMP reduction apparently aggravated the injected ankle joint circumference under MSU challenge in P2Y14R-KO rats (n=6). d SQ22536 treatment exhibited a significant exacerbation of inflammatory cell infiltration in histopathologic evaluation of P2Y14R-KO rat synovial tissues. e The increment of pyroptosis positivity could be observed in the macrophages derived from P2Y14R-KO rat synovium after SQ22536 stimulation by flow cytometry (n=4). f SQ22536 treatment enhanced the colocalization intensity of synovial NLRP3 and ASC in MSU-stimulated P2Y14R-KO rats in immunofluorescence staining. NLRP3 protein was marked with Alexa Fluor 488 (Green). ASC protein was marked with Alexa Fluor 647 (Red). DAPI (Blue) was used to mark the nucleus. g Western blotting showed that the activation of synovial NLRP3 inflammasome signaling was markedly provoked by decreased cAMP in SQ22536-treated P2Y14R-KO rats. The relative optical density was exhibited in the supplementary materials (n=4). The data were presented as means±SDs. One-way analysis of variance (ANOVA) with Tukey multiple comparison test was performed. Compared with P2Y14R-KO+vehicle group: #P<0.05, ##P<0.01, ###P<0.001. Compared with P2Y14R-KO+MSU group: *P<0.05, **P<0.01, ***P<0.001. - figure



provided by CiteAb. Source: PMID32457291

Western Blotting

Image 2. Formation of the NLRP3 inflammasome was induced in PBMCs by CSFV infection. (A) PBMCs were mock-infected or infected with CSFV at an MOI of 1 for 48 h. The cell lysate was collected and incubated with the anti-CAS and anti-ASC antibodies in order to immunoprecipitate (IP) caspase 1 (CAS) and ASC. Normal rabbit IgG was used as a negative control. Moreover, ASC and CAS protein levels in the lysates of whole cells were used as input controls. GAPDH was used as an internal loading control. CSFV infection was verified by immunoblotting using anti-CSFV Npro antibody. (B) The level of NLRP3 inflammatory complexes induced by CSFV infection was linked to ATP-dependent K+ channel activation. (C) CSFV infection inhibited the expression of NLRP3 and ASC. *P < 0.05. P-values were calculated using an One-way ANOVA test. - figure provided by CiteAb. Source: PMID30013955



Western Blotting

Image 3. Formation of the NLRP3 inflammasome was induced in PBMCs by CSFV infection. (A) PBMCs were mock-infected or infected with CSFV at an MOI of 1 for 48 h. The cell lysate was collected and incubated with the anti-CAS and anti-ASC antibodies in order to immunoprecipitate (IP) caspase 1 (CAS) and ASC. Normal rabbit IgG was used as a negative control. Moreover, ASC and CAS protein levels in the lysates of whole cells were used as input controls. GAPDH was used as an internal loading control. CSFV infection was verified by immunoblotting using anti-CSFV Npro antibody. (B) The level of NLRP3 inflammatory complexes induced by CSFV infection was linked to ATP-dependent K+ channel activation. (C) CSFV infection inhibited the expression of NLRP3 and ASC. *P < 0.05. P-values were calculated using an One-way ANOVA test. -

Images

figure provided by CiteAb. Source: PMID30013955

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