

href="http://www.uniprot.org/citations/17599095" target="_blank">17599095, PubMed:19158675, PubMed:19158676, PubMed:19234215, PubMed:19494289, PubMed:21487011, PubMed:24630722, PubMed:25847972, PubMed:30674671, PubMed:34678144, PubMed:36050480). Promotes caspase- mediated apoptosis involving predominantly caspase-8 and also caspase-9 in a probable cell type-specific manner (PubMed:11103777, PubMed:12646168). 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 (PubMed:14730312, PubMed:16964285). Involved in innate immune response by acting as an integral adapter in the assembly of various inflammasomes (NLRP1, NLRP2, NLRP3, NLRP6, AIM2 and probably IFI16) which recruit and activate caspase-1 leading to processing and secretion of pro-inflammatory cytokines (PubMed:15030775, PubMed:16982856, PubMed:17349957, PubMed:17599095, PubMed:19158675, PubMed:19158676, PubMed:19234215, PubMed:21487011, PubMed:23530044, PubMed:24630722, PubMed:25847972, PubMed:29440442, PubMed:30674671, PubMed:33980849, PubMed:34678144, PubMed:34706239). Caspase-1-dependent inflammation leads to macrophage pyroptosis, a form of cell death (PubMed:24630722). The function as activating adapter in different types of inflammasomes is mediated by the pyrin and CARD domains and their homotypic interactions (PubMed:14499617, PubMed:19234215, PubMed:24630722). Clustered PYCARD nucleates the formation of caspase-1 filaments through the interaction of their respective CARD domains, acting as a platform for of caspase-1 polymerization (PubMed:24630722). In the NLRP1 and NLRC4 inflammasomes seems not be required but facilitates the processing of procaspase-1 (PubMed:17349957). In cooperation with NOD2 involved in an inflammasome activated by bacterial muramyl dipeptide leading to caspase-1 activation (PubMed:16964285). May be involved in RIGI-triggered pro-inflammatory responses and inflammasome activation (PubMed:19915568). 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 (PubMed:19158675, PubMed:19158676). In adaptive immunity may be involved in maturation of dendritic cells to stimulate T-cell immunity and in cytoskeletal rearrangements coupled to chemotaxis and antigen uptake may be involved in post-transcriptional regulation of the guanine nucleotide exchange factor DOCK2; the latter function is proposed to involve the nuclear form (PubMed:22732093). Also involved in transcriptional activation of cytokines and chemokines independent of the inflammasome; this function may involve AP-1, NF-kappa-B, MAPK and caspase-8 signaling pathways (PubMed:12486103, PubMed:16585594). For regulation of NF-kappa-B activating and inhibiting functions have been reported (PubMed:12486103). Modulates NF-kappa-B induction at the level of the IKK complex by inhibiting kinase activity of CHUK and IKBK (PubMed:12486103, PubMed:16585594). Proposed to compete with RIPK2 for association with CASP1 thereby down-regulating CASP1-mediated RIPK2-dependent NF-kappa-B activation and activating interleukin-1 beta processing (PubMed:16585594). Modulates host resistance to DNA virus infection, probably by inducing the cleavage of and inactivating CGAS in presence of cytoplasmic double-stranded DNA (PubMed:28314590).

Cellular Location

Cytoplasm. Inflammasome. Endoplasmic reticulum. Mitochondrion. Nucleus Note=Upstream of caspase activation, a redistribution from the cytoplasm to the aggregates occurs. These appear as hollow, perinuclear spherical, ball-like structures (PubMed:11103777, PubMed:12191486, PubMed:15030775). Upon NLRP3 inflammasome activation redistributes to the perinuclear space localizing to endoplasmic reticulum and mitochondria (PubMed:12191486, PubMed:15030775). Localized primarily to the nucleus in resting monocytes/macrophages and rapidly redistributed to the cytoplasm upon pathogen infection (PubMed:19234215). Localized to large cytoplasmic aggregate appearing as a speck containing AIM2, PYCARD, CASP8 and bacterial DNA after infection with Francisella tularensis (By similarity). {ECO:0000250|UniProtKB:Q9EPB4, ECO:0000269|PubMed:11103777, ECO:0000269|PubMed:12191486, ECO:0000269|PubMed:15030775, ECO:0000269|PubMed:19234215}

Tissue Location

Widely expressed at low levels. Detected in peripheral blood leukocytes, lung, small intestine, spleen, thymus, colon and at lower levels in placenta, liver and kidney. Very low expression in skeletal muscle, heart and brain. Expressed in lung epithelial cells (at protein level) (PubMed:23229815). Detected in the leukemia cell lines HL-60 and U-937, but not in Jurkat T-cell lymphoma and Daudi Burkitt's lymphoma. Detected in the melanoma cell line WM35, but not in WM793. Not detected in HeLa cervical carcinoma cells and MOLT-4 lymphocytic leukemia cells.

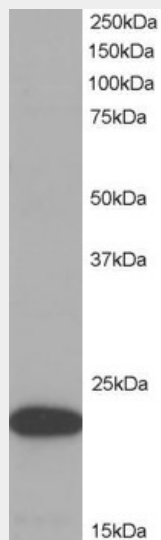
Goat Anti-ASC / TMS1 Antibody - Protocols

Provided below are standard protocols that you may find useful for product applications.

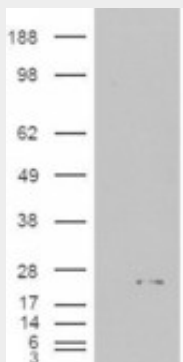
- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)

- [Flow Cytometry](#)
- [Cell Culture](#)

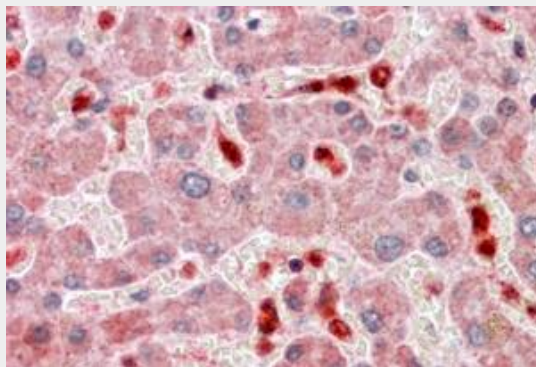
Goat Anti-ASC / TMS1 Antibody - Images



AF1118a (1 $\mu\text{g/ml}$) staining of HeLa lysate (35 μg protein in RIPA buffer). Primary incubation was 1 hour. Detected by chemiluminescence.



HEK293 overexpressing ASC (RC215592) and probed with AF1118a (mock transfection in first lane).



AF1118a (2.5 $\mu\text{g/ml}$) staining of paraffin embedded Human Liver. Steamed antigen retrieval with citrate buffer pH 6, AP-staining.

Goat Anti-ASC / TMS1 Antibody - Background

This gene encodes an adaptor protein that is composed of two protein-protein interaction domains: a N-terminal PYRIN-PAAD-DAPIN domain (PYD) and a C-terminal caspase-recruitment domain (CARD). The PYD and CARD domains are members of the six-helix bundle death domain-fold superfamily that mediates assembly of large signaling complexes in the inflammatory and apoptotic signaling pathways via the activation of caspase. In normal cells, this protein is localized to the cytoplasm; however, in cells undergoing apoptosis, it forms ball-like aggregates near the nuclear periphery. Two transcript variants encoding different isoforms have been found for this gene.

Goat Anti-ASC / TMS1 Antibody - References

Activation of ASC induces apoptosis or necrosis, depending on the cell type, and causes tumor eradication. Motani K, et al. *Cancer Sci*, 2010 Aug. PMID 20500518.

Epigenetic alterations in disseminated neuroblastoma tumour cells: influence of TMS1 gene hypermethylation in relapse risk in NB patients. Grau E, et al. *J Cancer Res Clin Oncol*, 2010 Sep. PMID 20140741.

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A splice variant of ASC regulates IL-1beta release and aggregates differently from intact ASC. Matsushita K, et al. *Mediators Inflamm*, 2009. PMID 19759850.

Structure and interdomain dynamics of apoptosis-associated speck-like protein containing a CARD (ASC). de Alba E. *J Biol Chem*, 2009 Nov 20. PMID 19759015.