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ASC Antibody

Catalog # ASC10110

Product Information

Application	WB, ICC, E
Primary Accession	Q9ULZ3
Other Accession	BAA87339, 10801602
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	21627
Concentration (mg/ml)	1 mg/mL
Conjugate	Unconjugated
Application Notes	ASC antibody can be used for detection of ASC/TMS1 by Western blot at 1 g/mL. Antibody can also be used for immunocytochemistry starting at 5 g/mL.

Additional Information

Gene ID	29108
Other Names	ASC Antibody: ASC, TMS, TMS1, CARD5, TMS-1, ASC, Apoptosis-associated speck-like protein containing a CARD, Caspase recruitment domain-containing protein 5, hASC, PYD and CARD domain containing
Target/Specificity	PYCARD;
Reconstitution & Storage	ASC antibody can be stored at 4℃ for three months and -20℃, stable for up to one year. As with all antibodies care should be taken to avoid repeated freeze thaw cycles. Antibodies should not be exposed to prolonged high temperatures.
Precautions	ASC Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	PYCARD {ECO:0000303|Ref.4, ECO:0000312|HGNC:HGNC:16608}
Function	Functions as a key mediator in apoptosis and inflammation (PubMed:11103777, PubMed:12646168, PubMed:15030775, PubMed:17349957, PubMed: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.

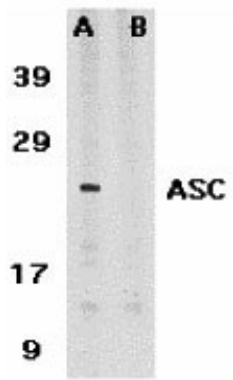
Background

ASC Antibody: Apoptosis is regulated by death domain (DD) and/or caspase recruitment domain (CARD) containing molecules and a caspase family of proteases. CARD containing cell death regulators include RAIDD, RICK, BCL10, Apaf-1, ARC, caspase-9, and caspase-2. A novel CARD domain containing protein was recently identified in human and mouse and designated ASC and TMS1. Ectopic expression of ASC/TMS1 induced apoptosis through activation of caspase-9 and inhibited the survival of human breast cancer cells (3, 4). Overexpression of ASC/TMS1 induced DNA fragmentation. ASC/TMS1 is expressed in a variety of human and mouse tissues.

References

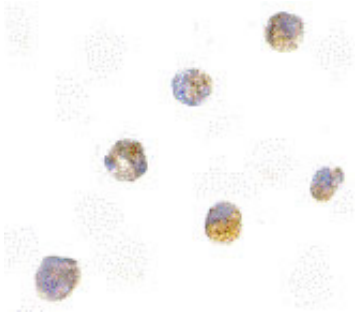
Masumoto J, Taniguchi S, Ayukawa K, Sarvotham H, Kishino T, Niikawa N, Hidaka E, Katsuyama T, Higuchi T, Sagara J. ASC, a novel 22-kDa protein, aggregates during apoptosis of human promyelocytic leukemia HL-60 cells. J Biol Chem. 1999;274(48):33835-8.
Masumoto J, Taniguchi S, Nakayama K, Ayukawa K, Sagara J. Murine Ortholog of ASC, a CARD-Containing Protein, Self-Associates and Exhibits Restricted Distribution in Developing Mouse Embryos. Exp Cell Res. 2001;262(2):128-133.
Conway KE, McConnell BB, Bowring CE, Donald CD, Warren ST , Vertino PM. TMS1, a novel proapoptotic caspase recruitment domain protein, is a target of methylation-induced gene silencing in human breast cancers. Cancer Res. 2000;60(22):6236-42.
McConnell BB, Vertino PM. Activation of a caspase-9-mediated apoptotic pathway by subcellular redistribution of the novel caspase recruitment domain protein TMS1. Cancer Res. 2000;60(22):6243-7.
(WD0102)

Images



Western blot analysis of ASC in HL60 whole cell lysate in the absence (A) or presence (B) of blocking peptide with ASC antibody at 1 µg /ml.

Immunocytochemistry of ASC in HL60 cells with ASC antibody at 5 µg/mL.



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