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ASC Polyclonal Antibody

Catalog # AP68548

Product Information

Application	WB, IHC-P, IF, ICC, E
Primary Accession	Q9ULZ3
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Calculated MW	21627

Additional Information

Gene ID	29108
Other Names	PYCARD; ASC; CARD5; TMS1; 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
Dilution	WB~~Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. Immunofluorescence: 1/200 - 1/1000. ELISA: 1/40000. Not yet tested in other applications. IHC-P~~Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. Immunofluorescence: 1/200 - 1/1000. ELISA: 1/40000. Not yet tested in other applications. IF~~1:50~200 ICC~~N/A E~~N/A
Format	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.
Storage Conditions	-20℃

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

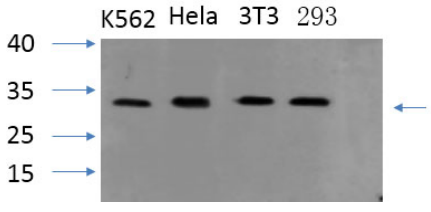
Widely expressed at low levels. Detected in peripheral blood leukocytes, lung,

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

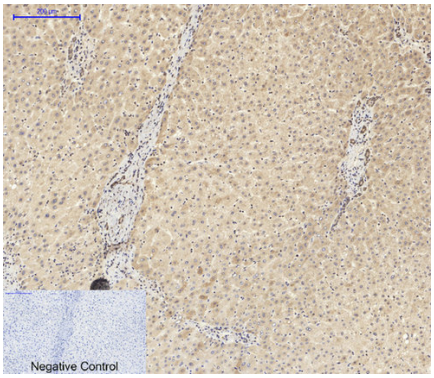
Functions as key mediator in apoptosis and inflammation. Promotes caspase-mediated apoptosis involving predominantly caspase-8 and also caspase-9 in a probable cell type-specific manner. Involved in activation of the mitochondrial apoptotic pathway, promotes caspase-8-dependent proteolytic maturation 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 pyrin 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. 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. 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. For regulation of NF-kappa-B activating and inhibiting functions have been reported. Modulates NF-kappa-B induction at the level of the IKK complex by inhibiting kinase activity of CHUK and IKBK. 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. 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).

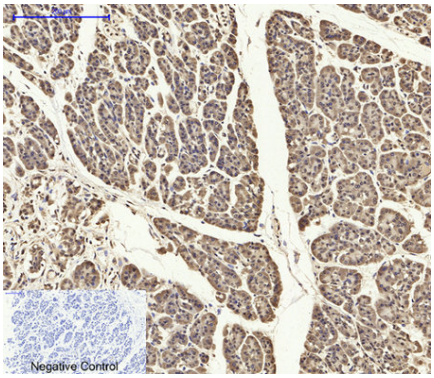
Images



Western Blot analysis of various cells using primary antibody diluted at 1:1000(4°C overnight). Secondary antibody：Goat Anti-rabbit IgG IRDye 800(diluted at 1:5000, 25°C, 1 hour). Cell lysate was extracted by Minute™ Plasma Membrane Protein Isolation and Cell Fractionation Kit(SM-005, Inventbiotech,MN,USA).

Immunohistochemical analysis of paraffin-embedded Human-liver tissue. 1,ASC Polyclonal Antibody was diluted at 1:200(4°C,overnight). 2, Sodium citrate pH 6.0 was used for antibody retrieval(>98°C,20min). 3,Secondary antibody was diluted at 1:200(room tempeRature, 30min). Negative control was used by secondary antibody only.





Immunohistochemical analysis of paraffin-embedded Human-stomach-cancer tissue. 1,ASC Polyclonal Antibody was diluted at 1:200(4°C,overnight). 2, Sodium citrate pH 6.0 was used for antibody retrieval(>98°C,20min). 3,Secondary antibody was diluted at 1:200(room tempeRature, 30min). Negative control was used by secondary antibody only.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.