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Anti-PYCARD / ASC / TMS1 Antibody (aa10-59)

Rabbit Anti Human Polyclonal Antibody
Catalog # ALS17833

Product Information

Application	WB, IHC-P, E
Primary Accession	Q9ULZ3
Predicted	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	21627

Additional Information

Gene ID	29108
Alias Symbol	PYCARD
Other Names	PYCARD, CARD5, HASC, ASC, TMS, TMS-1, PYD and CARD domain containing, TMS1
Target/Specificity	ASC Antibody detects endogenous levels of total ASC protein.
Reconstitution & Storage	Immunoaffinity purified
Precautions	Anti-PYCARD / ASC / TMS1 Antibody (aa10-59) 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.

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