**Product Name:** **TMS1 (2N17) Rabbit Monoclonal Antibody**
**Catalog #:** **AMRe19077**
For research use only.

## Summary

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| **Description** | Recombinant rabbit monoclonal antibody |
| **Host** | Rabbit |
| **Application** | WB,ICC/IF,FC |
| **Reactivity** | Human |
| **Conjugation** | Unconjugated |
| **Modification** | Unmodified |
| **Isotype** | IgG |
| **Clonality** | Monoclonal |
| **Form** | Liquid |
| **Concentration** | 0.5mg/ml. The concentration of this product may be batch-dependent. |
| **Storage** | Aliquot and store at -20°C (valid for 12 months). Avoid freeze/thaw cycles. |
| **Shipping** | Ice bags |
| **Buffer** | Rabbit IgG in phosphate buffered saline , pH 7.4, 150mM NaCl, 0.02% New type preservative N and 50% glycerol. Store at +4°C short term. Store at -20°C long term. Avoid freeze / thaw cycle. |
| **Purification** | Affinity purification |

## Application

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| **Dilution Ratio** | WB 1:1000-1:5000,ICC/IF 1:100-1:200,FC 1:10-1:100 |
| **Molecular Weight** | 22kDa |

## Antigen Information

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| **Gene Name** | PYCARD |
| **Alternative Names** | PYCARD; ASC; CARD5; TMS; TMS-1; TMS1; |
| **Gene ID** | 29108.0 |
| **SwissProt ID** | Q9ULZ3 |
| **Immunogen** | A synthetic peptide of human TMS1 |

## Background

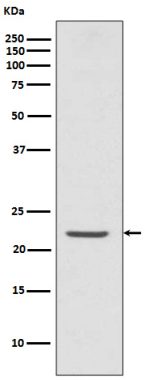
TMS1 (target of methylation-induced silencing)/ASC (apoptosis-associated speck-like protein containing a CARD), also

referred to as PYCARD and CARD5, is a 22-kDa pro-apoptotic protein containing an N-terminal pyrin domain (PYD) and a C-terminal caspase recruitment domain (CARD). 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).

## Research Area

Cell Biology

## Image Data




Western blot analysis of TMS1 expression in U937 cell lysate.