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KD-Validated Anti-TMEM173 Rabbit Monoclonal Antibody

Rabbit monoclonal antibody
Catalog # AGI1003

Product Information

Application	WB, FC, ICC
Primary Accession	Q86WV6
Reactivity	Human
Clonality	Monoclonal
Isotype	Rabbit IgG
Clone Names	24GB5890
Calculated MW	42193
Gene Name	STING1
Aliases	STING1; Stimulator Of Interferon Response CGAMP Interactor 1; STING; ERIS; MITA; Transmembrane; Protein 173; TMEM173; NET23; MPYS; Endoplasmic Reticulum Interferon Stimulator; Stimulator Of Interferon Genes Protein; Endoplasmic Reticulum IFN Stimulator; FLJ38577; HSTING; HMITA; N-Terminal Methionine-Proline-Tyrosine-Serine Plasma Membrane Tetraspanner; Mitochondrial Mediator Of IRF3 Activation; Stimulator Of Interferon Protein; Stimulator Of Interferon Gene; Mediator Of IRF3 Activation; STING-Beta; Sting 1; SAVI
Immunogen	A synthesized peptide derived from human TMEM173

Additional Information

Gene ID	340061
Other Names	Stimulator of interferon genes protein, hSTING, Endoplasmic reticulum interferon stimulator, ERIS, Mediator of IRF3 activation, hMITA, Transmembrane protein 173, STING1 (HGNC:27962)

Protein Information

Name	STING1 (HGNC:27962)
Function	Key innate immune signaling adapter that promotes the production of type I interferon (IFN-alpha and IFN-beta) in response to the presence of DNA from bacteria and viruses in the cytosol (PubMed:18724357, PubMed:18818105, PubMed:19433799, PubMed:19776740, PubMed:23027953, PubMed:23747010, PubMed:23910378, PubMed:25704810, PubMed:27801882, PubMed:29973723, PubMed:30842659, PubMed:31992625, PubMed:32926474, PubMed:35045565, PubMed:35388221, PubMed:36808561, PubMed:37086726, PubMed:37832545, PubMed:39255680, PubMed:39947179). Innate immune response is triggered by non-CpG double-stranded DNA from viruses and bacteria delivered to the cytoplasm, which induces production of cyclic dinucleotides that bind and activate STING1: STING1 recognizes and binds

cyclic di-GMP (c-di-GMP), a second messenger produced by bacteria, cyclic UMP-AMP (2',3'-cUAMP), and cyclic GMP-AMP (cGAMP), a messenger produced by CGAS in response to DNA in the cytosol (PubMed:21947006, PubMed:23258412, PubMed:23707065, PubMed:23722158, PubMed:23747010, PubMed:23910378, PubMed:26229117, PubMed:26300263, PubMed:30842659, PubMed:35388221, PubMed:37086726, PubMed:37379839). Upon binding to c-di-GMP, cUAMP or cGAMP, STING1 oligomerizes and buds from the endoplasmic reticulum into COPII vesicles, which then form the endoplasmic reticulum-Golgi intermediate compartment (ERGIC) (PubMed:30842662, PubMed:41639452, PubMed:41639454, PubMed:41887218). It is then phosphorylated by TBK1 on the pLxIS motif, leading to recruitment and subsequent activation of the transcription factor IRF3 to induce expression of type I interferon and exert a potent antiviral state (PubMed:22394562, PubMed:25636800, PubMed:29973723, PubMed:30643259, PubMed:30842653, PubMed:32926474, PubMed:35045565, PubMed:35388221, PubMed:38917796). Also involved in intercellular immune signaling: cross-activated by 2',3'-cGAMP previously generated in virus-infected cells, triggering type I interferon signaling in macrophages and uninfected neighboring cells to propagate and amplify the antiviral immune response (PubMed:24077100, PubMed:31992625). In addition to promote the production of type I interferon, cGAS-STING signaling also activates the NF-kappa-B signaling: mechanistically, STING1 recruits TRAF6, leading to degradation of the NF-kappa-B inhibitor and subsequent translocation of NF-kappa-B into the nucleus (PubMed:32268090, PubMed:38917796, PubMed:39262777, PubMed:40973797, PubMed:41747053). Activation of NF-kappa-B signaling takes place in the endolysosome and is independent of type I interferon response via direct activation by RNA viruses (PubMed:40973797, PubMed:41747053). Independently of type I interferon production, plays a direct role in autophagy (PubMed:30568238, PubMed:30842662, PubMed:32926474). The ERGIC serves as the membrane source for WIPI2 recruitment and LC3 lipidation, leading to formation of autophagosomes that target cytosolic DNA or DNA viruses for degradation by the lysosome (PubMed:30842662). Promotes autophagy by acting as a proton channel that directs proton efflux from the Golgi to facilitate MAP1LC3B/LC3B lipidation (PubMed:37535724, PubMed:39947179). The autophagy- and interferon-inducing activities can be uncoupled and autophagy induction is independent of TBK1 phosphorylation (PubMed:30568238, PubMed:30842662). Autophagy is also triggered upon infection by bacteria: following c-di-GMP-binding, which is produced by live Gram-positive bacteria, promotes reticulophagy (By similarity). The proton channel activity also regulates lysosome biogenesis by activating transcription factors TFEB and TFE3, driving the expression of lysosome-related genes: activated STING1 in post-Golgi vesicles induces proton efflux and lipidation of GABARAP, sequestering the FLCN- FNIP complex and blocking mTORC1-dependent inhibition of TFEB and TFE3 (PubMed:39423796, PubMed:39689715). May be involved in transduction of apoptotic signals via its association with the major histocompatibility complex class II (MHC-II) (By similarity).

Cellular Location

Endoplasmic reticulum-Golgi intermediate compartment membrane; Multi-pass membrane protein {ECO:0000255, ECO:0000269|PubMed:32690950, ECO:0000269|PubMed:41639452}. Endoplasmic reticulum membrane; Multi-pass membrane protein {ECO:0000255, ECO:0000269|PubMed:30842659, ECO:0000269|PubMed:32690950}. Cytoplasm, perinuclear region. Golgi apparatus membrane; Multi-pass membrane protein. Golgi apparatus, trans-Golgi network membrane; Multi-pass membrane protein. Cytoplasmic vesicle, autophagosome membrane; Multi-pass membrane protein. Endosome membrane; Multi-pass membrane protein. Lysosome membrane; Multi-pass membrane protein. Mitochondrion outer membrane; Multi-pass

membrane protein. Cell membrane {ECO:0000250|UniProtKB:Q3TBT3}; Multi-pass membrane protein. Note=Localizes to the endoplasmic reticulum when inactive (PubMed:19433799, PubMed:29694889, PubMed:30842653, PubMed:30842659). Following activation by cyclic dinucleotides, such as cGAMP, translocates from the endoplasmic reticulum to the endoplasmic reticulum-Golgi intermediate compartment (ERGIC) in a COPII vesicles-dependent process, where the kinase TBK1 is recruited (PubMed:19433799, PubMed:29694889, PubMed:30842653, PubMed:30842659, PubMed:37832545) Translocation to the ERGIC compartment is also dependent on cholesterol and phosphoinositide, such as phosphatidylinositol 3,5-bisphosphate (PtdIns(3,5)P2), which directly bind STING1 at the interface between dimers and promote STING1 homooligomerization (PubMed:41639452, PubMed:41639454). STING1-containing ERGIC serves as a membrane source for LC3 lipidation, which is a key step in autophagosome biogenesis (PubMed:30842662). Activates NF-kappa-B signaling when localized to the endolysosome compartment (PubMed:40973797). Localizes in the lysosome membrane in a TMEM203-dependent manner (By similarity) {ECO:0000250|UniProtKB:Q3TBT3, ECO:0000269|PubMed:19433799, ECO:0000269|PubMed:29694889, ECO:0000269|PubMed:30842653, ECO:0000269|PubMed:30842659, ECO:0000269|PubMed:30842662, ECO:0000269|PubMed:32690950, ECO:0000269|PubMed:37832545, ECO:0000269|PubMed:40973797, ECO:0000269|PubMed:41639452, ECO:0000269|PubMed:41639454}

Tissue Location

Ubiquitously expressed (PubMed:18724357, PubMed:18818105). Expressed in skin endothelial cells, alveolar type 2 pneumocytes, bronchial epithelium and alveolar macrophages (PubMed:25029335).

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