10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

STING/TMEM173 Recombinant Rabbit mAb

Catalog # AP100278

Product Information

Application	WB, IHC-P, IHC-F, IF, IP, E
Primary Accession	Q86WV6
Other Accession	Q86WV6
Reactivity	Rat, Human
Host	Rabbit
Clonality	Recombinant
Calculated MW	42193
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SIMILARITY	Belongs to the TMEM173 family.
SUBUNIT	Associates with the MHC-II complex (By similarity). Homodimer; 'Lys-63'-linked ubiquitination at Lys-150 is required for homodimerization. Interacts with DDX58/RIG-I, MAVS and SSR2. Interacts with RNF5 and TRIM56. Interacts with TBK1; when homodimer, leading to subsequent production of IFN-beta. Interacts with IFIT1 and IFIT2.
Post-translational modifications	Phosphorylated on tyrosine residues upon MHC-II aggregation (By similarity). Phosphorylated on Ser-358 by TBK1, leading to activation and production of IFN-beta. Ubiquitinated. 'Lys-63'-linked ubiquitination mediated by TRIM56 at Lys-150 promotes homodimerization and recruitment of the antiviral kinase TBK1 and subsequent production of IFN-beta. 'Lys-48'-linked polyubiquitination at Lys-150 occurring after viral infection is mediated by RNF5 and leads to proteasomal degradation.
Background Descriptions	This gene encodes a five transmembrane protein that functions as a major regulator of the innate immune response to viral and bacterial infections. The encoded protein is a pattern recognition receptor that detects cytosolic nucleic acids and transmits signals that activate type I interferon responses. The encoded protein has also been shown to play a role in apoptotic signaling by associating with type II major histocompatibility complex. Mutations in this gene are the cause of infantile-onset STING-associated vasculopathy. Alternate splicing results in multiple transcript variants. [provided by RefSeq, Sep 2014],

Additional Information

Gene ID	340061
Other Names	ERIS; MITA; MPYS; NET23; SAVI; STING; STING-beta; TMEM173; hMITA; hSTING; STING_HUMAN; STING1; Endoplasmic reticulum interferon stimulator (ERIS); Mediator of IRF3 activation (hMITA); Transmembrane protein 173; stimulator of interferon response cGAMP interactor 1; stimulator of interferon genes; endoplasmic reticulum IFN stimulator; Endoplasmic reticulum interferon stimulator; Mediator of IRF3 activation; Stimulator of

interferon genes protein

Dilution IHC-P=1:50-200,IHC-F=1:50-200,WB=1:500-2000,IF=1:50-200,ELISA=1:5000-10000,IP=1:50-200

Storage Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

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).

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