

TMEM173 Antibody (C-term)
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP9747b**Specification**

TMEM173 Antibody (C-term) - Product Information

Application	WB, FC,E
Primary Accession	Q86WV6
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Antigen Region	311-340

TMEM173 Antibody (C-term) - 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, TMEM173, ERIS, MITA, STING

Target/Specificity

This TMEM173 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 311-340 amino acids from the C-terminal region of human TMEM173.

Dilution

WB~~1:1000

FC~~1:10~50

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

TMEM173 Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

TMEM173 Antibody (C-term) - Protein Information**Name** STING1 ([HGNC:27962](#))

Function Facilitator of innate immune signaling that acts as a sensor of cytosolic DNA from bacteria and viruses and promotes the production of type I interferon (IFN-alpha and IFN-beta)

(PubMed:[18724357](#), PubMed:[18818105](#), PubMed:[19433799](#), PubMed:[19776740](#), PubMed:[23027953](#), PubMed:[23910378](#), PubMed:[23747010](#), PubMed:[29973723](#), PubMed:[30842659](#), PubMed:[35045565](#), PubMed:[27801882](#), PubMed:[36808561](#), PubMed:[37832545](#), PubMed:[35388221](#)). Innate immune response is triggered in response to non-CpG double-stranded DNA from viruses and bacteria delivered to the cytoplasm (PubMed:[26300263](#)). Acts by binding cyclic dinucleotides: 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 virus in the cytosol (PubMed:[21947006](#), PubMed:[23258412](#), PubMed:[23707065](#), PubMed:[23722158](#), PubMed:[26229117](#), PubMed:[23910378](#), PubMed:[23747010](#), PubMed:[30842659](#), PubMed:[35388221](#), PubMed:[37379839](#)). Upon binding to c- di-GMP, cUAMP or cGAMP, STING1 oligomerizes, translocates from the endoplasmic reticulum and is 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 anti-viral state (PubMed:[22394562](#), PubMed:[25636800](#), PubMed:[29973723](#), PubMed:[30842653](#), PubMed:[35045565](#), PubMed:[35388221](#)). Exhibits 2',3' phosphodiester linkage-specific ligand recognition: can bind both 2'-3' linked cGAMP (2'-3'-cGAMP) and 3'-3' linked cGAMP but is preferentially activated by 2'-3' linked cGAMP (PubMed:[26300263](#), PubMed:[23910378](#), PubMed:[23747010](#)). The preference for 2'-3'-cGAMP, compared to other linkage isomers is probably due to the ligand itself, which adopts an organized free-ligand conformation that resembles the STING1-bound conformation and pays low energy costs in changing into the active conformation (PubMed:[26150511](#)). In addition to promote the production of type I interferons, plays a direct role in autophagy (PubMed:[30568238](#), PubMed:[30842662](#)). Following cGAMP-binding, STING1 buds from the endoplasmic reticulum into COPII vesicles, which then form the endoplasmic reticulum-Golgi intermediate compartment (ERGIC) (PubMed:[30842662](#)). 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](#)). 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). May be involved in translocon function, the translocon possibly being able to influence the induction of type I interferons (PubMed:[18724357](#)). 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 membrane; Multi-pass membrane protein {ECO:0000255, ECO:0000269|PubMed:30842659, ECO:0000269|PubMed:32690950}. Cytoplasm, perinuclear region. Endoplasmic reticulum-Golgi intermediate compartment membrane; Multi-pass membrane protein {ECO:0000255, ECO:0000269|PubMed:32690950}. Golgi apparatus membrane; Multi-pass membrane protein. Cytoplasmic vesicle, autophagosome membrane; Multi-pass membrane protein. Mitochondrion outer membrane; Multi-pass membrane protein. Cell membrane {ECO:0000250|UniProtKB:Q3TBT3}; Multi-pass membrane protein. Note=In response to double-stranded DNA stimulation, translocates from the endoplasmic reticulum through the endoplasmic reticulum-Golgi intermediate compartment and Golgi to post-Golgi vesicles, where the kinase TBK1 is recruited (PubMed:19433799, PubMed:30842659, PubMed:30842653, PubMed:29694889). Upon cGAMP-binding, translocates to the endoplasmic reticulum-Golgi intermediate compartment (ERGIC) in a process that is dependent on COPII vesicles; STING1-containing ERGIC serves as a membrane source for LC3 lipidation, which is a key step in autophagosome biogenesis (PubMed:30842662, PubMed:37832545). 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}

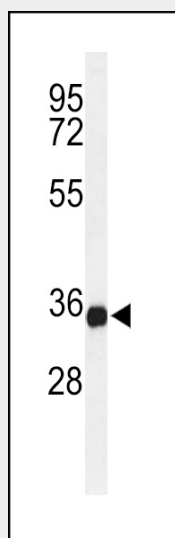
Tissue Location

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

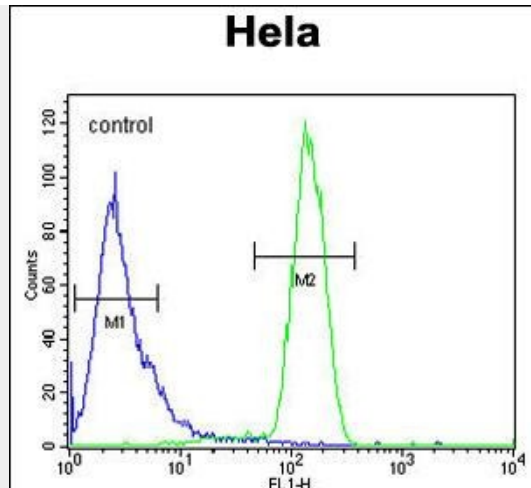
TMEM173 Antibody (C-term) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

TMEM173 Antibody (C-term) - Images

Western blot analysis of lysate from U-937 cell line, using TMEM173 Antibody (C-term)(Cat. #AP9747b). AP9747b was diluted at 1:1000 at each lane. A goat anti-rabbit IgG H&L(HRP) at 1:5000 dilution was used as the secondary antibody. Lysate at 35ug per lane.



TMEM173 Antibody (C-term) (Cat. #AP9747b) flow cytometric analysis of Hela cells (right histogram) compared to a negative control cell (left histogram). FITC-conjugated goat-anti-rabbit secondary antibodies were used for the analysis.

TMEM173 Antibody (C-term) - Background

Acts as a facilitator of innate immune signaling. Able to activate both NF-kappa-B and IRF3 transcription pathways to induce expression of type I interferon (IFN-alpha and IFN-beta) and exert a potent anti-viral state following expression. May be involved in translocon function, the translocon possibly being able to influence the induction of type I interferons. May be involved in transduction of apoptotic signals via its association with the major histocompatibility complex class II (MHC-II). Mediates death signaling via activation of the extracellular signal-regulated kinase (ERK) pathway.

TMEM173 Antibody (C-term) - References

Sun, W., et al. Proc. Natl. Acad. Sci. U.S.A. 106(21):8653-8658(2009)
Zhong, B., et al. Immunity 30(3):397-407(2009)
Graubert, T.A., et al. PLoS ONE 4 (2), E4583 (2009)