

**TM173 Antibody (C-term)**  
**Affinity Purified Rabbit Polyclonal Antibody (Pab)**  
**Catalog # AP9807b****Specification**

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**TM173 Antibody (C-term) - Product Information**

Application	FC, IHC-P, WB,E
Primary Accession	<a href="#">Q86WV6</a>
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	42193
Antigen Region	311-340

**TM173 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 TM173 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 311-340 amino acids from the C-terminal region of human TM173.

**Dilution**

FC~~1:10~50

IHC-P~~1:50~100

WB~~1:1000

E~~Use at an assay dependent concentration.

**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**

TM173 Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

**TM173 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:[23747010](#), PubMed:[23910378](#), PubMed:[27801882](#), PubMed:[29973723](#), PubMed:[30842659](#), PubMed:[35045565](#), PubMed:[35388221](#), PubMed:[36808561](#), PubMed:[37832545](#), PubMed:[25704810](#), PubMed:[39255680](#)). 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:[23747010](#), PubMed:[23910378](#), PubMed:[26229117](#), 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:[23747010](#), PubMed:[23910378](#), PubMed:[26300263](#)). 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:[29694889](#), PubMed:[30842653](#), PubMed:[30842659](#)). 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

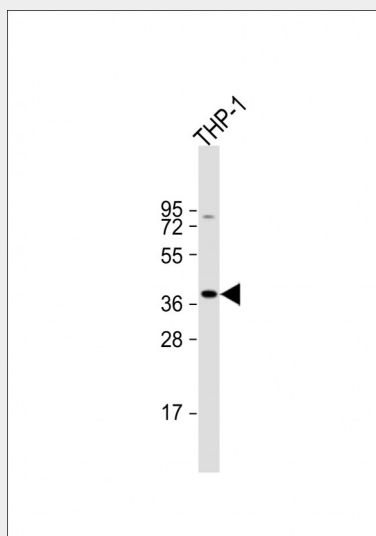
Ubiquitously expressed (PubMed:18724357, PubMed:18818105). Expressed in skin endothelial cells, alveolar type 2 pneumocytes, bronchial epithelium and alveolar macrophages (PubMed:25029335).

#### TM173 Antibody (C-term) - Protocols

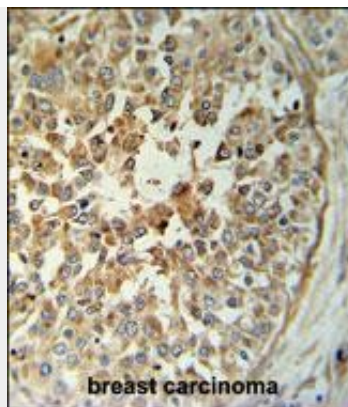
Provided below are standard protocols that you may find useful for product applications.

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

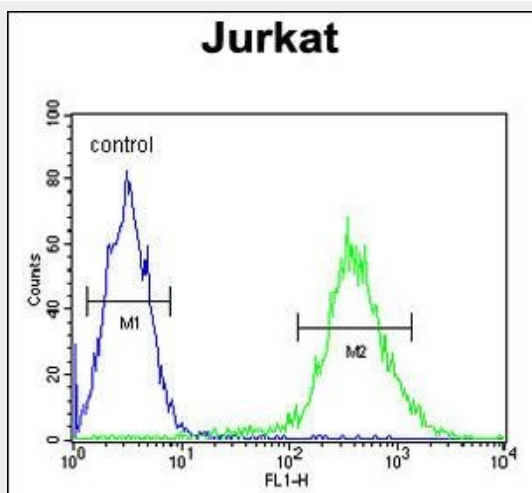
#### TM173 Antibody (C-term) - Images



Anti-TM173 Antibody (C-term) at 1:1000 dilution + THP-1 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 42 kDa Blocking/Dilution buffer: 5% NFDm/TBST.



TM173 Antibody (C-term) (Cat. #AP9807b) IHC analysis in formalin fixed and paraffin embedded breast carcinoma followed by peroxidase conjugation of the secondary antibody and DAB staining. This data demonstrates the use of the TM173 Antibody (C-term) for immunohistochemistry. Clinical relevance has not been evaluated.



TM173 Antibody (C-term) (Cat. #AP9807b) flow cytometric analysis of Jurkat cells (right histogram) compared to a negative control cell (left histogram). FITC-conjugated goat-anti-rabbit secondary antibodies were used for the analysis.

#### **TM173 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)
- Zhong, B., et al. Immunity 29(4):538-550(2008)
- Ishikawa, H., et al. Nature 455(7213):674-678(2008)
- Jin, L., et al. Mol. Cell. Biol. 28(16):5014-5026(2008)