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MPYS Antibody

Catalog # ASC10946

Product Information

Application	WB, IF, ICC, E
Primary Accession	Q86WV6
Other Accession	NP_938023, 38093659
Reactivity	Human, Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	42193
Concentration (mg/ml)	1 mg/mL
Conjugate	Unconjugated
Application Notes	MPYS antibody can be used for detection of MPYS by Western blot at 1 - 2 g/mL. Antibody can also be used for immunocytochemistry starting at 5 g/mL. For immunofluorescence start at 20 g/mL.

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	TMEM173;
Reconstitution & Storage	MPYS antibody can be stored at 4℃ for three months and -20℃, stable for up to one year. As with all antibodies care should be taken to avoid repeated freeze thaw cycles. Antibodies should not be exposed to prolonged high temperatures.
Precautions	MPYS Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

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).

Background

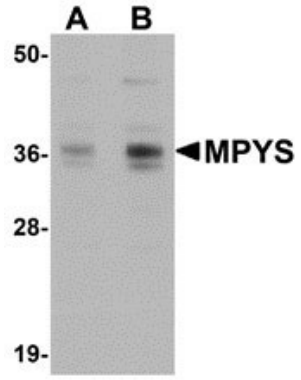
MPYS Antibody: MPYS is a recently identified plasma membrane tetraspanner that is associated with major histocompatibility complex class II (MHC-II) and mediates its transduction of apoptotic signals. It has also been found to be associated with VISA, a mitochondrial protein that acts as an adaptor in virus-triggered signaling. MPYS also interacts with IRF3 and recruits the kinase TBK1 to the VISA-associated complex, acting as a critical mediator of virus-triggered IRF3 activation and interferon (IFN) expression. It is thought that the binding of nucleic acid to the innate immune protein RIG-I causes complex formation between RIG-I, VISA, and MPYS. This complex then recruits TBK1 to phosphorylate IRF3 which then directly activates IFN transcription. At least three isoforms of MPYS are known to exist.

References

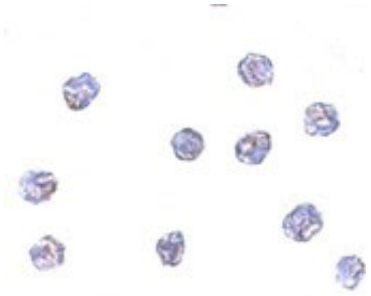
Jin L, Waterman PM, Jonscher KR, et al. MPYS, a novel membrane tetraspanner, is associated with major histocompatibility complex class II and mediates transduction of apoptotic signals. Mol. Cell. Biol.2008; 28:5014-26.
Zhong B, Yang Y, Li S, et al. The adaptor protein MITA links virus-sensing receptors to IRF3 transcription factor activation. Immunity2008; 29:538-50.

Images

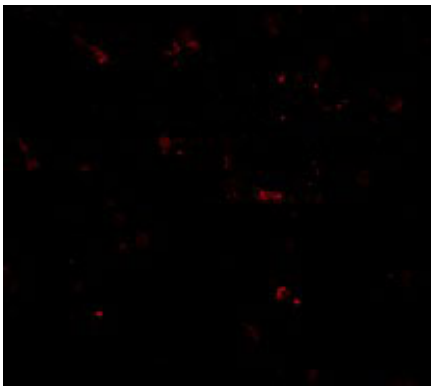
Western blot analysis of MPYS in A-20 cell lysate with



MPYS antibody at (A) 1 and (B) 2 µg/mL.



Immunocytochemistry of MPYS in A-20 cells with MPYS antibody at 5 µg/mL.



Immunofluorescence of MPYS in A-20 cells with MPYS antibody at 20 ug/mL.

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