

**IFITM3 Antibody (N-term)**  
**Purified Mouse Monoclonal Antibody (Mab)**  
**Catalog # AM8579b**

**Specification**

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**IFITM3 Antibody (N-term) - Product Information**

|                   |                        |
|-------------------|------------------------|
| Application       | WB,E                   |
| Primary Accession | <a href="#">Q01628</a> |
| Reactivity        | Human                  |
| Host              | Mouse                  |
| Clonality         | monoclonal             |
| Isotype           | IgG1,k                 |
| Calculated MW     | 14632                  |

**IFITM3 Antibody (N-term) - Additional Information**

**Gene ID** 10410

**Other Names**

Interferon-induced transmembrane protein 3, Dispanin subfamily A member 2b, DSPA2b, Interferon-inducible protein 1-8U, IFITM3

**Target/Specificity**

This IFITM3 antibody is generated from a mouse immunized with a recombinant protein KLH conjugated synthetic peptide between 1-133 amino acids from the N-terminal region of human IFITM3.

**Dilution**

WB~~1:4000

**Format**

Purified monoclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein G column, followed by dialysis against PBS.

**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**

IFITM3 Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

**IFITM3 Antibody (N-term) - Protein Information**

**Name** IFITM3 ([HGNC:5414](#))

**Function** IFN-induced antiviral protein which disrupts intracellular cholesterol homeostasis. Inhibits the entry of viruses to the host cell cytoplasm by preventing viral fusion with cholesterol

depleted endosomes. May inactivate new enveloped viruses which buds out of the infected cell, by letting them go out with a cholesterol depleted membrane. Active against multiple viruses, including influenza A virus, SARS coronaviruses (SARS-CoV and SARS-CoV-2), Marburg virus (MARV), Ebola virus (EBOV), Dengue virus (DNV), West Nile virus (WNV), human immunodeficiency virus type 1 (HIV-1), hepatitis C virus (HCV) and vesicular stomatitis virus (VSV) (PubMed:[26354436](#), PubMed:[33239446](#), PubMed:[33270927](#)). Can inhibit: influenza virus hemagglutinin protein- mediated viral entry, MARV and EBOV GP1,2-mediated viral entry, SARS-CoV and SARS-CoV-2 S protein-mediated viral entry and VSV G protein- mediated viral entry (PubMed:[33270927](#)). Plays a critical role in the structural stability and function of vacuolar ATPase (v-ATPase). Establishes physical contact with the v-ATPase of endosomes which is critical for proper clathrin localization and is also required for the function of the v-ATPase to lower the pH in phagocytic endosomes thus establishing an antiviral state. In hepatocytes, IFITM proteins act in a coordinated manner to restrict HCV infection by targeting the endocytosed HCV virion for lysosomal degradation (PubMed:[26354436](#)). IFITM2 and IFITM3 display anti-HCV activity that may complement the anti-HCV activity of IFITM1 by inhibiting the late stages of HCV entry, possibly in a coordinated manner by trapping the virion in the endosomal pathway and targeting it for degradation at the lysosome (PubMed:[26354436](#)). Exerts opposing activities on SARS-CoV-2, including amphipathicity-dependent restriction of virus at endosomes and amphipathicity-independent enhancement of infection at the plasma membrane (PubMed:[33270927](#)).

#### **Cellular Location**

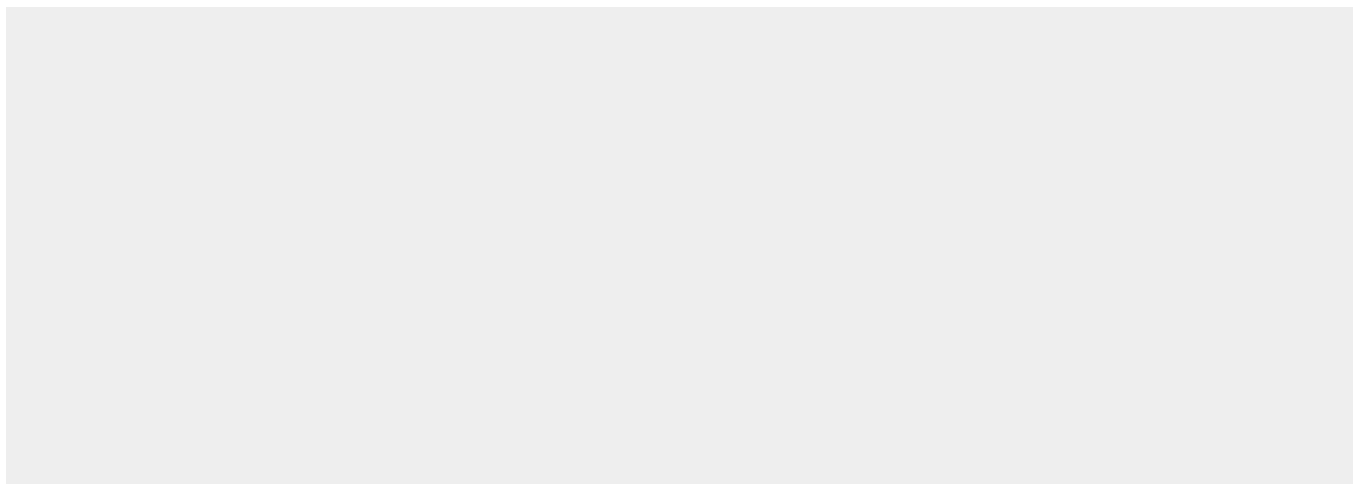
Cell membrane; Single-pass type II membrane protein. Late endosome membrane; Single-pass type II membrane protein. Early endosome membrane; Single-pass type II membrane protein. Lysosome membrane; Single-pass type II membrane protein. Cytoplasm, perinuclear region. Note=Co-localizes with BRI3 isoform 1 at the perinuclear region.

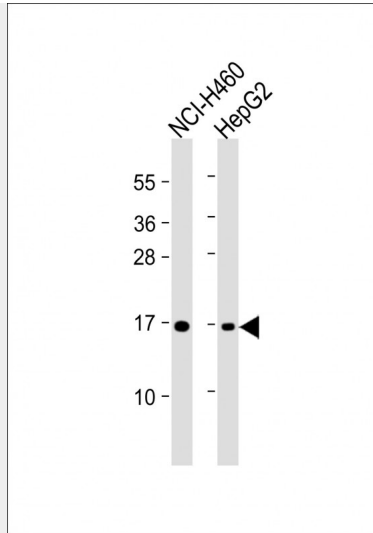
#### **IFITM3 Antibody (N-term) - Protocols**

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](#)

#### **IFITM3 Antibody (N-term) - Images**





All lanes : Anti-Fragilis (IFITM3) Antibody (N-term) at 1:4000 dilution Lane 1: NCI-H460 whole cell lysate Lane 2: HepG2 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-mouse IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 15 kDa Blocking/Dilution buffer: 5% NFDM/TBST.

### **IFITM3 Antibody (N-term) - Background**

IFN-induced antiviral protein which disrupts intracellular cholesterol homeostasis. Inhibits the entry of viruses to the host cell cytoplasm by preventing viral fusion with cholesterol depleted endosomes. May inactivate new enveloped viruses which buds out of the infected cell, by letting them go out with a cholesterol depleted membrane. Active against multiple viruses, including influenza A virus, SARS coronavirus (SARS-CoV), Marburg virus (MARV) and Ebola virus (EBOV), Dengue virus (DENV), West Nile virus (WNV), human immunodeficiency virus type 1 (HIV-1) and vesicular stomatitis virus (VSV). Can inhibit: influenza virus hemagglutinin protein-mediated viral entry, MARV and EBOV GP1,2- mediated viral entry, SARS-CoV S protein-mediated viral entry and VSV G protein-mediated viral entry. Plays a critical role in the structural stability and function of vacuolar ATPase (v-ATPase). Establishes physical contact with the v-ATPase of endosomes which is critical for proper clathrin localization and is also required for the function of the v-ATPase to lower the pH in phagocytic endosomes thus establishing an antiviral state.

### **IFITM3 Antibody (N-term) - References**

- Lewin A.R., et al. *Eur. J. Biochem.* 199:417-423(1991).
- Everitt A.R., et al. *Nature* 484:519-523(2012).
- Kalnina N., et al. Submitted (MAY-2003) to the EMBL/GenBank/DDBJ databases.
- Ota T., et al. *Nat. Genet.* 36:40-45(2004).
- Taylor T.D., et al. *Nature* 440:497-500(2006).