

**IFIT3 Antibody**  
**Catalog # ASC11788****Specification**

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**IFIT3 Antibody - Product Information**

Application  
Primary Accession  
Other Accession  
Reactivity  
Host  
Clonality  
Isotype  
Calculated MW

**WB, IHC**  
[O14879](#)  
[NP\\_001026853](#), [72534658](#)  
**Human**  
**Rabbit**  
**Polyclonal**  
**IgG**  
**Predicted: 54 kDa**

Application Notes

**Observed: 50 kDa KDa**  
**IFIT3 antibody can be used for detection of IFIT3 by Western blot at 1 - 2 µg/ml. Antibody can also be used for Immunohistochemistry at 5 µg/mL.**

**IFIT3 Antibody - Additional Information**

Gene ID **3437**  
**Target/Specificity**

IFIT3; IFIT3 antibody is human specific. This antibody is predicted to not cross-react with other members of the IFIT protein family.

**Reconstitution & Storage**

IFIT3 antibody can be stored at 4°C for three months and -20°C, stable for up to one year.

**Precautions**

IFIT3 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

**IFIT3 Antibody - Protein Information**

**Name** IFIT3

**Synonyms** CIG-49, IFI60, IFIT4, ISG60

**Function**

IFN-induced antiviral protein which acts as an inhibitor of cellular as well as viral processes, cell migration, proliferation, signaling, and viral replication. Enhances MAVS-mediated host antiviral responses by serving as an adapter bridging TBK1 to MAVS which leads to the activation of TBK1 and phosphorylation of IRF3 and phosphorylated IRF3 translocates into nucleus to promote antiviral gene transcription. Exhibits an antiproliferative activity via the up-regulation of cell cycle negative regulators CDKN1A/p21 and CDKN1B/p27. Normally, CDKN1B/p27 turnover is regulated by COPS5, which binds CDKN1B/p27 in the nucleus and exports it to the cytoplasm for ubiquitin-dependent degradation. IFIT3 sequesters COPS5 in the cytoplasm, thereby increasing

nuclear CDKN1B/p27 protein levels. Up-regulates CDKN1A/p21 by down-regulating MYC, a repressor of CDKN1A/p21. Can negatively regulate the apoptotic effects of IFIT2.

**Cellular Location**

Cytoplasm. Mitochondrion

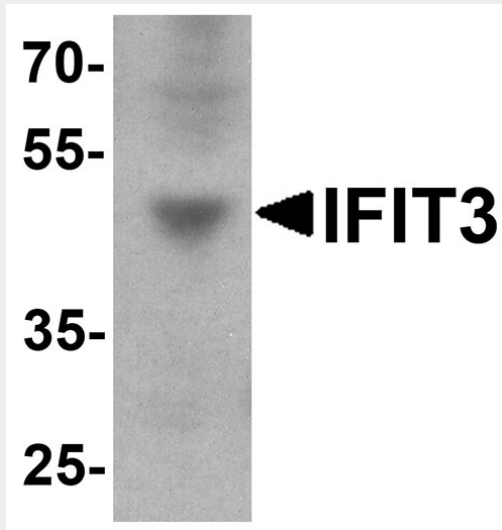
**Tissue Location**

Expression significantly higher in peripheral blood mononuclear cells (PBMCs) and monocytes from systemic lupus erythematosus (SLE) patients than in those from healthy individuals (at protein level). Spleen, lung, leukocytes, lymph nodes, placenta, bone marrow and fetal liver.

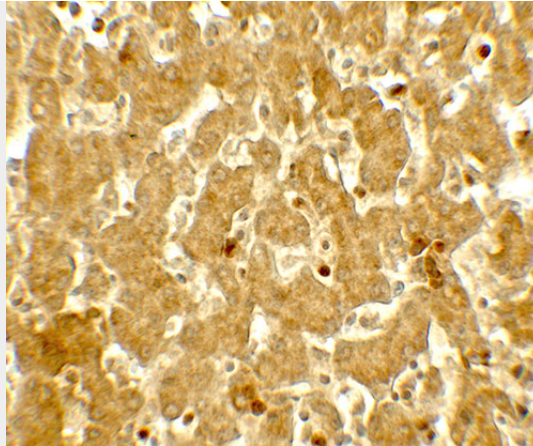
**IFIT3 Antibody - Protocols**

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](#)

**IFIT3 Antibody - Images**

Western blot analysis of IFIT3 in HepG2 cell lysate with IFIT3 antibody at 1 µg/ml.



Immunohistochemistry of IFIT3 in human liver tissue with IFIT3 antibody at 5 µg/mL.

### **IFIT3 Antibody - Background**

The interferon-induced protein with tetratricopeptide repeats 3 (IFIT3) protein is a member of a family of tetratricopeptide repeat-containing proteins whose transcription is upregulated by interferons, virus infection, and molecular patterns such as dsRNA or lipopolysaccharides (1,2). These proteins have been suggested to induce anti-viral cellular activities in response to infection (2). IFIT3 has been shown to interact with TBK1 following RNA virus infection, thereby bridging TBK1 to VISA on the mitochondrion and activating IRF3 and thus the immune response (3).

### **IFIT3 Antibody - References**

Yu M, Tong JH, Mao M, et al. Cloning of a gene (RIG-G) associated with retinoic acid-induced differentiation of acute promyelocytic leukemia cells and representing a new member of a family of interferon-stimulating genes. *Proc. Natl. Acad. Sci. USA* 1997; 94:7406-11.  
Fensterl V and Sen GC. The ISG56/IFIT1 gene family. *J. Interferon Cytokine Res.* 2011; 31:71-8.  
Raychoudhuri A, Shrivastava S, Steele R, et al. ISG56 and IFITM1 proteins inhibit hepatitis C virus replication. *J. Virol.* 2011; 85:12881-9.