

<http://www.uniprot.org/citations/31340999> target="_blank">31340999, PubMed:36603579). Regulates the transcription of type I IFN genes (IFN-alpha and IFN-beta) and IFN-stimulated genes (ISG) by binding to an interferon-stimulated response element (ISRE) in their promoters (PubMed:8524823, PubMed:11846977, PubMed:16846591, PubMed:16979567, PubMed:20049431, PubMed:36603579, PubMed:32972995). Acts as a more potent activator of the IFN-beta (IFNB) gene than the IFN-alpha (IFNA) gene and plays a critical role in both the early and late phases of the IFNA/B gene induction (PubMed:16846591, PubMed:16979567, PubMed:20049431, PubMed:36603579). Found in an inactive form in the cytoplasm of uninfected cells and following viral infection, double-stranded RNA (dsRNA), or toll-like receptor (TLR) signaling, is phosphorylated by IKBKE and TBK1 kinases (PubMed:22394562, PubMed:25636800, PubMed:36603579, PubMed:27302953). This induces a conformational change, leading to its dimerization and nuclear localization and association with CREB binding protein (CREBBP) to form dsRNA-activated factor 1 (DRAF1), a complex which activates the transcription of the type I IFN and ISG genes (PubMed:16154084, PubMed:27302953, PubMed:33440148, PubMed:36603579). Can activate distinct gene expression programs in macrophages and can induce significant apoptosis in primary macrophages (PubMed:16846591). In response to Sendai virus infection, is recruited by TOMM70:HSP90AA1 to mitochondrion and forms an apoptosis complex TOMM70:HSP90AA1:IRF3:BAX inducing apoptosis (PubMed:25609812). Key transcription factor regulating the IFN response during SARS-CoV-2 infection (PubMed:33440148).

Cellular Location

Cytoplasm. Nucleus Mitochondrion. Note=Shuttles between cytoplasmic and nuclear compartments, with export being the prevailing effect (PubMed:10805757, PubMed:35922005). When activated, IRF3 interaction with CREBBP prevents its export to the cytoplasm (PubMed:10805757). Recruited to mitochondria via TOMM70:HSP90AA1 upon Sendai virus infection (PubMed:25609812).

Tissue Location

Expressed constitutively in a variety of tissues.

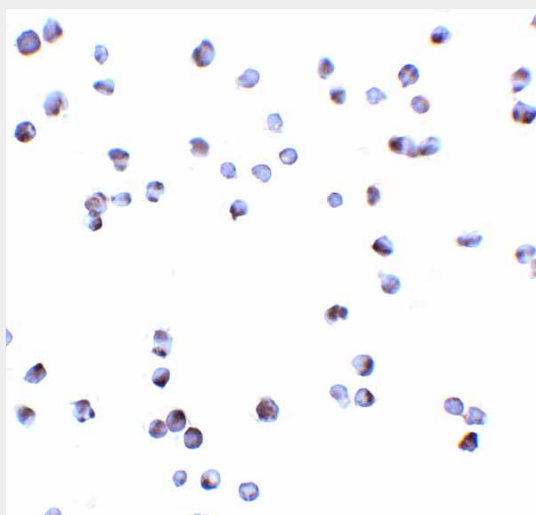
IRF3 Antibody - Protocols

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](#)

IRF3 Antibody - Images



Immunocytochemistry of ZIPK in Jurkat cells with ZIPK antibody at 5 µg/ml.

IRF3 Antibody - Background

IRF3 Antibody: Interferons (IFN)s are involved in a multitude of immune interactions during viral infections and play a major role in both the induction and regulation of innate and adaptive antiviral mechanisms. During infection, host-virus interactions signal downstream molecules such as transcription factors such as IFN regulatory factor-3 (IRF3) which can act to stimulate transcription of IFN-alpha/beta genes. IRF3 is present in an inactive form in the cytoplasm of most cells. Following viral infection, IRF3 can be activated by I κ B kinase- ϵ and TANK-binding kinase 1 (TBK1), whereupon IRF3 translocates to the nucleus. IRF3 can also be activated by stimulation of toll-like receptor 3 (TLR3) by dsRNA. IRF3 exists as at least two distinct isoforms.

IRF3 Antibody - References

- Malmgaard L. Induction and regulation of IFNs during viral infections. J. Interferon & Cyto. Res. 2004; 24:439-54.
- Au WC, Moore PA, Lowther W, et al. Identification of a member of the interferon regulatory factor family that binds to the interferon-stimulated response element and activates expression of interferon-induced genes. Proc. Natl. Acad. Sci. USA 1995; 92:11657-61.
- Fitzgerald KA, McWhirter SM, Faia KL, et al. IKKepsilon and TBK1 are essential components of the IRF3 signaling pathway. Nat. Immunol. 2003; 4:491-6.
- Sharma S, Tenover BR, Grandvaux N, et al. Triggering the interferon antiviral response through an IKK-related pathway. Science 2003; 300:1148-51.