10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

IRF8 Antibody

Catalog # ASC10272

Product Information

Application	WB, IF, E
Primary Accession	Q14653
Other Accession	NP_002154, 4504567
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	47219
Concentration (mg/ml)	1 mg/mL
Conjugate	Unconjugated
Application Notes	IRF8 antibody can be used for detection of IRF8 by Western blot at 1 g/mL. For immunofluorescence start at 10 g/mL.

Additional Information

Gene ID	3661
Other Names	IRF8 Antibody: Interferon regulatory factor 3, IRF-3, interferon regulatory factor 3
Target/Specificity	IRF3;
Reconstitution & Storage	IRF8 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	IRF8 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	IRF3 {ECO:0000303|PubMed:9803267, ECO:0000312|HGNC:HGNC:6118}
Function	Key transcriptional regulator of type I interferon (IFN)- dependent immune responses which plays a critical role in the innate immune response against DNA and RNA viruses (PubMed:22394562, PubMed:24049179, PubMed:25636800, PubMed:27302953, PubMed:31340999, PubMed:36603579, PubMed:8524823, PubMed:39362857). 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:11846977, PubMed:16846591, PubMed:16979567, PubMed:20049431, PubMed:32972995, PubMed:36603579, PubMed:8524823). 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:27302953, PubMed:36603579). 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.

Background

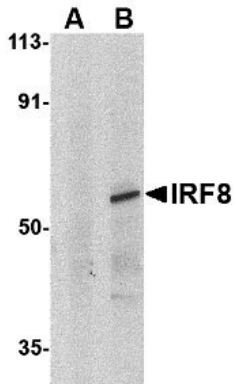
IRF8 Antibody: Interferons (IFN)s are involved in a multitude of immune interactions during viral infections and play a major role in both the induction and regulation of innate and adaptive antiviral mechanisms. During infection, host-virus interactions signal downstream molecules such as transcription factors such as IFN regulatory factor-3 (IRF3) which can act to stimulate transcription of IFN-alpha/beta genes. Unlike IRF3, IRF8 appears to act as a negative regulator of IFN-induced genes in most cases, but IRF8 mediates activation of NF-κB by the toll-like receptor 9 (TLR9) after stimulation by unmethylated CpG DNA in dendritic cells. Finally, it has been shown that IRF8 decreases bcl-2 expression and thus may play a role in chronic myelogenous leukemia.

References

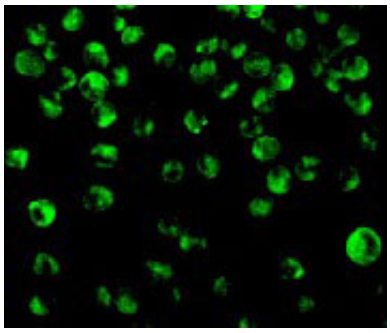
Malmgaard L. Induction and regulation of IFNs during viral infections. J. Interferon & Cyto. Res. 2004; 24:439-54
Weisz A, Marx P, Sharf R, et al. The human interferon consensus sequence binding protein (H-ICSBP) is a negative regulator of enhancer elements common to interferon inducible genes. J. Biol. Chem. 1992; 267:25589-96.
Nelson N, Marks MS, Driggers PH, et al. Interferon consensus sequence-binding protein, a member of the interferon regulatory factor family, suppresses interferon-induced gene transcription. Mol. Cell. Biol. 1993; 13:588-99.
Tsujimura H, Tamura T, Kong HJ, et al. Toll-like receptor 9 signaling activates NF-κB through IFN regulatory factor-8/IFN consensus sequence binding protein in dendritic cells. J. Immunol. 2004; 172:6820-7.

Images

Western blot analysis of IRF8 in human thymus tissue lysate with IRF8 antibody at 1 µg/mL in (A) the presence



and (B) absence of blocking peptide.



Immunofluorescence of IRF8 in K562 cells with IRF8 antibody at 10 µg/mL.

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