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IRF3

Reactivity: Human

Tested applications:WB IHC CHIP CHIPseq

Recommended Dilution:WB 1:500 - 1:2000 IHC 1:50 - 1:200 IF 1:50 - 1:200 IP 1:50 - 1:200 ChIP 1:20 - 1:100

Calculated MW:49kDa

Observed MW: Refer to Figures

Immunogen:

Recombinant protein of human IRF3

Storage Buffer:

Store at -20. Avoid freeze / thaw cycles. Buffer: PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

Synonym:

IRF-3; IRF3;

Catalog #:A11118

Antibody Type:

Polyclonal Antibody

Species: Rabbit

Gene ID:3661

Isotype: IgG

Swiss Prot:Q14653

Purity: Affinity purification

For research use only.

Background:

Interferon regulatory factors (IRFs) comprise a family of transcription factors that function within the Jak/Stat pathway to regulate interferon (IFN) and IFN-inducible gene expression in response to viral infection (1). IRFs play an important role in pathogen defense, autoimmunity, lymphocyte development, cell growth, and susceptibility to transformation. The IRF family includes nine members: IRF-1, IRF-2, ISGF3/p48, IRF-3, IRF-4 (Pip/LSIRF/ICSAT), IRF-5, IRF-6, IRF-7, and IRF-8/ICSBP. All IRF proteins share homology in their amino-terminal DNA-binding domains. IRF family members regulate transcription through interactions with proteins that share similar DNA-binding motifs, such as IFN-stimulated response elements (ISRE), IFN consensus sequences (ICS), and IFN regulatory elements (IRF-E) (2).IRF-3 can inhibit cell growth and plays a critical role in controlling the expression of genes in the innate immune response (1-4). In unstimulated cells, IRF-3 is present in the cytoplasm. Viral infection results in phosphorylation of IRF-3 and leads to its translocation to the nucleus where it activates promoters containing IRF-3-binding sites. Phosphorylation of IRF-3 occurs at a cluster of C-terminal Ser and Thr residues (between 385 and 405), leading to its association with the p300/CBP coactivator protein that promotes DNA binding and transcriptional activity (5). During infection, IRF-3 is likely activated through a pathway that includes activation of Toll-like receptors and a kinase complex that includes IKK and TBK1 (6,7). IRF-3 is phosphorylated at Ser396 following viral infection, expression of viral nucleocapsid, and double-stranded RNA treatment. These events likely play a role in activation of IRF-3 (8).

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