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Phospho-IRF3-S386

Reactivity: Human

Tested applications:WB ICC

Recommended Dilution:WB 1:500 - 1:2000 ICC 1:50 - 1:200

Observed MW: Refer to Figures

Immunogen:

A phospho specific peptide corresponding to residues surrounding Serine 386 of human IRF3

Storage Buffer:

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

Concentration:

ks

Synonym:

IRF3 ; Interferon regulatory factor 3 ;

Catalog #:AP0091

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 serine and threonine 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 of 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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