

## IRF4

**Reactivity:**Human Mouse Rat

**Tested applications:**WB IHC IF

**Recommended Dilution:**WB 1:500 - 1:2000 IHC 1:50 - 1:200 IF 1:50 - 1:200

**Calculated MW:**52kDa

**Observed MW:**Refer to Figures

**Immunogen:**

Recombinant protein of human IRF4

**Storage Buffer:**

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

**Concentration:**

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**Synonym:**

IRF4;LSIRF;MUM1 ;

**Catalog #:**A1052

**Antibody Type:**

Polyclonal Antibody

**Species:**Rabbit

**Gene ID:**3662

**Isotype:**IgG

**Swiss Prot:**Q15306

**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-4 was independently cloned by three groups and demonstrated to have roles in different contexts of lymphoid regulation (3-5). First, IRF-4 (Pip) was found to associate with PU.1, a hematopoietic specific member of the ETS family, and to regulate the expression of B-cell specific genes (3). Second, it was characterized as a lymphoid-specific member of the IRF family (LSIRF) and able to bind to ISRE (4). Third, it was identified in activated T cells as a factor that binds to the promoter of the interleukin-5 gene (ICSAT), and shown to repress gene activation induced by IFN (5). IRF-4 is expressed in all stages of B cell development and in mature T cells, and is inducible in primary lymphocytes by antigen mimetic stimuli such as Concanavalin A, CD3 crosslinking, anti-IgM and PMA treatment (4,5). Mice deficient in IRF-4 show normal distribution of B and T lymphocytes at 4 to 5 weeks, but later develop progressive generalized lymphadenopathy, suggesting a role for IRF-4 in the function and homeostasis of mature B- and T-lymphocytes (6).

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