

## RFX5 rabbit pAb

<b>Catalog No :</b>	YT7052
<b>Reactivity :</b>	Human;Mouse
<b>Applications :</b>	WB;IHC
<b>Target :</b>	RFX5
<b>Fields :</b>	>>Antigen processing and presentation;>>Tuberculosis;>>Primary immunodeficiency
<b>Gene Name :</b>	RFX5
<b>Protein Name :</b>	RFX5
<b>Human Gene Id :</b>	5993
<b>Human Swiss Prot No :</b>	P48382
<b>Mouse Gene Id :</b>	53970
<b>Mouse Swiss Prot No :</b>	Q9JL61
<b>Immunogen :</b>	Synthesized peptide derived from human RFX5 AA range: 491-541
<b>Specificity :</b>	This antibody detects endogenous levels of RFX5 at Human/Mouse
<b>Formulation :</b>	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
<b>Source :</b>	Polyclonal, Rabbit,IgG
<b>Dilution :</b>	WB 1:500-2000;IHC 1:50-300
<b>Purification :</b>	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
<b>Concentration :</b>	1 mg/ml

**Storage Stability :** -15°C to -25°C/1 year(Do not lower than -25°C)

**Molecularweight :** 68kD

**Background :** A lack of MHC-II expression results in a severe immunodeficiency syndrome called MHC-II deficiency, or the bare lymphocyte syndrome (BLS; MIM 209920). At least 4 complementation groups have been identified in B-cell lines established from patients with BLS. The molecular defects in complementation groups B, C, and D all lead to a deficiency in RFX, a nuclear protein complex that binds to the X box of MHC-II promoters. The lack of RFX binding activity in complementation group C results from mutations in the RFX5 gene encoding the 75-kD subunit of RFX (Steimle et al., 1995). RFX5 is the fifth member of the growing family of DNA-binding proteins sharing a novel and highly characteristic DNA-binding domain called the RFX motif. Multiple alternatively spliced transcript variants have been found but the full-length natures of only two have been determined. [provided by RefSeq, Jul 2008],

**Function :** disease:Defects in RFX5 are a cause of bare lymphocyte syndrome type 2 (BLS2) [MIM:209920]; also known as hereditary MHC class II deficiency or HLA class II-deficient combined immunodeficiency. BLS2 is a severe combined immunodeficiency disease with early onset. It is characterized by a profound defect in constitutive and interferon-gamma induced MHC II expression, absence of cellular and humoral T-cell response to antigen challenge, hypogammaglobulinemia and impaired antibody production. The consequence include extreme susceptibility to viral, bacterial and fungal infections.,domain:The N-terminus is required for its association with RFXANK and RFXAP, for assembly of the RFX complex, and for binding of this complex to its X box target site in the MHC-II promoter. The C-terminus mediates cooperative binding between the RFX complex and NF-Y.,function:Activates transcription from class II

**Subcellular Location :** Nucleus.

**Expression :** Ubiquitous.

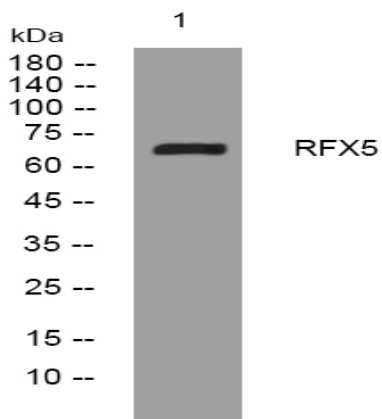
**Sort :** 14423

**No4 :** 1

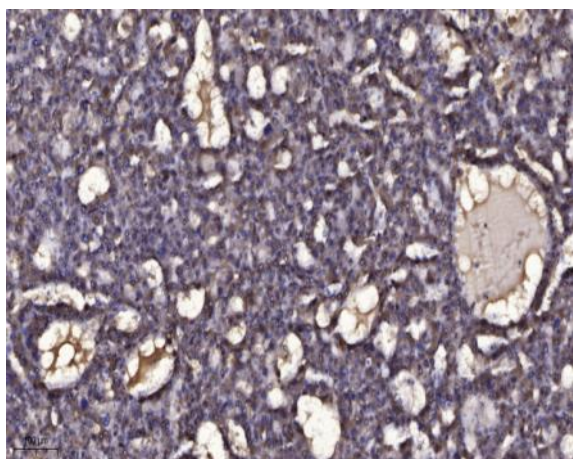
**Host :** Rabbit

**Modifications :** Unmodified

**Products Images**



Western blot analysis of lysates from A431 cells, primary antibody was diluted at 1:1000, 4° over night



Immunohistochemical analysis of paraffin-embedded human liver cancer. 1, Antibody was diluted at 1:200(4° overnight). 2, Tris-EDTA,pH9.0 was used for antigen retrieval. 3,Secondary antibody was diluted at 1:200(room temperature, 45min).