ELK Biotechnology

Stat1 (Acetyl Lys410/K413) rabbit pAb

Cat No.:ES20130

For research use only

Overview

Product Name	Stat1 (Acetyl Lys410/K413) rabbit pAb
Host species	Rabbit
Applications	WB;ELISA;IHC
Species Cross-Reactivity	Human;Mouse;Rat
Recommended dilutions	WB 1:500-2000;IHC-p 1:50-300; ELISA 2000-20000
Immunogen	Synthesized peptide derived from human Stat1 (Acetyl Lys410/K413)
Specificity	This antibody detects endogenous levels of Human,Mouse,Rat Stat1 (Acetyl Lys410/K413)
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Storage	Store at -20°C. Avoid repeated freeze-thaw cycles.
Protein Name	Stat1 (Acetyl Lys410/K413)
Gene Name	STAT1
Cellular localization	Cytoplasm . Nucleus . Translocated into the nucleus upon tyrosine phosphorylation and dimerization, in response to IFN-gamma and signaling by activated FGFR1, FGFR2, FGFR3 or FGFR4 (PubMed:15322115). Monomethylation at Lys-525 is required for phosphorylat
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
Clonality	Polyclonal
Concentration	1 mg/ml
Observed band	85kD
Human Gene ID	6772
Human Swiss-Prot Number	P42224
Alternative Names	Signal transducer and activator of transcription 1-alpha/beta (Transcription factor ISGF-3 components p91/p84)
Background	disease:Defects in STAT1 are a cause of mendelian susceptibility to mycobacterial disease (MSMD)

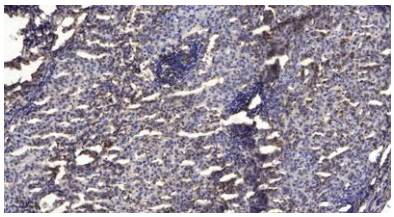
+86-27-59760950 ELKbio@ELKbiotech.com www.elkbiotech.com

23-2, No.388 Gaoxin 2nd Road,Wuhan East Lake Hi-tech Development Zone, Hubei , P.R.C

[MIM:209950]; also known as familial disseminated atypical mycobacterial infection. This rare condition confers predisposition to illness caused by moderately virulent mycobacterial species, such as Bacillus Calmette-Guerin (BCG) vaccine and environmental non-tuberculous mycobacteria, and by the more virulent Mycobacterium tuberculosis. Other microorganisms rarely cause severe clinical disease in individuals with susceptibility to mycobacterial infections, with the exception of Salmonella which infects less than 50% of these individuals. The pathogenic mechanism underlying MSMD is the impairment of interferon-gamma mediated immunity whose severity determines the clinical outcome. Some patients die of overwhelming mycobacterial disease with lepromatous-like lesions in early childhood, whereas others develop, later in life, disseminated but curable infections with tuberculoid granulomas. MSMD is a genetically heterogeneous disease with autosomal recessive, autosomal dominant or X-linked inheritance.,disease:Defects in STAT1 are the cause of STAT1 deficiency [MIM:600555]. Patients generally suffer from mycobacterial or viral diseases. In the case of complete deficiency, patients can die of viral disease.,function:Signal transducer and activator of transcription that mediates signaling by interferons (IFNs). Following type I IFN (IFN-alpha and IFN-beta) binding to cell surface receptors, Jak kinases (TYK2 and JAK1) are activated, leading to tyrosine phosphorylation of STAT1 and STAT2. The phosphorylated STATs dimerize, associate with ISGF3G/IRF-9 to form a complex termed ISGF3 transcription factor, that enters the nucleus. ISGF3 binds to the IFN stimulated response element (ISRE) to activate the transcription of interferon stimulated genes, which drive the cell in an antiviral state. In response to type II IFN (IFN-gamma), STAT1 is tyrosine- and serine-phosphorylated. It then forms a homodimer termed IFN-gamma-activated factor (GAF), migrates into the nucleus and binds to the IFN gamma

activated sequence (GAS) to drive the expression of the target genes, inducing a cellular antiviral state.,online information:STAT1 entry,online information:STAT1 mutation db,PTM:Phosphorylated on tyrosine and serine residues in response to IFN-alpha, IFN-gamma, PDGF and EGF. Phosphorylation on Tyr-701 (lacking in beta form) by JAK promotes dimerization and subsequent translocation to the nucleus. Phosphorylation on Ser-727 by several kinases including MAPK14, ERK1/2 and CAMKII on IFN-gamma stimulation, regulates STAT1 transcriptional activity. Phosphorylation on Ser-727 promotes sumoylation though increasing interaction with PIAS. Phosphorylation on Ser-727 by PKCdelta induces apoptosis in response to DNA-damaging agents.,PTM:Sumoylated by SUMO1, SUMO2 and SUMO3. Sumoylation is enhanced by IFN-gamma-induced phosphorylation on Ser-727, and by interaction with PIAS proteins. Enhances the transactivation activity.,similarity:Belongs to the transcription factor STAT family.,similarity:Contains 1 SH2 domain.,subcellular location:Translocated into the nucleus in response to IFN-gamma-induced tyrosine phosphorylation and dimerization.,subunit:Isoform alpha homodimerizes upon IFN-gamma induced phosphorylation. Heterodimer with STAT2 upon IFN-alpha/beta induced phosphorylation. Interacts with NMI. Interacts with Sendai virus C', C, Y1 and Y2 proteins, Nipah virus P, V and W proteins, and rabies virus phosphoprotein preventing activation of ISRE and GAS promoter (By similarity). Interacts with HCV core protein; the interaction results in STAT1 degradation. Interacts with PIAS1; the interaction requires phosphorylation on Ser-727 and inhibits STAT1 activation.,

ELK Biotechnology



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).

+86-27-59760950 ELKbio@ELKbiotech.com www.elkbiotech.com

23-2, No.388 Gaoxin 2nd Road,Wuhan East Lake Hi-tech Development Zone, Hubei , P.R.C