Anti-STAT1 Antibody

R1408-2



Product Type:	Rabbit polyclonal IgG, primary antibodies
Species reactivity:	Human, Mouse, Rat, Zebrafish
Applications:	WB, IF-Cell
Molecular Wt:	Predicted band size: 87 kDa

Description: All STAT molecules are phosphorylated by receptor associated kinases, that causes activation, dimerization by forming homo- or heterodimers and finally translocate to nucleus to work as transcription factors. Specifically STAT1 can be activated by several ligands such as Interferon alpha (IFN α), Interferon gamma (IFN γ), Epidermal Growth Factor (EGF), Platelet Derived Growth Factor (PDGF), Interleukin 6 (IL-6), or IL-27. Type I interferons (IFN- α , IFN- β) bind to receptors, cause signaling via kinases, phosphorylate and activate the Jak kinases TYK2 and JAK1 and also STAT1 and STAT2. STAT molecules form dimers and bind to ISGF3G/IRF-9, which is Interferon stimulated gene factor 3 complex with Interferon regulatory Factor 9. This allows STAT1 to enter the nucleus.[6] STAT1 has a key role in many gene expressions that cause survival of the cell, viability or pathogen response. There are two possible transcripts (due to alternative splicing) that encode 2 isoforms of STAT1. STAT1a, the full-length version of the protein, is the main active isoform, responsible for most of the known functions of STAT1. STAT1B, which lacks a portion of the C-terminus of the protein, is less-studied, but has variously been reported to negatively regulate activation of STAT1 or to mediate IFN-y-dependent anti-tumor and anti-infection activities. STAT1 is involved in upregulating genes due to a signal by either type I, type II, or type III interferons. In response to IFN-y stimulation, STAT1 forms homodimers or heterodimers with STAT3 that bind to the GAS (Interferon-Gamma-Activated Sequence) promoter element; in response to either IFN- α or IFN- β stimulation, STAT1 forms a heterodimer with STAT2 that can bind the ISRE (Interferon-Stimulated Response Element) promoter element. In either case, binding of the promoter element leads to an increased expression of ISG (Interferon-Stimulated Genes). Expression of STAT1 can be induced with diallyl disulfide, a compound in garlic. Immunogen: Recombinant protein within human STAT1 aa 87-262. Positive control: Hela, 293, A431, NIH/3T3 Subcellular location: Cytoplasm, nucleus Database links: SwissProt: P42224 Human | P42225 Mouse Entrez Gene: 25124 Rat **Recommended Dilutions:** WB 1:1,000 IF-Cell 1:200

Storage Buffer: 1*PBS (pH7.4), 0.2% BSA, 40% Glycerol. Preservative: 0.05% Sodium Azide.

Storage Instruction:Store at +4 $^{\circ}$ C after thawing. Aliquot store at -20 $^{\circ}$ C or -80 $^{\circ}$ C. Avoid repeated freeze / thaw
cycles.

Purity: Immunogen affinity purified.

Hangzhou Huaan Biotechnology Co., Ltd.

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Applications:WB=Western blot IHC-P=Immunohistochemistry (paraffin) IF-Cell=Immunofluorescence (Cell) IF-Tissue=Immunofluorescence (Tissue) FC=Flow cytometry IP=Immunoprecipitation

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Images

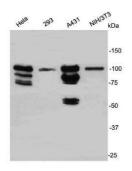


Fig1: Western blot analysis on cell lysates using anti- STAT1 rabbit polyclonal antibodies.

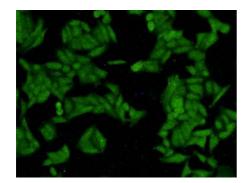


Fig2: ICC staining STAT1 in Hela cells (green). Cells were fixed in paraformaldehyde, permeabilised with 0.25% Triton X100/PBS.

Note: All products are "FOR RESEARCH USE ONLY AND ARE NOT INTENDED FOR DIAGNOSTIC OR THERAPEUTIC USE".

Background References

- 1. "Fibroblast growth factor receptor-induced phosphorylation of STAT1 at the Golgi apparatus without translocation to the nucleus." Citores L., Bai L., Sorensen V., Olsnes S. J. Cell. Physiol. 212:148-156(2007)
- "Analysis of STAT1 activation by six FGFR3 mutants associated with skeletal dysplasia undermines dominant role of STAT1 in FGFR3 signaling in cartilage." Krejci P., Salazar L., Kashiwada T.A., Chlebova K., Salasova A., Thompson L.M., Bryja V., Kozubik A., Wilcox W.R. PLoS ONE 3:E3961-E3961(2008)
- "A novel form of human STAT1 deficiency impairing early but not late responses to interferons." Kong X.F., Ciancanelli M., Al-Hajjar S., Alsina L., Zumwalt T., Bustamante J., Feinberg J., Audry M., Prando C., Bryant V., Kreins A., Bogunovic D., Halwani R., Zhang X.X., Abel L., Chaussabel D., Al-Muhsen S., Casanova J.L., Boisson-Dupuis S. Blood 116:5895-5906(2010)

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