3050 Spruce Street, St. Louis, MO 63103 USA
Tel: (800) 521-8956 (314) 771-5765 Fax: (800) 325-5052 (314) 771-5757
email: techservice@sial.com sigma-aldrich.com

Product Information

STAT6, GST-tagged, human recombinant, expressed in *Sf*9 cells

Catalog Number **SRP5143**
Storage Temperature –70 °C

Synonyms: STAT6B, STAT6C, D12S1644, IL-4-STAT

Product Description

The STAT6 protein is a member of the STAT family of transcription factors that are activated by cytokines and phosphorylated by the receptor associated kinases. STATs form homo or heterodimers that translocate to the cell nucleus where they act as transcription activators.[1] STAT6 plays a central role in exerting IL4 mediated biological responses. STAT6 induces the expression of BCL2L1/BCL-XL, which is responsible for the antiapoptotic activity of IL4. STAT6 mRNA has been detected in various tissues including peripheral blood lymphocytes, colon, intestine, ovary, prostate, thymus, spleen, kidney, liver, lung, and placenta. STAT6 is critically involved in Th2 immune response.[2]

Recombinant, full-length human STAT6 was expressed in *Sf*9 insect cells using an N-terminal GST tag. The gene accession number is BC075852. Recombinant protein stored in 50 mM Tris-HCl, pH 7.5, 150 mM NaCl, 10 mM glutathione, 0.1 mM EDTA, 0.25 mM DTT, 0.1 mM PMSF, and 25% glycerol.

Molecular mass: ~138 kDa

Purity: 70–95% (SDS-PAGE, see Figure 1)

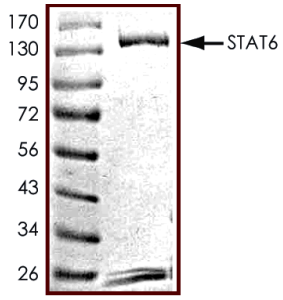
Precautions and Disclaimer

This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

The product ships on dry ice and storage at –70 °C is recommended. After opening, aliquot into smaller quantities and store at –70 °C. Avoid repeated handling and multiple freeze/thaw cycles.

Figure 1.
SDS-PAGE Gel of Typical Lot
70–95% (densitometry)



References

1. Weidinger, S. et al., Association of a STAT 6 haplotype with elevated serum IgE levels in a population based cohort of white adults. J. Med. Genet., **41**, 658-663 (2004).
2. Wang, W. et al., The susceptibility to experimental myasthenia gravis of STAT6-/- and STAT4-/- BALB/c mice suggests a pathogenic role of Th1 cells. J. Immun., **172**, 97-103 (2004).

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