

## STAT1

### Signal Transducer and Activator of Transcription 1

#### human, recombinant, Sf9 Insect cells

Cat. No.	Amount
PR-862	10 µg

For *in vitro* use only  
Quality guaranteed for 12 months  
Store at -80°C

#### Avoid freeze / thaw cycles

#### Form

Liquid. Supplied in 20 mM Tris-HCl pH 8.0, 20% glycerol, 100 mM KCl, 0.2 mM EDTA and 1 mM DTT.

#### Activity

1 ng is the amount sufficient for a gel mobility shift assay in a 20 µl reaction, 100 ng are sufficient for a protein-protein interaction assay detected by immuno-blot system.

#### Application

Use only for Research and not for Drug or Diagnostic purposes. STAT1 can be used for gel mobility shift assay, for protein-protein and small molecules-protein interactions assay.

#### Molecular Weight

90 kDa

#### Purity

> 95% by SDS-PAGE

#### Description

Signal transducer and activator of transcription (STAT) proteins are a family of latent cytoplasmic transcription factors involved in cytokine, hormone, and growth factor signal transduction. Seven members of the STAT family of transcription factors have been identified in mammalian cells: STAT1, STAT2, STAT3, STAT4, STAT5a, STAT5b, and STAT6. STAT proteins mediate broadly diverse biologic processes, including cell growth, differentiation, apoptosis, fetal development, transformation, inflammation, and immune response. Receptor-recruited STATs are phosphorylated on a single tyrosine residue in the carboxy terminal portion. The modified STATs are released from the cytoplasmic region of the receptor subunits to form homodimers or heterodimers through reciprocal interaction between the phosphotyrosine of one STAT and the SH2 domain of another.

Following dimerization, STATs rapidly translocate to the nucleus and interact with specific regulatory elements to induce target gene transcription. Recently, STAT-1 has been implicated in modulating pro- and anti-apoptotic genes following several stress-induced responses.

These effects are dependent on STAT-1 phosphorylation on serine-727 and require the C-terminal transactivation domain of STAT-1 to enhance its pro-apoptotic effect or inhibit its antiapoptotic effects. The STAT-1 C-terminal domain has been demonstrated to be important for protein-protein interaction with other transcriptional activators. The reports that STAT-1-deficient mice develop spontaneous and chemically induced tumours more rapidly compared to wild-type mice and that STAT-1-deficient cells are more resistant to agents that induce apoptosis strongly support the argument that STAT-1 acts as a tumour suppressor.

#### Selected References:

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