Data sheet

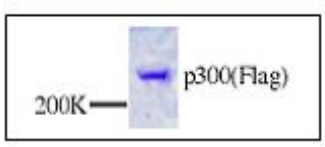
Jena Bioscience

p300

Tumor Suppressor Protein and Transcription Factor

human, recombinant, Sf9 insect cells

Cat. No.	Amount
PR-762	4 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, 100 mM KCl, 0.2 mM EDTA, 1 mM DTT, 20% glycerol.

Activity
1 unit equals 1 nanogram of purified protein. 1 unit is sufficient for a gel mobility shift assay in a 20 µl reaction; 50 units are sufficient for reconstituted transcription assay and 100 units are sufficient for a protein-protein interaction assay.

Purity
> 95% by SDS-PAGE

Description
Human p300 and CBP (CREB Binding Protein) are highly related transcriptional coactivators. Both proteins have been identified through protein interaction assays. In addition to interacting with variety of cellular factors and onco-proteins, loss of the wild type CBP alleles in isolated tumors suggests that CBP/p300 might serve as tumor suppressors. The ability of p300 to acetylate many transcription factors, including p53, E2F, TFIIE, and TFIIF etc. demonstrated a novel mechanism of targeted p300 regulation of gene expression.
The wild type p300 (2414 amino acid residues) was expressed in baculovirus system and purified by an affinity column in combination with FPLC chromatography.
Recombinant p300 can be used 1) for protein-protein interaction assay; 2) for in vitro transcription assay; 3) for in vitro acetylation assay; and 4) for cell growth assay.
Purified protein is greater than 95% homogeneous and contains no detectable proteases, DNase, and RNase activity.

Selected References:

Stein et al. (1990) Analysis of E1A-mediated growth regulation functions: binding of the 300-kilodalton cellular product correlates with E1A enhancer repression function and DNA synthesis-inducing activity. *J. Virol.* **64**:4421.
Eckner et al. (1994) Molecular cloning and functional analysis of the adenovirus E1A-associated 300-kD protein (p300) reveals a protein with properties of a transcriptional adaptor. *Genes & Dev.* **8**:869.
Kung et al. (2000) Gene dose-dependent control of hematopoiesis and hematologic tumor suppression by CBP. *Genes & Dev.* **14**:272.
Imhof et al. (1997) Acetylation of general transcription factors by histone acetyltransferases. *Curr. Biol.* **7**:689.
Gu et al. (1997) Activation of p53 sequence-specific DNA binding by acetylation of the p53 C-terminal domain. *Cell* **90**:595.
Martinez-Balbas et al. (2000) Regulation of E2F1 activity by acetylation. *EMBO J.* **19**:662.