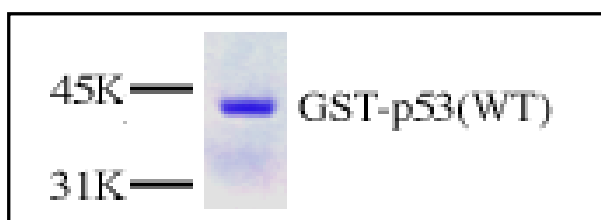


p53^{GST} (wild type)

Tumor Suppressor Protein and Transcription Factor, residues 1-81

human, recombinant, *E. coli*

Cat. No.	Amount
PR-782	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, 100 mM KCl, 0.2 mM EDTA, 1 mM DTT, 20% glycerol.

Activity

100 ng are sufficient for a protein-protein interaction assay.

Application

GST-p53 can be used for protein-protein interaction assay.

Purity

> 95% by SDS-PAGE

Description

p53 was identified as a tumor suppressor by showing that this protein has the ability to block transformation and to inhibit tumor cell growth. In addition, p53 is a transcription factor capable of regulating the expression of a subset of downstream genes. Mutation of two specific N-terminal residues in p53 (residues Leu²² and Trp²³) impairs the ability of p53 to transactivate and has been correlated with its ability to bind TAFII40 and TAFII60 (or TAFII31 and TAFII70) suggesting that one or both of these interactions is important for activation. Mutation of residues 22 and 23 to Ala does not affect binding to TBP, although mutation of these residues to charged amino acids has been reported to disrupt the p53-TBP interaction. Different mutations in p53 gene have been characterized in a variety of human cancers. Loss or mutation of p53 function is highly correlated with tumorigenesis.

GST-p53 is isolated from a strain of *E. coli* that contains the coding sequence of wild type human p53 (amino acids 1-81) under the control of T7 promoter.

The fusion protein is immunoreactive with the human p53-specific monoclonal antibody (cat.# AB-015).

Selected References:

- Finlay *et al.* (1989) The p53 proto-oncogene can act as a suppressor of transformation. *Cell* **57**:1083.
- Michalovitz *et al.* (1990) Conditional inhibition of transformation and of cell proliferation by a temperature-sensitive mutant of p53. *Cell* **62**:671.
- Baker *et al.* (1990) Suppression of human colorectal carcinoma cell growth by wild-type p53. *Science* **249**:912.
- Fields *et al.* (1990) Presence of a potent transcription activating sequence in the p53 protein. *Science* **249**:1046.
- Raycroft *et al.* (1990) Transcriptional activation by wild-type but not transforming mutants of the p53 anti-oncogene. *Science* **249**:1049.
- Hollstein *et al.* (1991) p53 mutations in human cancers. *Science* **253**:49.
- Bennett *et al.* (1992) Mutational spectra and immunohistochemical analyses of p53 in human cancers. *Chest* **101**:19S.
- Lin *et al.* (1994) Maturation of neurites in mixed cultures of spinal cord neurons and muscle cells from *Xenopus laevis* embryos followed with antibodies to neurofilament proteins. *Genes Dev.* **8**:1235.
- Thut *et al.* (1995) p53 transcriptional activation mediated by coactivators TAFII40 and TAFII60. *Science* **267**:100.
- Chang *et al.* (1995) Transactivation ability of p53 transcriptional activation domain is directly related to the binding affinity to TATA-binding protein. *J. Biol. Chem.* **270**:25014.
- Lin *et al.* (2005) p53 induces differentiation of mouse embryonic stem cells by suppressing Nanog expression. *Nature Cell Biology.* **7**:165.
- Baum *et al.* (2009) The prolyl cis/trans isomerase cyclophilin 18 interacts with the tumor suppressor p53 and modifies its functions in cell



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cycle regulation and apoptosis. *Oncogene* **28(44)**: 3915-3925.