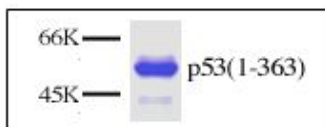


p53 (C-terminal deletion 1-363)

Tumor Suppressor Protein and Transcription Factor, residues 1-363, C-terminal deletion human, recombinant, Sf9 insect cells

Cat. No.	Amount
PR-760	5 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 and 20% glycerol.

Activity

1 ng is sufficient for a gel mobility shift assay in a 20 µl reaction, 50 ng are sufficient for reconstituted transcription assays and 100 ng are sufficient for a protein-protein interaction assay.

Application

Recombinant p53 can be used for
1) gel mobility shift assay or for a DNase I footprinting in the presence of double stranded DNA containing a consensus p53-binding sequence [5'-PuPuPuC(A/T)(T/A)GPyPyPy-3'],
2) *in vitro* transcription assay,
3) protein-protein interaction assay
4) cell growth assay.

Molecular Weight

52 kDa

Purity

> 95% by SDS-PAGE

Description

Human p53 protein is composed of 393 amino acid residues with several distinct regions. The N-terminal activation domain allows p53 protein to recruit the basal transcription machinery and activate the expression of target genes, whereas the core domain binds to target DNA in a sequence-specific manner and the majority of mutations found in human tumors occur in the region of the gene encoding this domain. The C-terminal domain is composed of predominantly basic residues and modification of the C-terminal basic domain, including acetylation, glycosylation and phosphorylation, is an essential mechanism for regulating p53 function.

The C-terminus-deleted p53 (amino acid 1-363) was expressed in baculovirus system and purified by an affinity column in combination with FPLC chromatography.

Purified protein is greater than 95% homogeneous and contains no detectable proteases, DNase, and RNase activity.

Selected References:

- Chen *et al.* (1996) p53 levels, functional domains, and DNA damage determine the extent of the apoptotic response of tumor cells. *Genes & Dev.* **10**:2438.
El-Deiry *et al.* (1992) Definition of a consensus binding site for p53. *Nature Genet.* **1**:45.
Hollstein *et al.* (1991) p53 mutations in human cancers. *Science* **253**:49.
Hupp *et al.* (1992) Regulation of the specific DNA binding function of p53. *Cell* **71**:875.
Gu *et al.* (1997) Activation of p53 sequence-specific DNA binding by acetylation of the p53 C-terminal domain. *Cell* **90**:595.
Mao *et al.* (2004) *Fbxw/Cdc4* is a p53-dependent, haploinsufficient tumour suppressor gene. *Nature* **432**:775.
Lin *et al.* (2005) p53 induces differentiation of mouse embryonic stem cells by suppressing Nanog expression. *Nature Cell Biology.* **7**:165.