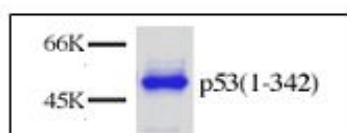


p53 (C-terminal deletion 1-342)

Tumor Suppressor Protein and Transcription Factor, residues 1-342, C-terminal deletion human, recombinant, Sf9 insect cells

Cat. No.	Amount
PR-761	5 μ g



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

Avoid freeze / thaw cycles

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

Activity

1 ng is sufficient for a gel mobility shift assay in a 20 μ l reaction, 50 ng are sufficient for reconstituted transcription assay 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) for cell growth assay.

Purity

> 95% by SDS-PAGE.

Description

Human p53 protein is composed of 393 amino acid residues with several distinct regions. In addition to that the N-terminal activation domain allows p53 protein to recruit the basal transcription machinery and activate the expression of target genes, 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, and modification of the C-terminal basic domain regulates p53 function, the tetramerization domain of p53 plays an important role in cell cycle. Disruption or loss of oligomerization function is associated with loss of cell cycle arrest. This mutant protein (with the deletion of C-terminal 51 residues including the entire basic domain and portion of tetramerization domain) can be used as a unique tool to study specific function of p53 related to the C-terminus.

The C-terminus-deleted p53 (amino acid 1-342) 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:

- Pellegata et al. (1995) The basic carboxy-terminal domain of human p53 is dispensable for both transcriptional regulation and inhibition of tumor cell growth. *Oncogene* **11**:337.
- 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.
- Ishioka et al. (1995) Mutational analysis of the carboxy-terminal portion of p53 using both yeast and mammalian cell assays *in vivo*. *Oncogene* **10**:1485.
- Waterman et al. (1996) An engineered four-stranded coiled coil substitutes for the tetramerization domain of wild-type p53 and alleviates transdominant inhibition by tumor-derived p53 mutants. *Cancer Res.* **56**:158.
- 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.