

Product datasheet for AP02357PU-N

p73 (TP73) pTyr99 Rabbit Polyclonal Antibody

Product data:

Product Type: Primary Antibodies

Applications: WB

Recommended Dilution: Suitable for use in Western blot (1:500~1:1000).

Reactivity: Human, Mouse

Host: Rabbit
Clonality: Polyclonal

Immunogen: The antiserum was produced against synthesized phosphopeptide derived from human p73

around the phosphorylation site of tyrosine 99 (S-P-YP-A-Q).

Specificity: The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using

epitope-specific phosphopeptide. The antibody against non-phosphopeptide was removed by chromatography using non-phosphopeptide corresponding to the phosphorylation site. p73 antibody (phospho-Tyr99) detects endogenous levels of p73 only when phosphorylated

at tyrosine 99.

Formulation: Phosphate buffered saline (without Mg2+ and Ca2+), pH 7.4, 150 mM NaCl, 0.02% Sodium

Azide and 50% Glycerol. State: Aff - Purified

State: Liquid purified Ig fraction.

Concentration: lot specific

Purification: Immunoaffinity chromatography.

Conjugation: Unconjugated

Storage: Store the antibody (in aliquots) at -20°C.

Avoid repeated freezing and thawing.

Stability: Shelf life: One year from despatch.

Gene Name: tumor protein p73

Database Link: Entrez Gene 7161 Human

015350



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Background:

p73 protein is a member of the p53 family of proteins. The tumor-suppressor protein p53 exhibits sequence specific DNA binding, directly interacts with various cellular and viral proteins, and induces cell cycle arrest in response to DNA damage. In response to signals generated by a variety of genotoxic stresses, e.g, UV irradiation or DNA damage, p53 is expressed and undergoes post translational modification that results in its accumulation in the nucleus. Activation of p53 leads to cell cycle arrest and in some cases to apoptosis, resulting in the inability of genetically damaged cells to proliferate. Thus, the p53 dependent pathways help to maintain genomic stability by eliminating damaged cells. The accumulation of high levels of p53 is a potential marker for malignancy. p73 protein is expressed in either full length form (p73 alpha 80 kDa) or a shorter (p73 beta 70 kDa) mRNA variant. p73 gene was predicted to encode a protein with significant amino acid sequence similarity to p53. Each of the p53 amino acid residues implicated in direct sequence specific DNA binding is conserved in p73.

p73 can activate the transcription of p53 responsive genes and inhibit cell growth in a p53 like manner by inducing apoptosis and cell cycle arrest. p73 has been mapped at chromosome 1p36.3, which is a locus of multiple suppressor genes for tumors including neuroblastoma and other cancers. c-Abl stimulates p73 mediated transactivation and apoptosis. This regulation of p73 by c-Abl in response to DNA damage was also demonstrated by a failure of ionizing radiation induced apoptosis after disruption of c-Abl-p73 interaction. The p73 protein can activate p53 responsive promoters and induce apoptosis in cells deficient in p53, which proves that inactivation of p53 family members may contribute to the biological properties of a subset of p53 mutants. The transcriptional activities of p53, p73 alpha, and p73 beta to induce their common cellular target genes differ among one another. Despite the high degree of similarity between p53 and p73, adenovirus E1B 55K, simian virus 40 T, and human papilloma virus E6 do not physically interact with p73, indicating that the functions of these two proteins may differ.

Synonyms:

OTTHUMP0000003479; OTTHUMP0000003480; P73

Product images:

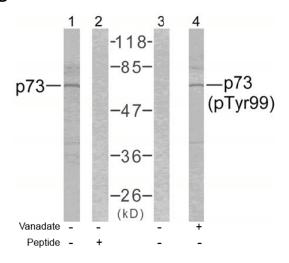


Figure 1. Western blot analysis of extracts from K562 cells using p73 antibody (Lane 1 and 2) and p73 (phospho-Tyr99) antibody (Lane 3 and 4).