

## **Product datasheet for TA329142**

## p53 (TP53) Rabbit Polyclonal Antibody

## **Product data:**

**Product Type:** Primary Antibodies

**Applications:** ELISA, WB

Recommended Dilution: ELISA, WB, CHIP

Reactivity: Human

Host: Rabbit

Isotype: IgG

Clonality: Polyclonal

**Immunogen:** The immunogen for anti-TP53 antibody: synthetic peptide directed towards the N terminal of

human TP53. Synthetic peptide located within the following region: PSQAMDDLMLSPDDIEQWFTEDPGPDEAPRMPEAAPPVAPAPAAPTPAAP

Formulation: Liquid. Purified antibody supplied in 1x PBS buffer with 0.09% (w/v) sodium azide and 2%

sucrose.

Note that this product is shipped as lyophilized powder to China customers.

Conjugation: Unconjugated

**Storage:** Store at -20°C as received.

**Stability:** Stable for 12 months from date of receipt.

Predicted Protein Size: 44 kDa

**Gene Name:** tumor protein p53

Database Link: NP 000537

Entrez Gene 7157 Human

P04637



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Background:

TP53 acts as a tumor suppressor in many tumor types; induces growth arrest or apoptosis depending on the physiological circumstances and cell type. Involved in cell cycle regulation as a trans-activator that acts to negatively regulate cell division by controlling a set of genes required for this process. This gene encodes tumor protein p53, which responds to diverse cellular stresses to regulate target genes that induce cell cycle arrest, apoptosis, senescence, DNA repair, or changes in metabolism. p53 protein is expressed at low level in normal cells and at a high level in a variety of transformed cell lines, where it's believed to contribute to transformation and malignancy. p53 is a DNA-binding protein containing transcription activation, DNA-binding, and oligomerization domains. It is postulated to bind to a p53binding site and activate expression of downstream genes that inhibit growth and/or invasion, and thus function as a tumor suppressor. Mutants of p53 that frequently occur in a number of different human cancers fail to bind the consensus DNA binding site, and hence cause the loss of tumor suppressor activity. Alterations of this gene occur not only as somatic mutations in human malignancies, but also as germline mutations in some cancer-prone families with Li-Fraumeni syndrome. Multiple p53 variants due to alternative promoters and multiple alternative splicing have been found. These variants encode distinct isoforms, which can regulate p53 transcriptional activity.

Synonyms: BCC7; LFS1; P53; TRP53

Note: Immunogen sequence homology: Human: 100%

**Protein Families:** Druggable Genome, Stem cell - Pluripotency, Transcription Factors

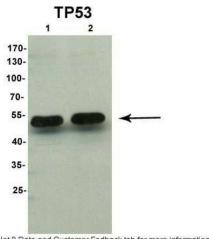
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Amyotrophic lateral sclerosis (ALS), Apoptosis, Basal cell carcinoma, Bladder cancer, Cell cycle, Chronic myeloid leukemia, Colorectal cancer, Endometrial cancer, Glioma, Huntington's disease, MAPK signaling pathway, Melanoma, Neurotrophin signaling pathway, Non-small cell lung cancer, p53 signaling pathway, Pancreatic cancer, Pathways in cancer, Prostate cancer,

Small cell lung cancer, Thyroid cancer, Wnt signaling pathway

## **Product images:**

**Protein Pathways:** 



See Immunoblot 2 Data and Customer Fedback tab for more information.

Sample Type :human cystic fibrosis bronchial epithelial cells; P53: 1/1 000Goat anti-rabbit-HRP: 1:5000





WB Suggested Anti-TP53 Antibody Titration: 0.2-1 ug/ml; ELISA Titer: 1:62500; Positive Control: 293T cell lysateTP53 is strongly supported by BioGPS gene expression data to be expressed in Human HEK293T cells

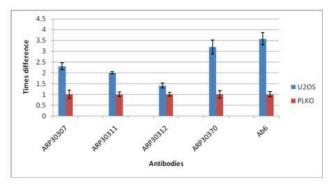


Figure 1. Binding of p53-specific antibodies to the p21 promoter.

Application: Chlp Assay Application data in forum Submitted by: Nick Barlev Department of Biochemistry University of Leicester U20S (p53+) cells were treated with 0.5 uM Doxorubicin for 14 hrs to induce DNA damage and hence activate p53. In parallel, PLKO cells (U2OS cells with stable shRNA-mediated knockdown of p53) were treated similarly and were used as negative control. Thedata for p21 promoter were normalised to actin (control for non-specific binding of DNA to the antibodies).