

p53 (Acetyl Lys372) Polyclonal Antibody
Catalog # AP63233**Specification****p53 (Acetyl Lys372) Polyclonal Antibody - Product Information**

Application	WB, IHC-P
Primary Accession	P04637
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal

p53 (Acetyl Lys372) Polyclonal Antibody - Additional Information**Gene ID** 7157**Other Names**

TP53; P53; Cellular tumor antigen p53; Antigen NY-CO-13; Phosphoprotein p53; Tumor suppressor p53

Dilution

WB~~Western Blot: 1/500 - 1/2000. IHC-p: 1:100-300 ELISA: 1/20000. Not yet tested in other applications.

IHC-P~~N/A

Format

Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.

Storage Conditions

-20°C

p53 (Acetyl Lys372) Polyclonal Antibody - Protein Information**Name** TP53**Synonyms** P53**Function**

Multifunctional transcription factor that induces cell cycle arrest, DNA repair or apoptosis upon binding to its target DNA sequence (PubMed:<[a href="http://www.uniprot.org/citations/11025664" target="_blank">11025664](http://www.uniprot.org/citations/11025664), PubMed:<[a href="http://www.uniprot.org/citations/12524540" target="_blank">12524540](http://www.uniprot.org/citations/12524540), PubMed:<[a href="http://www.uniprot.org/citations/12810724" target="_blank">12810724](http://www.uniprot.org/citations/12810724), PubMed:<[a href="http://www.uniprot.org/citations/15186775" target="_blank">15186775](http://www.uniprot.org/citations/15186775), PubMed:<[a href="http://www.uniprot.org/citations/15340061" target="_blank">15340061](http://www.uniprot.org/citations/15340061), PubMed:<[a href="http://www.uniprot.org/citations/17317671" target="_blank">17317671](http://www.uniprot.org/citations/17317671), PubMed:<[a href="http://www.uniprot.org/citations/17349958" target="_blank">17349958](http://www.uniprot.org/citations/17349958), PubMed:<[a href="http://www.uniprot.org/citations/19556538" target="_blank">19556538](http://www.uniprot.org/citations/19556538), PubMed:<[a href="http://www.uniprot.org/citations/20673990" target="_blank">20673990](http://www.uniprot.org/citations/20673990), PubMed:<[a href="http://www.uniprot.org/citations/20959462" target="_blank">20959462](http://www.uniprot.org/citations/20959462))

target="_blank">20959462, PubMed:22726440, PubMed:24051492, PubMed:24652652, PubMed:35618207, PubMed:36634798, PubMed:38653238, PubMed:9840937). Acts as a tumor suppressor in many tumor types; induces growth arrest or apoptosis depending on the physiological circumstances and cell type (PubMed:11025664, PubMed:12524540, PubMed:12810724, PubMed:15186775, PubMed:15340061, PubMed:17189187, PubMed:17317671, PubMed:17349958, PubMed:19556538, PubMed:20673990, PubMed:20959462, PubMed:22726440, PubMed:24051492, PubMed:24652652, PubMed:38653238, PubMed:9840937). Negatively regulates cell division by controlling expression of a set of genes required for this process (PubMed:11025664, PubMed:12524540, PubMed:12810724, PubMed:15186775, PubMed:15340061, PubMed:17317671, PubMed:17349958, PubMed:19556538, PubMed:20673990, PubMed:20959462, PubMed:22726440, PubMed:24051492, PubMed:24652652, PubMed:9840937). One of the activated genes is an inhibitor of cyclin-dependent kinases. Apoptosis induction seems to be mediated either by stimulation of BAX and FAS antigen expression, or by repression of Bcl-2 expression (PubMed:12524540, PubMed:17189187). Its pro-apoptotic activity is activated via its interaction with PPP1R13B/ASPP1 or TP53BP2/ASPP2 (PubMed:12524540). However, this activity is inhibited when the interaction with PPP1R13B/ASPP1 or TP53BP2/ASPP2 is displaced by PPP1R13L/iASPP (PubMed:12524540). In cooperation with mitochondrial PPIF is involved in activating oxidative stress-induced necrosis; the function is largely independent of transcription. Induces the transcription of long intergenic non-coding RNA p21 (lincRNA-p21) and lincRNA-Mkn1. LincRNA-p21 participates in TP53-dependent transcriptional repression leading to apoptosis and seems to have an effect on cell-cycle regulation. Implicated in Notch signaling cross-over. Prevents CDK7 kinase activity when associated to CAK complex in response to DNA damage, thus stopping cell cycle progression. Isoform 2 enhances the transactivation activity of isoform 1 from some but not all TP53-inducible promoters. Isoform 4 suppresses transactivation activity and impairs growth

suppression mediated by isoform 1. Isoform 7 inhibits isoform 1-mediated apoptosis. Regulates the circadian clock by repressing CLOCK-BMAL1-mediated transcriptional activation of PER2 (PubMed:24051492).

Cellular Location

Cytoplasm. Nucleus. Nucleus, PML body. Endoplasmic reticulum. Mitochondrion matrix. Cytoplasm, cytoskeleton, microtubule organizing center, centrosome Note=Recruited into PML bodies together with CHEK2 (PubMed:12810724) Translocates to mitochondria upon oxidative stress (PubMed:22726440) Translocates to mitochondria in response to mitomycin C treatment (PubMed:27323408). Competitive inhibition of TP53 interaction with HSPA9/MOT-2 by UBXN2A results in increased protein abundance and subsequent translocation of TP53 to the nucleus (PubMed:24625977) [Isoform 2]: Nucleus. Cytoplasm. Note=Localized mainly in the nucleus with minor staining in the cytoplasm [Isoform 4]: Nucleus. Cytoplasm. Note=Predominantly nuclear but translocates to the cytoplasm following cell stress [Isoform 8]: Nucleus. Cytoplasm. Note=Localized in both nucleus and cytoplasm in most cells. In some cells, forms foci in the nucleus that are different from nucleoli

Tissue Location

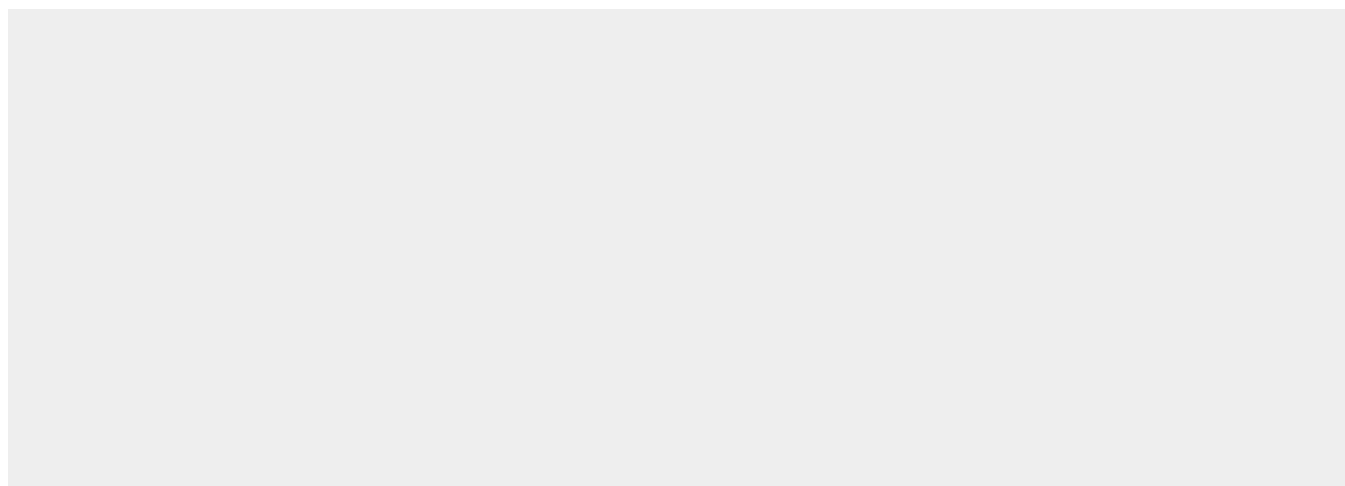
Ubiquitous. Isoforms are expressed in a wide range of normal tissues but in a tissue-dependent manner. Isoform 2 is expressed in most normal tissues but is not detected in brain, lung, prostate, muscle, fetal brain, spinal cord and fetal liver. Isoform 3 is expressed in most normal tissues but is not detected in lung, spleen, testis, fetal brain, spinal cord and fetal liver. Isoform 7 is expressed in most normal tissues but is not detected in prostate, uterus, skeletal muscle and breast. Isoform 8 is detected only in colon, bone marrow, testis, fetal brain and intestine. Isoform 9 is expressed in most normal tissues but is not detected in brain, heart, lung, fetal liver, salivary gland, breast or intestine

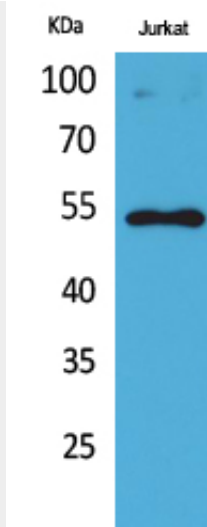
p53 (Acetyl Lys372) Polyclonal Antibody - Protocols

Provided below are standard protocols that you may find useful for product applications.

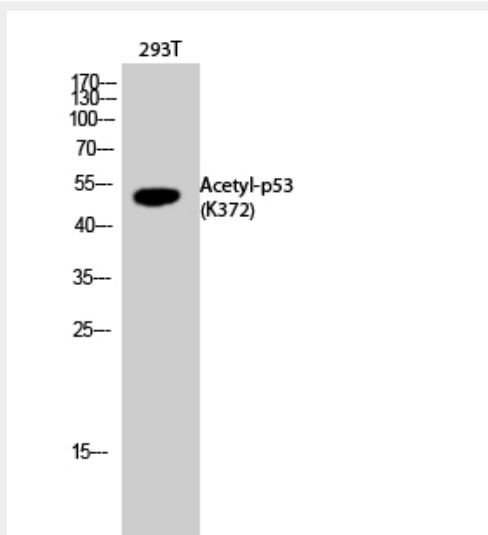
- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

p53 (Acetyl Lys372) Polyclonal Antibody - Images

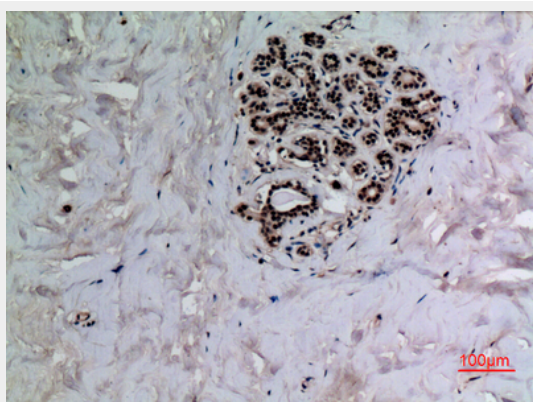




Western Blot analysis of Jurkat cells using Acetyl-p53 (K372) Polyclonal Antibody. Antibody was diluted at 1:2000. Secondary antibody was diluted at 1:20000



Western Blot analysis of 293T cells using Acetyl-p53 (K372) Polyclonal Antibody diluted at 1:2000. Secondary antibody was diluted at 1:20000



Immunohistochemical analysis of paraffin-embedded human-breast, antibody was diluted at 1:100

p53 (Acetyl Lys372) Polyclonal Antibody - Background

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. One of the activated genes is an inhibitor of cyclin-dependent kinases. Apoptosis induction seems to be mediated either by stimulation of BAX and FAS antigen expression, or by repression of Bcl-2 expression. In cooperation with mitochondrial PPIF is involved in activating oxidative stress-induced necrosis; the function is largely independent of transcription. Induces the transcription of long intergenic non-coding RNA p21 (lincRNA-p21) and lincRNA- Mkl1. LincRNA-p21 participates in TP53-dependent transcriptional repression leading to apoptosis and seems to have an effect on cell-cycle regulation. Implicated in Notch signaling cross-over. Prevents CDK7 kinase activity when associated to CAK complex in response to DNA damage, thus stopping cell cycle progression. Isoform 2 enhances the transactivation activity of isoform 1 from some but not all TP53-inducible promoters. Isoform 4 suppresses transactivation activity and impairs growth suppression mediated by isoform 1. Isoform 7 inhibits isoform 1-mediated apoptosis. Regulates the circadian clock by repressing CLOCK-ARNTL/BMAL1- mediated transcriptional activation of PER2 (PubMed:24051492).