

**p53 Antibody (Sumoylation Site Specific)
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP2505b**

Specification

p53 Antibody (Sumoylation Site Specific) - Product Information

Application	WB, IHC-P,E
Primary Accession	P04637
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	43653
Antigen Region	364-393

p53 Antibody (Sumoylation Site Specific) - Additional Information

Gene ID 7157

Other Names

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

Target/Specificity

This p53 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 364-393 amino acids from human p53.

Dilution

WB~~1:2000
IHC-P~~1:100

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

p53 Antibody (Sumoylation Site Specific) is for research use only and not for use in diagnostic or therapeutic procedures.

p53 Antibody (Sumoylation Site Specific) - 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:[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:[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:[38653238](#), 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-Mkln1. 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

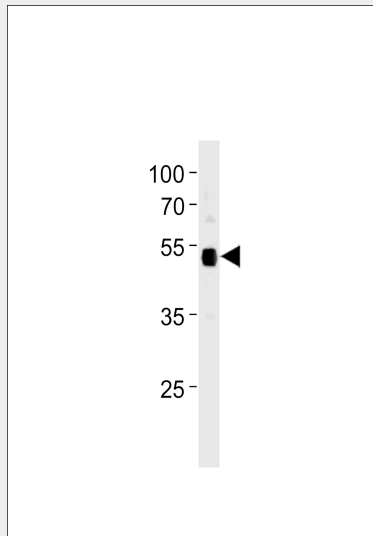
Ubiquitous. Isoforms are expressed in a wide range of normal tissues but in a tissue-dependent manner. Isoform 2 is expressed in most normal tissues but is not detected in brain, lung, prostate, muscle, fetal brain, spinal cord and fetal liver. Isoform 3 is expressed in most normal tissues but is not 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 Antibody (Sumoylation Site Specific) - Protocols

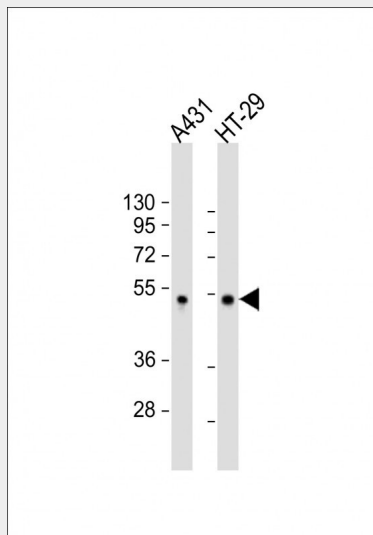
Provided below are standard protocols that you may find useful for product applications.

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

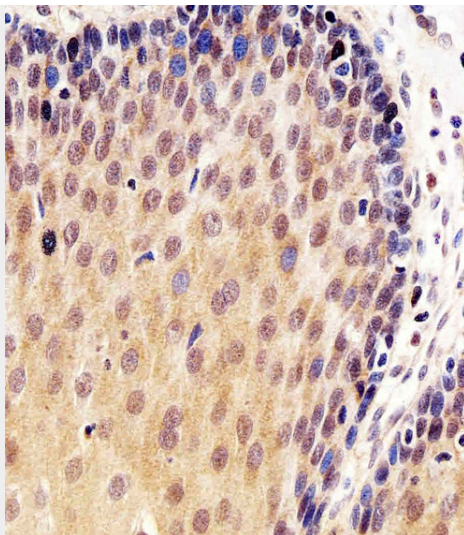
p53 Antibody (Sumoylation Site Specific) - Images



TP53 Antibody (C-term) (Cat. #AP2505b) western blot analysis in 293 cell line lysates (35ug/lane). This demonstrates the TP53 antibody detected the TP53 protein (arrow).



All lanes : Anti-Sumo-site Antibody (p53) at 1:2000 dilution Lane 1: A431 whole cell lysate Lane 2: HT-29 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-Rabbit IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 43 kDa Blocking/Dilution buffer: 5% NFDM/TBST.



Immunohistochemical analysis of paraffin-embedded H. esophagus section using p53 Antibody (Sumoylation Site Specific)(Cat#AP2505b). AP2505b was diluted at 1:100 dilution. A undiluted biotinylated goat polyvalent antibody was used as the secondary, followed by DAB staining.

p53 Antibody (Sumoylation Site Specific) - Background

Tumor protein p53, a nuclear protein, plays an essential role in the regulation of cell cycle, specifically in the transition from G0 to G1. It is found in very low levels in normal cells, however, in a variety of transformed cell lines, it is expressed in high amounts, and believed to contribute to transformation and malignancy. P53 is subject to modification by conjugation of SUMO-1. A p53 mutant deficient for MDM2 binding is poorly sumoylated *in vivo* compared to wild-type p53. Overexpression of MDM2 increases the level of p53 sumoylation, which is further stimulated by expression of ARF. These results show that p53 sumoylation is regulated by MDM2- and ARF-mediated nucleolar targeting.

p53 Antibody (Sumoylation Site Specific) - References

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- Melchior F, et al. *Cell Cycle*. 2002 Jul-Aug;1(4):245-9.
- Kahyo T, et al. *Mol Cell*. 2001 Sep;8(3):713-8.
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- Muller S, et al. *J Biol Chem*. 2000 May 5;275(18):13321-9.
- Gostissa M, et al. *EMBO J*. 1999 Nov 15;18(22):6462-71.
- Rodriguez MS, et al. *EMBO J*. 1999 Nov 15;18(22):6455-61.