

Anti-p53 (ac Lys305) (RABBIT) Antibody
p53 K305 Ac Antibody
Catalog # ASR5587

Specification

Anti-p53 (ac Lys305) (RABBIT) Antibody - Product Information

Host	Rabbit
Conjugate	Unconjugated
Target Species	Human
Reactivity	Human
Clonality	Polyclonal
Application	WB, E, I, LCI
Application Note	p53 K305 Ac Antibody has been tested by dot blot and western blotting and is suitable for ELISA. Specific conditions for reactivity should be optimized by the end user. Expect a band approximately ~43.6 kDa in size corresponding to p53 by western blotting in the appropriate cell lysate or extract.
Physical State	Liquid (sterile filtered)
Buffer	0.02 M Potassium Phosphate, 0.15 M Sodium Chloride, pH 7.2
Immunogen	Anti-p53 K305 Ac antibody was prepared from whole rabbit serum produced by repeated immunizations with a synthetic acetylated peptide surrounding Lysine 305 of human p53.
Preservative	0.01% (w/v) Sodium Azide

Anti-p53 (ac Lys305) (RABBIT) Antibody - Additional Information

Gene ID 7157

Other Names
7157

Purity

Anti-p53 K305 Ac antibody is directed against human p53 protein. p53 K305 Ac Antibody was affinity purified from monospecific antiserum by immunoaffinity chromatography. A BLAST analysis used to suggest reactivity with this protein from human based on 100% homology for the immunizing sequence.

Storage Condition

Store vial at -20° C prior to opening. Aliquot contents and freeze at -20° C or below for extended storage. Avoid cycles of freezing and thawing. Centrifuge product if not completely clear after standing at room temperature. This product is stable for several weeks at 4° C as an undiluted liquid. Dilute only prior to immediate use.

Precautions Note

This product is for research use only and is not intended for therapeutic or diagnostic applications.

Anti-p53 (ac Lys305) (RABBIT) 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: [11025664](http://www.uniprot.org/citations/11025664), PubMed: [12524540](http://www.uniprot.org/citations/12524540), PubMed: [12810724](http://www.uniprot.org/citations/12810724), PubMed: [15186775](http://www.uniprot.org/citations/15186775), PubMed: [15340061](http://www.uniprot.org/citations/15340061), PubMed: [17317671](http://www.uniprot.org/citations/17317671), PubMed: [17349958](http://www.uniprot.org/citations/17349958), PubMed: [19556538](http://www.uniprot.org/citations/19556538), PubMed: [20673990](http://www.uniprot.org/citations/20673990), PubMed: [20959462](http://www.uniprot.org/citations/20959462), PubMed: [22726440](http://www.uniprot.org/citations/22726440), PubMed: [24051492](http://www.uniprot.org/citations/24051492), PubMed: [24652652](http://www.uniprot.org/citations/24652652), PubMed: [35618207](http://www.uniprot.org/citations/35618207), PubMed: [36634798](http://www.uniprot.org/citations/36634798), PubMed: [38653238](http://www.uniprot.org/citations/38653238), PubMed: [9840937](http://www.uniprot.org/citations/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](http://www.uniprot.org/citations/11025664), PubMed: [12524540](http://www.uniprot.org/citations/12524540), PubMed: [12810724](http://www.uniprot.org/citations/12810724), PubMed: [15186775](http://www.uniprot.org/citations/15186775), PubMed: [15340061](http://www.uniprot.org/citations/15340061), PubMed: [17189187](http://www.uniprot.org/citations/17189187), PubMed: [17317671](http://www.uniprot.org/citations/17317671), PubMed: [17349958](http://www.uniprot.org/citations/17349958), PubMed: [19556538](http://www.uniprot.org/citations/19556538), PubMed: [20673990](http://www.uniprot.org/citations/20673990), PubMed: [20959462](http://www.uniprot.org/citations/20959462), PubMed: [22726440](http://www.uniprot.org/citations/22726440), PubMed: [24051492](http://www.uniprot.org/citations/24051492), PubMed: [24652652](http://www.uniprot.org/citations/24652652), PubMed: [38653238](http://www.uniprot.org/citations/38653238), PubMed: [9840937](http://www.uniprot.org/citations/9840937)). Negatively regulates cell division by controlling expression of a set of genes required for this process (PubMed: [11025664](http://www.uniprot.org/citations/11025664), PubMed: [12524540](http://www.uniprot.org/citations/12524540), PubMed: [12810724](http://www.uniprot.org/citations/12810724), PubMed: [15186775](http://www.uniprot.org/citations/15186775), PubMed: [15340061](http://www.uniprot.org/citations/15340061), PubMed: [17317671](http://www.uniprot.org/citations/17317671), PubMed: [17349958](http://www.uniprot.org/citations/17349958), PubMed: [19556538](http://www.uniprot.org/citations/19556538), PubMed: [20673990](http://www.uniprot.org/citations/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-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-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

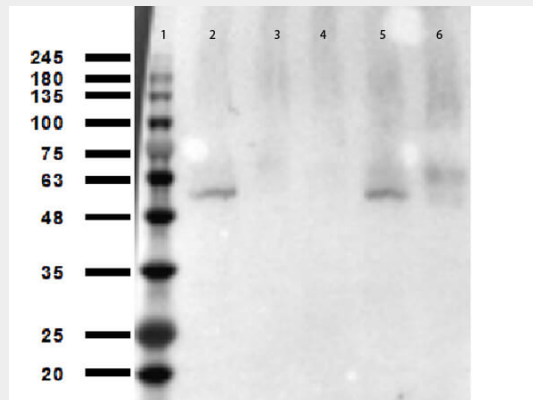
Anti-p53 (ac Lys305) (RABBIT) Antibody - Protocols

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](#)

Anti-p53 (ac Lys305) (RABBIT) Antibody - Images



Western Blot of RABBIT Anti-p53 (ac Lys305) Antibody. Lane 1: Ladder Opal PreStained (p/n MB-210-0500). Lane 2: MCF-7 (p/n W09-000-360). Lane 3: MCF-7 Nuclear Extract (p/n W09-001-A85). Lane 4: Jurkat Lysate (p/n W09-001-370). Lane 5: HeLa Whole Cell Lysate (p/n W09-000-364). Lane 6: A431 Whole Cell Lysate (p/n W09-000-361). Loaded at 10ug. Blocking: BlockOut Buffer (p/n MB-073) for 30 min at RT. Primary Antibody: Rb anti-p53 K305 Ac at 1ug/mL for 4°C overnight. Secondary Antibody: Gt-a-Rb HRP (p/n 611-103-122) at 1:70,000 in MB-073 for 30 min at RT. Expected: ~53 kDa.

Anti-p53 (ac Lys305) (RABBIT) Antibody - Background

TP53 (tumor suppressor gene p53) is one of the most well-studied genes that suppresses tumor formation and renders protection against DNA damage by inducing cell cycle arrest, DNA repair, or apoptosis. TP53 signaling is triggered through numerous cellular events ranging from DNA damage to hypoxia, stress and a plethora of other causes. Upon activation, p53 acts as zinc-containing transcriptional regulator and initiates a cascade of events that determines the cellular outcome including cell cycle arrest, apoptosis, senescence, DNA repair, development, differentiation and tissue homeostasis. Cell cycle arrest is induced by p53 via trans-activating genes such as p21 (CDK-inhibitor 1, cyclin dependent kinase) and others. Interestingly, p53 itself is capable of triggering cellular responses (survival or induced cell death) as well. Mutations or deletions in the TP53 gene are present in nearly 50% of human cancers, and primarily results in impaired tumor suppressor function. Anti-p53 (ac Lys305) antibody is ideal for researchers interested in developmental biology, cell growth and cancer research.