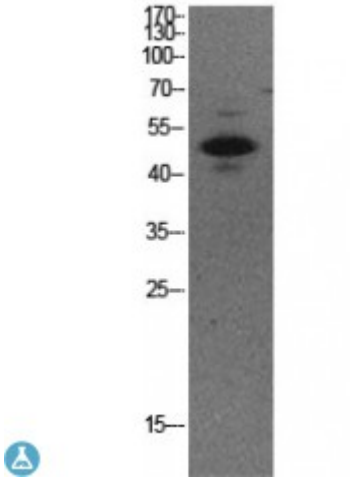
Anti-p53 (Acetyl K373) antibody



Description	Rabbit polyclonal to Acetyl-p53 (K373).

Model	STJ97354
Host	Rabbit
Reactivity	Human
Applications	ELISA, WB
Immunogen	Synthesized peptide derived from the human p53
Immunogen Region	acetylation site of K373.
Gene ID	7157
Gene Symbol	TP53
Dilution range	WB 1:500-1:2000ELISA 1:20000
Specificity	Acetyl-p53 (K373) Polyclonal Antibody detects endogenous levels of p53 protein only when acetylated at K373.
Tissue Specificity	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 detec
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.

Note	For Research Use Only (RUO).
Protein Name	Cellular tumor antigen p53 Antigen NY-CO-13 Phosphoprotein p53 Tumor suppressor p53
Clonality	Polyclonal
Conjugation	Unconjugated
Isotype	IgG
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Concentration	1 mg/ml
Storage Instruction	Store at -20°C, and avoid repeat freeze-thaw cycles.
Database Links	HGNC:11998OMIM:133239
Alternative Names	Cellular tumor antigen p53 Antigen NY-CO-13 Phosphoprotein p53 Tumor suppressor p53
Function	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-Mkln1. LincRNA-p21 participates in TP53-dependent transcriptional repression leading to apoptosis and seem to have to 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 .
Sequence and Domain Family	The nuclear export signal acts as a transcriptional repression domain. The TADI and TADII motifs (residues 17 to 25 and 48 to 56) correspond both to 9aaTAD motifs which are transactivation domains present in a large number of yeast and animal transcription factors.
Cellular Localization	Cytoplasm. Nucleus. Nucleus, PML body. Endoplasmic reticulum. Mitochondrion matrix. Interaction with BANP promotes nuclear localization. Recruited into PML bodies together with CHEK2. Translocates to mitochondria upon oxidative stress. Translocates to mitochondria in response to mitomycin C treatment . Isoform 1: Nucleus. Cytoplasm. Predominantly nuclear but localizes to the cytoplasm when expressed with isoform 4.. Isoform 2: Nucleus. Cytoplasm. Localized mainly in the nucleus with minor staining in the cytoplasm.. Isoform 3: Nucleus. Cytoplasm. Localized in the nucleus in most cells but found in the cytoplasm in some cells.. Isoform 4: Nucleus. Cytoplasm. Predominantly nuclear but translocates to the cytoplasm

following cell stress.. Isoform 7: Nucleus. Cytoplasm. Localized mainly in the nucleus with minor staining in the cytoplasm.. Isoform 8: Nucleus. Cytoplasm. Localized in both nucleus and cytoplasm in most cells. In some cells, forms foci in the nucleus that are different from nucleoli.. Isoform 9: Cytoplasm.

Post-translational Modifications

Acetylated. Acetylation of Lys-382 by CREBBP enhances transcriptional activity. Deacetylation of Lys-382 by SIRT1 impairs its ability to induce proapoptotic program and modulate cell senescence. Deacetylation by SIRT2 impairs its ability to induce transcription activation in a AKT-dependent manner. Phosphorylation on Ser residues mediates transcriptional activation. Phosphorylated by HIPK1 . Phosphorylation at Ser-9 by HIPK4 increases repression activity on BIRC5 promoter. Phosphorylated on Thr-18 by VRK1. Phosphorylated on Ser-20 by CHEK2 in response to DNA damage, which prevents ubiquitination by MDM2. Phosphorylated on Ser-20 by PLK3 in response to reactive oxygen species (ROS), promoting p53/TP53-mediated apoptosis. Phosphorylated on Thr-55 by TAF1, which promotes MDM2-mediated degradation. Phosphorylated on Ser-33 by CDK7 in a CAK complex in response to DNA damage. Phosphorylated on Ser-46 by HIPK2 upon UV irradiation. Phosphorylation on Ser-46 is required for acetylation by CREBBP. Phosphorylated on Ser-392 following UV but not gamma irradiation. Phosphorylated on Ser-15 upon ultraviolet irradiation; which is enhanced by interaction with BANP. Phosphorylated by NUAK1 at Ser-15 and Ser-392; was intially thought to be mediated by STK11/LKB1 but it was later shown that it is indirect and that STK11/LKB1-dependent phosphorylation is probably mediated by downstream NUAK1 . It is unclear whether AMP directly mediates phosphorylation at Ser-15. Phosphorylated on Thr-18 by isoform 1 and isoform 2 of VRK2. Phosphorylation on Thr-18 by isoform 2 of VRK2 results in a reduction in ubiquitination by MDM2 and an increase in acetylation by EP300. Stabilized by CDK5-mediated phosphorylation in response to genotoxic and oxidative stresses at Ser-15, Ser-33 and Ser-46, leading to accumulation of p53/TP53, particularly in the nucleus, thus inducing the transactivation of p53/TP53 target genes. Phosphorylated by DYRK2 at Ser-46 in response to genotoxic stress. Phosphorylated at Ser-315 and Ser-392 by CDK2 in response to DNA-damage. Dephosphorylated by PP2A-PPP2R5C holoenzyme at Thr-55. SV40 small T antigen inhibits the dephosphorylation by the AC form of PP2A.; May be O-glycosylated in the C-terminal basic region. Studied in EB-1 cell line. Ubiquitinated by MDM2 and SYVN1, which leads to proteasomal degradation . Ubiquitinated by RFWD3, which works in cooperation with MDM2 and may catalyze the formation of short polyubiquitin chains on p53/TP53 that are not targeted to the proteasome . Ubiquitinated by MKRN1 at Lys-291 and Lys-292, which leads to proteasomal degradation . Deubiquitinated by USP10, leading to its stabilization . Ubiquitinated by TRIM24, RFFL, RNF34 and RNF125, which leads to proteasomal degradation . Ubiquitination by TOPORS induces degradation . Deubiquitination by USP7, leading to stabilization . Isoform 4 is monoubiquitinated in an MDM2-independent manner . Ubiquitinated by RFWD2, which leads to proteasomal degradation . Ubiquitination and subsequent proteasomal degradation is negatively regulated by CCAR2 . Monomethylated at Lys-372 by SETD7, leading to stabilization and increased transcriptional activation. Monomethylated at Lys-370 by SMYD2, leading to decreased DNA-binding activity and subsequent transcriptional regulation activity. Lys-372 monomethylation prevents interaction with SMYD2 and subsequent monomethylation at Lys-370. Dimethylated at Lys-373 by

EHMT1 and EHMT2. Monomethylated at Lys-382 by KMT5A, promoting interaction with L3MBTL1 and leading to repress transcriptional activity. Dimethylation at Lys-370 and Lys-382 diminishes p53 ubiquitination, through stabilizing association with the methyl reader PHF20. Demethylation of dimethylated Lys-370 by KDM1A prevents interaction with TP53BP1 and represses TP53-mediated transcriptional activation.; Sumoylated with SUMO1. Sumoylated at Lys-386 by UBC9.