



<http://www.uniprot.org/citations/20959462> target="\_blank">20959462</a>, PubMed:<a href="http://www.uniprot.org/citations/22726440" target="\_blank">22726440</a>, PubMed:<a href="http://www.uniprot.org/citations/24051492" target="\_blank">24051492</a>, PubMed:<a href="http://www.uniprot.org/citations/24652652" target="\_blank">24652652</a>, PubMed:<a href="http://www.uniprot.org/citations/9840937" target="\_blank">9840937</a>). 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 (PubMed:<a href="http://www.uniprot.org/citations/11025664" target="\_blank">11025664</a>, PubMed:<a href="http://www.uniprot.org/citations/12524540" target="\_blank">12524540</a>, PubMed:<a href="http://www.uniprot.org/citations/12810724" target="\_blank">12810724</a>, PubMed:<a href="http://www.uniprot.org/citations/15186775" target="\_blank">15186775</a>, PubMed:<a href="http://www.uniprot.org/citations/15340061" target="\_blank">15340061</a>, PubMed:<a href="http://www.uniprot.org/citations/17317671" target="\_blank">17317671</a>, PubMed:<a href="http://www.uniprot.org/citations/17349958" target="\_blank">17349958</a>, PubMed:<a href="http://www.uniprot.org/citations/19556538" target="\_blank">19556538</a>, PubMed:<a href="http://www.uniprot.org/citations/20673990" target="\_blank">20673990</a>, PubMed:<a href="http://www.uniprot.org/citations/20959462" target="\_blank">20959462</a>, PubMed:<a href="http://www.uniprot.org/citations/22726440" target="\_blank">22726440</a>, PubMed:<a href="http://www.uniprot.org/citations/24051492" target="\_blank">24051492</a>, PubMed:<a href="http://www.uniprot.org/citations/24652652" target="\_blank">24652652</a>, PubMed:<a href="http://www.uniprot.org/citations/9840937" target="\_blank">9840937</a>). 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:<a href="http://www.uniprot.org/citations/12524540" target="\_blank">12524540</a>, PubMed:<a href="http://www.uniprot.org/citations/17189187" target="\_blank">17189187</a>). Its pro-apoptotic activity is activated via its interaction with PPP1R13B/ASPP1 or TP53BP2/ASPP2 (PubMed:<a href="http://www.uniprot.org/citations/12524540" target="\_blank">12524540</a>). However, this activity is inhibited when the interaction with PPP1R13B/ASPP1 or TP53BP2/ASPP2 is displaced by PPP1R13L/iASPP (PubMed:<a href="http://www.uniprot.org/citations/12524540" target="\_blank">12524540</a>). 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:<a href="http://www.uniprot.org/citations/24051492" target="\_blank">24051492</a>).

### 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

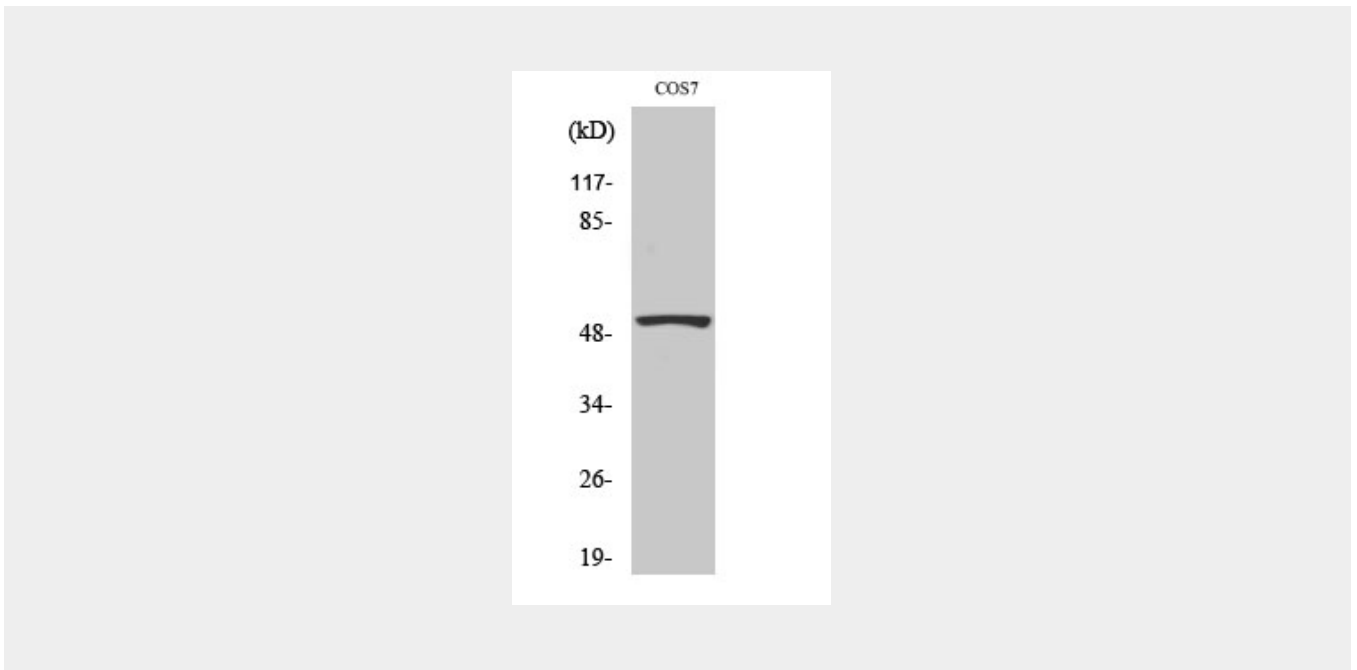
Ubiquitous. Isoforms are expressed in a wide range of normal tissues but in a tissue-dependent manner. Isoform 2 is expressed in most normal tissues but is not detected in brain, lung, prostate, muscle, fetal brain, spinal cord and fetal liver. Isoform 3 is expressed in most normal tissues but is not 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 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 Polyclonal Antibody - Images**



### **p53 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- Mkl1n1. 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).