

p53 Mutant Form (361 - 371), Pab 421
Synthetic Peptide
Catalog # SP2289a**Specification**

p53 Mutant Form (361 - 371), Pab 421 - Product Information

Primary Accession [Q29537](#)
Other Accession [P41685](#), [P02340](#), [Q95330](#), [P10361](#), [Q36006](#)
Sequence [NH2-KKGQSTSRHKK-CONH2](#)

p53 Mutant Form (361 - 371), Pab 421 - Additional Information**Other Names**

Cellular tumor antigen p53, Tumor suppressor p53, TP53, P53

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

p53 Mutant Form (361 - 371), Pab 421 - Protein Information

Name TP53

Synonyms 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. Its pro-apoptotic activity is activated via its interaction with PPP1R13B/ASPP1 or TP53BP2/ASPP2 (By similarity). However, this activity is inhibited when the interaction with PPP1R13B/ASPP1 or TP53BP2/ASPP2 is displaced by PPP1R13L/iASPP (By similarity). In cooperation with mitochondrial PPIF is involved in activating oxidative stress-induced necrosis; the function is largely independent of transcription. Prevents CDK7 kinase activity when associated to CAK complex in response to DNA damage, thus stopping cell cycle progression. 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. Regulates the circadian clock by repressing CLOCK-BMAL1-mediated transcriptional activation of PER2.

Cellular Location

Cytoplasm {ECO:0000250|UniProtKB:P04637}. Nucleus {ECO:0000250|UniProtKB:P04637}. Nucleus, PML body {ECO:0000250|UniProtKB:P04637}. Endoplasmic reticulum {ECO:0000250|UniProtKB:P04637}. Mitochondrion matrix {ECO:0000250|UniProtKB:P04637}. Cytoplasm, cytoskeleton, microtubule organizing center, centrosome {ECO:0000250|UniProtKB:P04637} Note=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 (By similarity). Competitive inhibition of TP53 interaction with HSPA9/MOT-2 by UBXN2A results in increased protein abundance and subsequent translocation of TP53 to the nucleus (By similarity) {ECO:0000250|UniProtKB:P04637}

p53 Mutant Form (361 - 371), Pab 421 - Images