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Nuclear Export Signal, NES p53

Synthetic Peptide
Catalog # SP2227b

Specification

Nuclear Export Signal, NES p53 - Product Information

Primary Accession	P41685
Other Accession	P02340, Q8SPZ3, P10361, Q95330, P56423
Sequence	NH2-FRELNEALELKD-COOH

Nuclear Export Signal, NES p53 - Additional Information

Gene ID 493847

Other Names
Cellular tumor antigen p53, Tumor suppressor p53, TP53, TRP53

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.

Nuclear Export Signal, NES p53 - Protein Information

Name TP53

Synonyms TRP53

Function
Multifunctional transcription factor that induces cell cycle arrest, DNA repair or apoptosis upon binding to its target DNA sequence. Acts as a tumor suppressor in many tumor types; induces growth arrest or apoptosis depending on the physiological circumstances and cell type. Negatively regulates cell division by controlling expression of 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 (By similarity). 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}

Nuclear Export Signal, NES p53 - Images