

Recombinant Rat P53 Protein

For Research Use Only

1. Basic Product Information

Catalog No.: REP52268

Size: 10ug, 100ug

Protein Name: Cellular tumor antigen p53 (Tumor suppressor p53)

Synonym: Tp53, P53

UniProt ID: P10361

UniProt Link: www.uniprot.org/uniprotkb/P10361/entry

Species Source: Rat

Expression System: HEK293T eukaryotic cell expression system Sequence

Length: 391 AA

Molecular Weight: 43 KD

Protein Tag: C-terminal His6

2. Amino Acid Sequence

MEDSQSDMSIELPLSQETFSCWLKLLPPDDILPTTATGSPNSMEDLFLPQDVAELLEGPEEALQVSA
PAAQEPGTEAPAPVAPASATPWPLSSSVPSQKTYQGNYGFHLGFLQSGTAKSVMCTYSISLNKLFC
QLAKTCPVQLWVTSTPPPGTRVRAMAIYKKSQHMTEVVRRCPPHHERCSDGDGLAPPQHLIRVEGN
PYAEYLDDRQTFRHSVVVPYEPPEVGSDYTTIHYYKMCNSSCMGGMNRRPILTIITLEDSSGNLLGR
DSFEVRVCACPGRDRRTEENFRKKEEHCPPELPPGSAKRALPTSTSSSPQQKKKPLDGEYFTLKIR
GRERFEMFRELNEALELKDARAAEESGDSRAHSSYPKTKKGQSTSRHKKPMIKKVGPDSD

3. Buffer Composition

Liquid format: PBS-based buffer containing 50% glycerol. Glycerol concentration can be customized upon customer request; please state special requirements when placing your order.

Lyophilized format: Buffer before lyophilization is PBS supplemented with 6% trehalose.

4. Storage Conditions



Upon receipt, store the product at -20 °C or -80 °C. Aliquot the product for on-demand use to preserve protein activity and prevent denaturation and inactivation caused by repeated freeze-thaw cycles.

5. Product Description

This product is recombinant Cellular tumor antigen p53 (Tumor suppressor p53) produced using the HEK293T eukaryotic cell expression system. It is suitable for in-vitro functional assays, protein-protein interaction studies, antibody preparation and other biomedical research applications.

Protein 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-Mkn1. 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-ARNTL/BMAL1-mediated transcriptional activation of PER2.

Subcellular Localization: Cytoplasm . Nucleus . Nucleus, PML body . Endoplasmic reticulum . Mitochondrion matrix . Cytoplasm, cytoskeleton, microtubule organizing center, centrosome . 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).

6. Notes

- Avoid repeated freeze-thaw cycles to maintain protein stability and biological activity.
- Please specify special buffer-composition requirements at the time of order placement.
- For research use only. Not intended for clinical diagnosis, therapeutic use or commercial manufacturing.