

DESCRIPTION

Source *E. coli*-derived human p53 protein
Glu2-Asp393, with an N-termina 6-His SUMO tag
Accession # P04637.4

N-terminal Sequence Analysis Met-His tag

Predicted Molecular Mass 55.7 kDa

SPECIFICATIONS

SDS-PAGE 66-73 kDa, under reducing conditions.

Activity Measured by its binding ability in a functional ELISA.
Recombinant Human p53 His-tag binds to Recombinant Human MDM2/HDM2 GST-tag (Catalog # 11802-DM) with an ED₅₀ of 12.50-125.0 ng/mL

Endotoxin Level <1.0 EU per 1 µg of the protein by the LAL method.

Purity >85%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.

Formulation Supplied as a 0.2 µm filtered solution in HEPES, NaCl, TCEP, ZnCl₂ and Glycerol with Trehalose. See Certificate of Analysis for details.

PREPARATION AND STORAGE

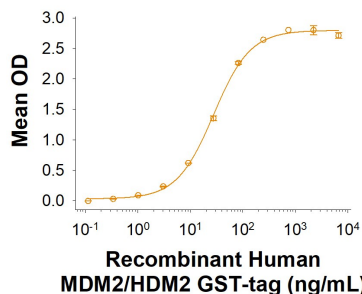
Shipping The product is shipped with dry ice or equivalent. Upon receipt, store it immediately at the temperature recommended below.

Stability & Storage Use a manual defrost freezer and avoid repeated freeze-thaw cycles.

- 6 months from date of receipt, -20 to -70 °C as supplied.
- 3 months, -20 to -70 °C under sterile conditions after opening.

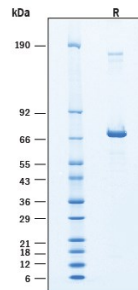
DATA

Binding Activity



Recombinant Human p53 His-tag Protein Binding Activity. Measured by its binding ability in a functional ELISA. Recombinant Human p53 His-tag Protein (Catalog # 11816-TP) binds to Recombinant Human MDM2/HDM2 GST-tag (Catalog # 11802-DM) with an ED₅₀ of 12.50-125.0 ng/mL.

SDS-Page



Recombinant Human p53 His-tag Protein SDS-PAGE. 2 µg/lane of Recombinant Human p53 His-tag Protein (Catalog # 11816-TP) was resolved with SDS-PAGE under reducing (R) condition and visualized by Coomassie® Blue staining, showing bands at 66-73 kDa.

BACKGROUND

The tumor suppressor protein p53 is a sequence-specific transcription factor that plays a central role in maintaining genomic integrity by coordinating cellular responses to DNA damage, oncogenic stress, hypoxia, and metabolic imbalance. p53 is encoded by the TP53 gene, which is the most frequently altered gene in human cancer (1). Under physiological, non-stress conditions, p53 protein levels are kept low through continuous ubiquitin-mediated degradation, primarily controlled by the E3 ubiquitin ligase MDM-2 (2). Human p53 is a ~53 kDa protein composed of an N-terminal transactivation domain, a proline-rich region, a central DNA-binding domain, an oligomerization domain, and a C-terminal regulatory domain (3). Upon cellular stress, post-translational modifications such as phosphorylation and acetylation disrupt p53–MDM-2 interactions, leading to rapid stabilization and activation of p53 (2, 4). Activated p53 induces transcriptional programs that promote cell-cycle arrest, DNA repair, senescence, ferroptosis, or apoptosis, depending on cellular context and damage severity (1, 3). In addition to its canonical transcription-dependent roles, p53 also exerts transcription-independent functions in the cytoplasm and mitochondria, where it directly influences apoptotic signaling, mitochondrial metabolism, and redox homeostasis (4, 5). These diverse activities position p53 as a master regulator of cell fate decisions and stress adaptation. p53 function is tightly modulated by an extensive network of cofactors, ubiquitin ligases, deubiquitinases, chromatin regulators, and metabolic enzymes, allowing for fine-tuned and context-specific signaling outputs (3, 5). Loss of p53 function through mutation, deletion, or functional inactivation enables tumor initiation and progression by allowing survival and proliferation of genetically unstable cells. While many cancers harbor missense mutations within the p53 DNA-binding domain, a substantial fraction of tumors retain wild-type p53 but suppress its activity through overexpression of negative regulators such as MDM-2 and MDM-X (2, 6). As a result, restoration of p53 signaling has emerged as a major therapeutic strategy, driving the development of small-molecule MDM-2 inhibitors, mutant-p53 reactivators, and p53-based combination therapies (6, 7). Recombinant human p53 is therefore an essential research reagent for studies of tumor suppressor biology, transcriptional regulation, protein–protein interactions, ubiquitin-proteasome pathways, stress signaling, and anticancer drug discovery.

References:

1. Kasthuber, E.R. and Lowe, S.W. (2017) *Cell* **170**:1062.
2. Kung, C.-P. and Weber, J.D. (2022) *Front. Cell Dev. Biol.* **10**:818744.
3. Liu, Y. *et al.* (2024) *Cancer Cell* **42**:946.
4. Hafner, A. *et al.* (2019) *Nat. Rev. Mol. Cell Biol.* **20**:199.
5. Bykov, V.J.N. *et al.* (2018) *Nat. Rev. Cancer* **18**:89.
6. Munisamy, M. *et al.* (2021) *Am. J. Cancer Res.* **11**:5762.
7. Sabapathy, K. and Lane, D.P. (2020) *Nat. Rev. Clin. Oncol.* **17**:471.