PRODUCT INFORMATION

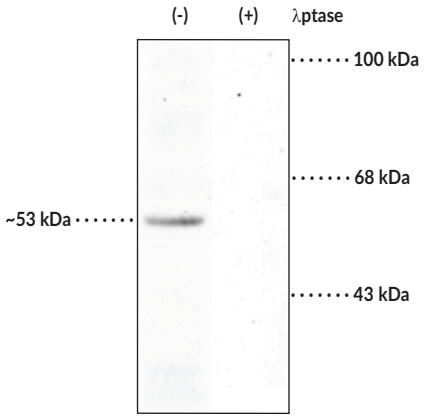
p53 (Phospho-Ser^{392}) Polyclonal Antibody

Item No. 10004807

Overview and Properties

Contents:	This vial contains 10 µg of affinity-purified IgG polyclonal antibody.
Synonyms:	Antigen NY-CO-13, Cellular Tumor Antigen p53, Phosphoprotein p53, Transformation-related Protein 53, Tumor Protein p53, Tumor Suppressor p53
Immunogen:	Synthetic peptide corresponding to amino acid residues surrounding phospho-Ser^{392} of human p53 conjugated to keyhole limpet hemocyanin (KLH)
Molecular Weight:	~53 kDa
Cross Reactivity:	(+) Human, rat p53 (phospho-Ser^{392})
Species Reactivity:	(+) Human, rat
Form:	Liquid
Storage:	-20°C (as supplied)
Stability:	≥1 year
Storage Buffer:	100 µl of 10 mM HEPES, pH 7.5, with 50% glycerol, 0.1% BSA, and 150 mM sodium chloride
Host:	Rabbit
Isotype:	IgG
Applications:	Western blot (WB); the recommended starting dilution is 1:1,000. Other applications were not tested, therefore optimal working concentration/dilution should be determined empirically.

Image



(-): WB of rat rat brain nuclear fraction lysate showing specific immunolabeling of the ~53 kDa p53 phosphorylated at Ser^{392}.
(+): Phosphospecificity is shown where the immunolabeling is completely eliminated by blot treatment with λ phosphatase (λptase, 1,200 units for 30 minutes).

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Description

p53 is a transcription factor and tumor suppressor encoded by *TP53* in humans with roles in cell cycle arrest, apoptosis, senescence, differentiation, and DNA repair.[1,2] It is composed of two N-terminal intrinsically disordered transactivation domains (TADs), a proline-rich domain (PRD), a DNA-binding domain (DBD), a tetramerization domain (TD), and an intrinsically disordered C-terminal domain.[1] p53 is ubiquitously expressed, with protein levels increasing under conditions of extra- or intracellular stress, such as DNA damage, oncogene activation, oxidative stress, or hypoxia.[3] It is activated and translocated to the nucleus in response to cellular stress *via* post-translational modifications, such as phosphorylation, methylation, and acetylation, where it binds p53 consensus DNA-binding elements and regulates transcription of its target genes in a cell-, tissue-, and stress signal type-specific manner.[1,3] p53 is phosphorylated at Ser^{392} by IκB kinase β (IKKβ) in glutamine-deficient cancer cells.[4] Loss-of-function and/or gain-of-function mutations in *TP53* occur in approximately 50% of human cancers.[2] Cayman's p53 (Phospho-Ser^{392}) Polyclonal Antibody can be used for Western blot (WB) applications. This antibody recognizes p53 at ~53 kDa from human and rat samples.

References

1. Liu, Y., Tavana, O., and Gu, W. p53 Modifications: Exquisite decorations of the powerful guardian. *J. Mol. Cell Biol.* **11(7)**, 564-577 (2019).
2. Stein, Y., Rotter, V., and Aloni-Grinstein, R. Gain-of-function mutant p53: All the roads lead to tumorigenesis. *Int. J. Mol. Sci.* **20(24)**, 6197 (2019).
3. Yue, X., Zhao, Y., Xu, Y., *et al.* Mutant p53 in cancer: Accumulation, gain-of-function and therapy. *J. Mol. Biol.* **429(11)**, 1595-1606 (2017).
4. Gabra, M.B.I., Yang, Y., Lowman, X.H., *et al.* IKKβ activates p53 to promote cancer cell adaptation to glutamine deprivation. *Oncogenesis* **7(11)**, 93 (2018).

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