www.neobiolab.com info@neobiolab.com 888.754.5670, +1 617.500.7103 United States 0800.088.5164, +44 020.8123.1558 United Kingdom

## Phospho-p53-S9

Reactivity: Human

Tested applications:WB IF

Recommended Dilution: WB 1:500 - 1:2000 IF 1:50 - 1:200

Observed MW:Refer to Figures

Immunogen:

A phospho specific peptide corresponding to residues surrounding S9 of human P53

Store at -20. Avoid freeze / thaw cycles. Buffer: PBS with 0.02% sodium azide, 50% glycerol,

pH7.3.

Concentration:

beik

Synonym:

P53; LFS1; TRP53; FLJ92943; TP53;

Catalog #:AP0085

**Antibody Type:** 

Polyclonal Antibody

Species: Rabbit

Gene ID:7157

Isotype:IgG

Swiss Prot:P04637

Purity: Affinity purification

For research use only.

## Background:

The p53 tumor suppressor protein plays a major role in cellular response to DNA damage and other genomic aberrations. Activation of p53 can lead to either cell cycle arrest and DNA repair or apoptosis (1). p53 is phosphorylated at multiple sites in vivo and by several different protein kinases in vitro (2,3). DNA damage induces phosphorylation of p53 at Ser15 and Ser20 and leads to a reduced interaction between p53 and its negative regulator, the oncoprotein MDM2 (4). MDM2 inhibits p53 accumulation by targeting it for ubiquitination and proteasomal degradation (5,6). p53 can be phosphorylated by ATM, ATR, and DNA-PK at Ser15 and Ser37. Phosphorylation impairs the ability of MDM2 to bind p53, promoting both the accumulation and activation of p53 in response to DNA damage (4,7). Chk2 and Chk1 can phosphorylate p53 at Ser20, enhancing its tetramerization, stability, and activity (8,9). p53 is phosphorylated at Ser392 in vivo (10,11) and by CAK in vitro (11). Phosphorylation of p53 at Ser392 is increased in human tumors (12) and has been reported to influence the growth suppressor function, DNA binding, and transcriptional activation of p53 (10,13,14). p53 is phosphorylated at Ser6 and Ser9 by CK1 and CK1 both in vitro and in vivo (13,15). Phosphorylation of p53 at Ser46 regulates the ability of p53 to induce apoptosis (16). Acetylation of p53 is mediated by p300 and CBP acetyltransferases. Inhibition of deacetylation suppressing MDM2 from recruiting HDAC1 complex by p19 (ARF) stabilizes p53. Acetylation appears to play a positive role in the accumulation of p53 protein in stress response (17).

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