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TP53

Reactivity: Human

Tested applications:WB IF CHIP CHIPseq

Recommended Dilution:WB 1:500 - 1:2000 IF 1:50 - 1:200 ChIP 1:20 - 1:100 ChIPseq 1:20 - 1:100

Calculated MW:44kDa

Observed MW: Refer to Figures

Immunogen:

Recombinant protein of human TP53

Storage Buffer:

Store at -20. Avoid freeze / thaw cycles. Buffer: PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

Concentration:

beik

Synonym:

TP53;FLJ92943;LFS1;TRP53;p53 ;

Catalog #:A0263

Antibody Type:

Polyclonal Antibody

Species: Rabbit

Gene ID:7157

Isotype: IgG

Swiss Prot:P04637

Purity: Affinity purification

For research use only.

Background:

The TP53 tumor suppressor protein plays a major role in cellular response to DNA damage and other genomic aberrations. Activation of TP53 can lead to either cell cycle arrest and DNA repair or apoptosis (1). TP53 is phosphorylated at multiple sites in vivo and by several different protein kinases in vitro (2,3). DNA damage induces phosphorylation of TP53 at Ser15 and Ser20 and leads to a reduced interaction between TP53 and its negative regulator, the oncoprotein MDM2 (4). MDM2 inhibits TP53 accumulation by targeting it for ubiquitination and proteasomal degradation (5,6). TP53 can be phosphorylated by ATM, ATR, and DNA-PK at Ser15 and Ser37. Phosphorylation impairs the ability of MDM2 to bind TP53, promoting both the accumulation and activation of TP53 in response to DNA damage (4,7). Chk2 and Chk1 can phosphorylate TP53 at Ser20, enhancing its tetramerization, stability, and activity (8,9). TP53 is phosphorylated at Ser392 in vivo (10,11) and by CAK in vitro (11). Phosphorylation of TP53 at Ser392 is increased in human tumors (12) and has been reported to influence the growth suppressor function, DNA binding, and transcriptional activation of TP53 (10,13,14). TP53 is phosphorylated at Ser6 and Ser9 by CK1 and CK1 both in vitro and in vivo (13,15). Phosphorylation of TP53 at Ser46 regulates the ability of TP53 to induce apoptosis (16). Acetylation of TP53 is mediated by p300 and CBP acetyltransferases. Inhibition of deacetylation suppressing MDM2 from recruiting HDAC1 complex by p19 (ARF) stabilizes TP53. Acetylation appears to play a positive role in the accumulation of TP53 protein in stress response (17).

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