

#9285 Store at -20°C

Phospho-p53 (Ser6) Antibody



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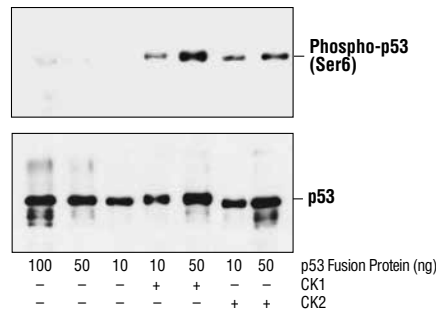
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Applications	Species Cross-Reactivity*	Molecular Wt.	Source
W, IP, IC Endogenous	H, Mk, (Hm)	53 kDa	Rabbit**

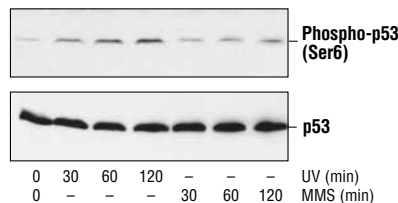
Background: The p53 tumor suppressor protein plays a major role in cellular response to DNA damage and other genomic aberrations. Activation of p53 can lead to either cell cycle arrest and DNA repair or apoptosis (1). p53 is phosphorylated at multiple sites *in vivo* and by several different protein 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 CK1δ and CK1ε 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). Following DNA damage, human p53 becomes acetylated at Lys382 (Lys379 in mouse) *in vivo* to enhance p53-DNA binding (18). Deacetylation of p53 occurs through interaction with the SIRT1 protein, a deacetylase that may be involved in cellular aging and the DNA damage response (19).

Specificity/Sensitivity: Phospho-p53 (Ser6) Antibody detects endogenous levels of p53 only when phosphorylated at serine 6. The antibody does not cross-react with p53 phosphorylated at other sites.

Source/Purification: Polyclonal antibodies are produced by immunizing animals with a synthetic phosphopeptide corresponding to residues surrounding Ser6 of hamster p53. Antibodies are purified by protein A and peptide affinity chromatography.



Western blot analysis of a p53 fusion protein, untreated or phosphorylated by CK1 or CK2, using Phospho-p53 (Ser6) Antibody (upper) or p53 Antibody #9282 (lower).



Western blot analysis of extracts from COS cells treated with UV or MMS for the indicated times, using Phospho-p53 (Ser6) Antibody (upper) or p53 Antibody #9282 (lower).

Entrez-Gene ID #7157
UniProt ID #P04637

Storage: Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA and 50% glycerol. Store at -20°C. Do not aliquot the antibody.

*Species cross-reactivity is determined by western blot.

**Anti-rabbit secondary antibodies must be used to detect this antibody.

Recommended Antibody Dilutions:

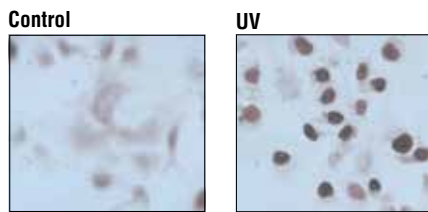
Western blotting	1:1000
Immunoprecipitation	1:100
Immunocytochemistry (ABC)	1:1000

For application specific protocols please see the web page for this product at www.cellsignal.com.

Please visit www.cellsignal.com for a complete listing of recommended companion products.

IMPORTANT: For western blots, incubate membrane with diluted antibody in 5% w/v BSA, 1X TBS, 0.1% Tween®20 at 4°C with gentle shaking, overnight.

Tween® is a registered trademark of ICI Americas, Inc.



Immunocytochemical analysis of COS cells, untreated or UV-treated, using Phospho-p53 (Ser6) Antibody.

Background References:

- (1) Levine, A.J. (1997) *Cell* 88, 323-331.
- (2) Meek, D.W. (1994) *Semin. Cancer Biol.* 5, 203-210.
- (3) Milczarek, G.J. et al. (1997) *Life Sci.* 60, 1-11.
- (4) Shieh, S.Y. et al. (1997) *Cell* 91, 325-334.
- (5) Chehab, N.H. et al. (1999) *Proc. Natl. Acad. Sci. USA* 96, 13777-13782.
- (6) Honda, R. et al. (1997) *FEBS Lett.* 420, 25-27.
- (7) Tibbetts, R.S. et al. (1999) *Genes Dev.* 13, 152-157.
- (8) Shieh, S.Y. et al. (1999) *EMBO J.* 18, 1815-1823.
- (9) Hirao, A. et al. (2000) *Science* 287, 1824-1827.
- (10) Hao, M. et al. (1996) *J. Biol. Chem.* 271, 29380-29385.
- (11) Lu, H. et al. (1997) *Mol. Cell. Biol.* 17, 5923-5934.
- (12) Ullrich, S.J. et al. (1993) *Proc. Natl. Acad. Sci. USA* 90, 5954-5958.
- (13) Kohn, K.W. (1999) *Mol. Biol. Cell* 10, 2703-2734.
- (14) Lohrum, M. and Scheidtmann, K.H. (1996) *Oncogene* 13, 2527-2539.
- (15) Knippschild, U. et al. (1997) *Oncogene* 15, 1727-1736.
- (16) Oda, K. et al. (2000) *Cell* 102, 849-862.
- (17) Ito, A. et al. (2001) *EMBO J.* 20, 1331-1340.
- (18) Sakaguchi, K. et al. (1998) *Genes Dev.* 12, 2831-2841.
- (19) Solomon, J.M. et al. (2006) *Mol. Cell. Biol.* 26, 28-38.