Revision 1

#9919 Store at -20°C

Phospho-p53 Antibody Sampler Kit

()

Cell Signaling TECHNOLOGY®

Orders: 877-616-CELL (2355) orders@cellsignal.com

Support: 877-678-TECH (8324)

Web: info@cellsignal.com cellsignal.com

3 Trask Lane | Danvers | Massachusetts | 01923 | USA

For Research Use Only. Not for Use in Diagnostic Procedures.

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Phospho-p53 (Ser6) Antibody	9285	20 µl	53 kDa	Rabbit
Phospho-p53 (Ser9) Antibody	9288	20 µl	53 kDa	Rabbit
Phospho-p53 (Ser15) (16G8) Mouse mAb	9286	20 µl	53 kDa	Mouse IgG1
Phospho-p53 (Thr18) Antibody	2529	20 µl	53 kDa	Rabbit
Phospho-p53 (Ser20) Antibody	9287	20 µl	53 kDa	Rabbit
Phospho-p53 (Ser46) Antibody	2521	20 µl	53 kDa	Rabbit
Phospho-p53 (Thr81) Antibody	2676	20 µl	53 kDa	Rabbit
Phospho-p53 (Ser392) Antibody	9281	20 µl	53 kDa	Rabbit
p53 (7F5) Rabbit mAb	2527	20 µl	53 kDa	Rabbit IgG
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat

Please visit cellsignal.com for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

Description

The Phospho-p53 Antibody Sampler Kit provides a fast and economical means of evaluating multiple phosphorylation sites of p53 protein. The kit contains enough primary and secondary antibodies to perform two Western blot experiments.

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.

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).

Background References

1. Levine, A.J. (1997) *Cell* 88, 323-31.
2. Meek, D.W. (1994) *Semin Cancer Biol* 5, 203-10.
3. Milczarek, G.J. et al. (1997) *Life Sci* 60, 1-11.
4. Shieh, S.Y. et al. (1997) *Cell* 91, 325-34.
5. Chehab, N.H. et al. (1999) *Proc Natl Acad Sci U S A* 96, 13777-82.
6. Honda, R. et al. (1997) *FEBS Lett* 420, 25-7.
7. Tibbetts, R.S. et al. (1999) *Genes Dev* 13, 152-7.
8. Shieh, S.Y. et al. (1999) *EMBO J* 18, 1815-23.
9. Hirao, A. et al. (2000) *Science* 287, 1824-7.
10. Hao, M. et al. (1996) *J Biol Chem* 271, 29380-5.

11. Lu, H. et al. (1997) *Mol Cell Biol* 17, 5923-34.
12. Ullrich, S.J. et al. (1993) *Proc Natl Acad Sci U S A* 90, 5954-8.
13. Kohn, K.W. (1999) *Mol Biol Cell* 10, 2703-34.
14. Lohrum, M. and Scheidtmann, K.H. (1996) *Oncogene* 13, 2527-39.
15. Knippschild, U. et al. (1997) *Oncogene* 15, 1727-36.
16. Oda, K. et al. (2000) *Cell* 102, 849-62.
17. Ito, A. et al. (2001) *EMBO J* 20, 1331-40.
18. Sakaguchi, K. et al. (1998) *Genes Dev* 12, 2831-41.
19. Solomon, J.M. et al. (2006) *Mol Cell Biol* 26, 28-38.

Trademarks and Patents

Limited Uses