Store at -20°C

#40106

PhosphoPlus® p53 (Ser15) Antibody Duet

Cell Signaling TECHNOLOGY®

Support: +1-978-867-2388 (U.S.)
www.cellsignal.com/support

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

Entrez-Gene ID #7157
UniProt ID #P04637

New 05/21

For Research Use Only. Not For Use In Diagnostic Procedures.

Products Included	Product #	Quantity	Mol. Wt.	Isotype
p53 (7F5) Rabbit mAb	2527	100 µl	53 kDa	Rabbit IgG
Phospho-p53 (Ser15) (E9Y4U) Rabbit mAb	82530	100 µl	53 kDa	Rabbit IgG

See www.cellsignal.com for individual component applications, species cross-reactivity, dilutions, and additional application protocols.

Description: PhosphoPlus® Duets from Cell Signaling Technology (CST) provide a means to assess protein activation status. Each Duet contains an activation-state and total protein antibody to your target of interest. These antibodies have been selected from CST's product offering based upon superior performance in specified applications.

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: p53 (7F5) Rabbit mAb detects endogenous levels of total p53 protein. This antibody binding has been mapped to the amino terminus region of human p53 protein. Phospho-p53 (Ser15) (E9Y4U) Rabbit mAb recognizes endogenous levels of p53 protein only when phosphorylated at Ser15.

Source/Purification: Monoclonal antibodies are produced by immunizing animals with a full-length human p53 fusion protein and a synthetic phosphopeptide corresponding to residues surrounding Ser15 of human p53 protein.

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 antibodies.*

For product specific protocols and a complete listing of recommended companion products please see the product web page at www.cellsignal.com.

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.

Thank you for your recent purchase. If you would like to provide a review visit cellsignal.com/comments.

www.cellsignal.com

Applications: **W**—Western **IP**—Immunoprecipitation **IHC**—Immunohistochemistry **ChIP**—Chromatin Immunoprecipitation **IF**—Immunofluorescence **F**—Flow cytometry **E-P**—ELISA-Peptide **Species Cross-Reactivity:** **H**—human **M**—mouse **R**—rat **Hm**—hamster **Mk**—monkey **Mi**—mink **C**—chicken **Dm**—D. melanogaster **X**—Xenopus **Z**—zebrafish **B**—bovine **Dg**—dog **Pg**—pig **Sc**—S. cerevisiae **Ce**—C. elegans **Hr**—Horse **All**—all species expected Species enclosed in parentheses are predicted to react based on 100% homology.