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## Tumor Suppressor Inactivation Antibody Sampler Kit



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Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
COPS5 Antibody	6895	40 µl	37 kDa	Rabbit
p27 Kip1 (D69C12) XP <sup>®</sup> Rabbit mAb	3686	40 µl	27 kDa	Rabbit IgG
Smad4 Antibody	9515	40 µl	70 kDa	Rabbit
p53 (7F5) Rabbit mAb	2527	40 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 Tumor Suppressor Inactivation Antibody Sampler Kit provides a fast and economical means of evaluating the role of COPS5 in the inhibition of the tumor suppressors p27 Kip1, p53, and Smad4. The kit contains enough primary antibody to perform four western blot experiments with each primary antibody.
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	COPS5/CSN5/Jab1 (c-Jun activation domain-binding protein-1) was originally identified as a transcriptional coactivator of c-Jun and subsequently discovered to be a fifth component and integral part of the CSN (1). As the catalytic center of the CSN, COPS5 is able to integrate multiple functions of the CSN complex such as cell cycle control, transcription, and DNA damage response by regulating the activity of CRLs through deneddylation of cullins (2). Indeed, COPS5 harbors an Mpr1-Pad1-N-terminal (MPN) domain with an embedded Jab1/CSN5 MPN domain metalloenzyme (JAMM) motif that is essential for the CSN isopeptidase activity responsible for deneddylation of CRLs. COPS5 is an evolutionarily conserved 38 kDa protein in humans, mice, fission yeast, and plants, which suggests that it is critical to cell survival and proliferation. A role for COPS5 as a positive regulator of cellular proliferation is supported by evidence that it functionally inactivates several key tumor suppressors such as p53, RUNX3, Smad4, and p27 Kip1 through altered subcellular localization, degradation, and deneddylation (3-7). These findings are underscored by the observation that COPS5 overexpression has been identified in a number of different tumor types and has been implicated in the initiation and progression of several types of cancer (8). Moreover, COPS5 targets such as p53 and p27 (9,10).
Background References	<ol> <li>Claret, F.X. et al. (1996) Nature 383, 453-7.</li> <li>Wei, N. et al. (2008) Trends Biochem Sci 33, 592-600.</li> <li>Bech-Otschir, D. et al. (2001) EMBO J 20, 1630-9.</li> <li>Oh, W. et al. (2006) J Biol Chem 281, 17457-65.</li> <li>Wan, M. et al. (2002) EMBO Rep 3, 171-6.</li> <li>Tomoda, K. et al. (2002) J Biol Chem 277, 2302-10.</li> <li>Kim, J.H. et al. (2009) J Cell Biochem 107, 557-65.</li> <li>Shackleford, T.J. and Claret, F.X. (2010) Cell Div 5, 26.</li> <li>Tian, L. et al. (2010) Oncogene 29, 6125-37.</li> <li>Tomoda, K. et al. (2004) J Biol Chem 279, 43013-8.</li> </ol>
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