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Progesterone Receptor Signaling Antibody Sampler Kit



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

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
Phospho-p44/42 MAPK (Erk1/2) (Thr202/Tyr204) (D13.14.4E) XP [®] Rabbit mAb	4370	40 µl	44, 42 kDa	Rabbit IgG
Phospho-Progesterone Receptor (Ser190) Antibody	3171	40 µl	90, 118 kDa	Rabbit
Phospho-Progesterone Receptor (Ser345) Antibody	12783	40 µl	90 (PR-A), 118 (PR-B) kDa	Rabbit
Progesterone Receptor A/B (D8Q2J) XP [®] Rabbit mAb	8757	40 µl	90 (PR-A), 118 (PR-B) kDa	Rabbit IgG
Progesterone Receptor B (C1A2) Rabbit mAb	3157	40 µl	118 kDa	Rabbit
Phospho-Src Family (Tyr416) (D49G4) Rabbit mAb	6943	40 µl	60 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	Progesterone Receptor Signaling Antibody Sampler Kit provides an economical means of detecting total and active levels of progesterone receptor (PR) as well as the active forms of PR downstream targets. The kit contains enough primary antibody to perform four western blots per 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	Human progesterone receptor (PR) is expressed as two forms: the full length PR-B and the short form PR-A. PR-A lacks the first 164 amino acid residues of PR-B (1,2). Both PR-A and PR-B are ligand activated, but differ in their relative ability to activate target gene transcription (3,4). The activity of PR is regulated by phosphorylation; at least seven serine residues are phosphorylated in its amino-terminal domain. Three sites (Ser81, Ser102, and Ser162) are unique to full length PR-B, while other sites (Ser190, Ser294, Ser345, and Ser400) are shared by both isoforms (5). Phosphorylation of PR-B at Ser190 (equivalent to Ser26 of PR-A) is catalyzed by CDK2 (6). Mutation of Ser190 results in decreased activity of PR (7), suggesting that the phosphorylation at Ser190 may be critical to its biological function. Research studies have demonstrated ligand-dependent phosphorylation of PR-B at Ser345 is catalyzed by MAPK and plays an important role in mediating the proliferation of breast cancer cells. Investigators have shown that Ser345-phosphorylated PR-B associates with Sp1 to regulate EGFR and p21 transcription (8). PR signaling has been shown to crosstalk with other kinase signaling cascades. Upon stimulation and the subsequent interaction with estrogen receptor α and c-Src, PR-B has been shown to promote the activation of the Src/p21 ^{ras} /Erk pathway (9).
Background References	 Evans, R.M. (1988) Science 240, 889-95. Kastner, P. et al. (1990) EMBO J 9, 1603-14. Giangrande, P.H. et al. (2000) Mol Cell Biol 20, 3102-15. Wen, D.X. et al. (1994) Mol Cell Biol 14, 8356-64. Clemm, D.L. et al. (2000) Mol Endocrinol 14, 52-65. Zhang, Y. et al. (1997) Mol Endocrinol 11, 823-32. Takimoto, G.S. et al. (1996) J Biol Chem 271, 13308-16. Faivre, E.J. et al. (2008) Mol Endocrinol 22, 823-37. Blunt, R.J. et al. (1975) Pflugers Arch 355, 189-204.
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