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Nuclear Receptor Antibody Sampler Kit



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

1 Kit (8 x 20 microliters)

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
RARα (E6Z6K) Rabbit mAb	62294	20 µl	60 kDa	Rabbit IgG
RARγ1 (D3A4) XP [®] Rabbit mAb	8965	20 µl	58 kDa	Rabbit IgG
RXRα (D6H10) Rabbit mAb	3085	20 µl	53 kDa	Rabbit IgG
Glucocorticoid Receptor (D8H2) XP [®] Rabbit mAb	3660	20 µl	80, 91, 94 kDa	Rabbit IgG
Progesterone Receptor A/B (D8Q2J) XP [®] Rabbit mAb	8757	20 µl	90 (PR-A), 118 (PR-B) kDa	Rabbit IgG
Androgen Receptor (D6F11) XP [®] Rabbit mAb	5153	20 µl	110 kDa	Rabbit IgG
Estrogen Receptor α (D8H8) Rabbit mAb	8644	20 µl	66 kDa	Rabbit IgG
PPARγ (C26H12) Rabbit mAb	2435	20 µl	53, 57 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 Nuclear Receptor Antibody Sampler Kit provides an economical means to evaluate the presence and status of nuclear receptors. This kit contains enough primary antibody to perform two western blots per primary.
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	Nuclear Receptors are transcription factors responsible for sensing bioactive molecules, including steroid and thyroid hormones. They are regulated by multiple posttranslational modifications, which in turn impacts their ability to regulate the expression of specific genes involved in the control of reproduction, development, and metabolism.
	Androgen receptor (AR), a zinc finger transcription factor belonging to the nuclear receptor superfamily, is activated by phosphorylation and dimerization upon ligand binding (1). This promotes nuclear localization and binding of AR to androgen response elements in androgen target genes. AR plays a crucial role in several stages of male development and the progression of prostate cancer (2,3).
	Estrogen receptor α (ERα), a member of the steroid receptor superfamily, contains highly conserved DNA binding and ligand binding domains (4). Through its estrogen-independent and estrogen- dependent activation domains (AF-1 and AF-2, respectively), ERα regulates transcription by recruiting coactivator proteins and interacting with general transcriptional machinery (5).
	Glucocorticoid hormones control cellular proliferation, inflammation, and metabolism through their association with the glucocorticoid receptor (GR)/NR3C1, a member of the nuclear hormone receptor superfamily of transcription factors (6).
	Peroxisome proliferator-activated receptor γ (PPARγ) is a member of the ligand-activated nuclear receptor superfamily and functions as a transcriptional activator (7). PPARγ is preferentially expressed in adipocytes, as well as in vascular smooth muscle cells and macrophages (8). Besides its role in mediating adipogenesis and lipid metabolism (8), PPARγ also modulates insulin sensitivity, cell proliferation, and inflammation (9).
	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 (10,11). Both PR A and PR B are ligand activated, but differ in their relative ability to activate target gene transcription (12,13).
	Nuclear retinoic acid receptors (RARs) consist of three subtypes encoded by separate genes: α (NR1B1), β (NR1B2), and γ (NR1B3). For each subtype, there are at least two isoforms, which are generated by differential promoter usage and alternative splicing and differ only in their N-terminal regions.

	Retinoids, which are metabolites of vitamin A, serve as ligands for RARs (14). RARs function as ligand- dependent transcriptional regulators and are found to be heterodimerized with retinoid X receptors (RXRs). These transcriptionally active dimers regulate the expression of genes involved in cellular differentiation, proliferation, and apoptosis (15,16). The human retinoid X receptors are encoded by three distinct genes (<i>RXR</i> α, <i>RXR</i> β, and <i>RXR</i> γ) and bind selectively and with high affinity to the vitamin A derivative, 9- <i>cis</i> -retinoic acid. RXRs are type-II nuclear hormone receptors that are largely localized to the nuclear compartment independent of ligand binding. Nuclear RXRs form heterodimers with nuclear hormone receptor subfamily 1 proteins, including thyroid hormone receptors, retinoic acid receptors, vitamin D receptor, peroxisome proliferator-activated receptors, liver X receptors, and farnesoid X receptor (17).
Background References	 Li, J. and Al-Azzawi, F. (2009) <i>Maturitas</i> 63, 142-8. Avila, D.M. et al. (2010) <i>J Steroid Biochem Mol Biol</i> 76, 135-42. Montgomery, J.S. et al. (2001) <i>J Pathol</i> 195, 138-46. Mangelsdorf, D.J. et al. (1995) <i>Cell</i> 83, 835-9. Glass, C.K. and Rosenfeld, M.G. (2000) <i>Genes Dev</i> 14, 121-41. Yamamoto, K.R. (1985) <i>Annu Rev Genet</i> 19, 209-52. Tontonoz, P. et al. (1995) <i>Curr Opin Genet Dev</i> 5, 571-6. Rosen, E.D. et al. (1999) <i>Mol Cell</i> 4, 611-7. Murphy, G.J. and Holder, J.C. (2000) <i>Trends Pharmacol Sci</i> 21, 469-74. Evans, R.M. (1988) <i>Science</i> 240, 889-95. Kastner, P. et al. (1990) <i>EMBO J</i> 9, 1603-14. Giangrande, P.H. et al. (2000) <i>Mol Cell Biol</i> 20, 3102-15. Wen, D.X. et al. (1994) <i>Mol Cell Biol</i> 14, 8356-64. Rochette-Egly, C. and Germain, P. (2009) <i>Nucl Recept Signal</i> 7, e005. Delacroix, L. et al. (2010) <i>Mol Cell Biol</i> 30, 231-44. Eifert, C. et al. (2006) <i>Mol Reprod Dev</i> 73, 796-824. Gronemeyer, H. et al. (2004) <i>Nat Rev Drug Discov</i> 3, 950-64.
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