Store at -20C

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## Retinoic Acid and Retinoid X Receptors Antibody Sampler Kit



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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
RARα (E6Z6K) Rabbit mAb	62294	20 µl	60 kDa	Rabbit IgG
RXRα (D6H10) Rabbit mAb	3085	20 µl	53 kDa	Rabbit IgG
RXRβ Antibody	8715	20 µl	70-72 kDa	Rabbit
RARγ1 (D3A4) XP <sup>®</sup> Rabbit mAb	8965	20 µl	58 kDa	Rabbit IgG
RXRγ Antibody	5629	20 µl	55 kDa	Rabbit
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 Retinoic Acid and Retinoid X Receptors Antibody Sampler Kit provides an economical means to investigate the expression of various subtypes of retinoic acid and retinoid X receptors. The kit contains enough primary antibody to perform two western blot experiments 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 retinoic acid (RA) receptors (RARs) consist of three subtypes encoded by separate genes: $\alpha$ (NR1B1), $\beta$ (NR1B2), and $\gamma$ (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 (1). 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 (2,3). Consequently, RARs play critical roles in a variety of biological processes, including development, reproduction, immunity, and organogenesis (4-6). RAR mutations, fusion proteins, altered expression levels, or aberrant post-translational modifications result in multiple diseases due to altered RAR function and disruption of homeostasis.
	In contrast to the ubiquitously expressed RARα subtype, RARγ displays a complex tissue-specific expression pattern (7). The hematopoietic system expresses significant levels of RARγ, and a recent study identified a role for RARγ in hematopoietic stem cell maintenance (8). RARγ is the predominant subtype in human and mouse epidermis, representing 90% of the RARs in this tissue (9-11). Given the high level of RARγ expression in the skin, it has been suggested that this nuclear receptor participates in a transcriptional program that governs maintenance and differentiation of normal epidermis and skin appendages. The transcriptional activity of RARγ is under stringent control, in part, through retinoic acid-induced phosphorylation and proteasomal degradation (12).
	The human retinoid X receptors (RXRs) 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 receptor, retinoic acid receptors, vitamin D receptor, peroxisome proliferator-activated receptors, liver X receptors, and farnesoid X receptor (13). Since RXRs heterodimerize with multiple nuclear hormone receptors, they play a central role in transcriptional control of numerous hormonal signaling pathways by binding to <i>cis</i> -acting response elements in the promoter/enhancer region of target genes (14).
Background References	<ol> <li>Rochette-Egly, C. and Germain, P. (2009) <i>Nucl Recept Signal</i> 7, e005.</li> <li>Delacroix, L. et al. (2010) <i>Mol Cell Biol</i> 30, 231-44.</li> <li>Eifert, C. et al. (2006) <i>Mol Reprod Dev</i> 73, 796-824.</li> <li>Mark, M. et al. (2006) <i>Annu Rev Pharmacol Toxicol</i> 46, 451-80.</li> <li>Niederreither, K. and Dollé, P. (2008) <i>Nat Rev Genet</i> 9, 541-53.</li> <li>Mark, M. et al. (2009) <i>Nucl Recept Signal</i> 7, e002.</li> <li>Dollé, P. (2009) <i>Nucl Recept Signal</i> 7, e006.</li> </ol>

	8. Purton, L.E. et al. (2006) <i>J Exp Med</i> 203, 1283-93. 9. Fisher, G.J. et al. (1994) <i>J Biol Chem</i> 269, 20629-35. 10. Zelent, A. et al. (1989) <i>Nature</i> 339, 714-7. 11. Elder, J.T. et al. (1991) <i>J Invest Dermatol</i> 96, 425-33. 12. Giannì, M. et al. (2002) <i>EMBO J</i> 21, 3760-9. 13. Gronemeyer, H. et al. (2004) <i>Nat Rev Drug Discov</i> 3, 950-64. 14. Mangelsdorf, D.J. et al. (1992) <i>Genes Dev</i> 6, 329-44.
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