



## MONOCLONAL ANTI-RETINOID X RECEPTOR $\beta$ CLONE 147

Purified Mouse Immunoglobulin

Product Number **R 2777**

### Product Description

Monoclonal Anti-Retinoid X Receptor  $\beta$  (RXR $\beta$ ) (mouse IgG1 isotype) is derived from the hybridoma produced by the fusion of mouse myeloma p3-NS-1/Ag4.1 cells with splenocytes from BALB/c mice immunized with a synthetic peptide derived from the hinge of the mouse RXR $\beta$ . The antibody is purified by protein A chromatography.

Monoclonal Anti-Retinoid X Receptor  $\beta$  recognizes mouse retinoid X receptor  $\beta$  (~60 kDa). It does not react with mouse RXR $\alpha$  or RXR $\gamma$  isotypes, or with mouse retinoic acid receptors (RAR). It has been used in immunoblotting.

Retinoids are metabolites of vitamin A and play important roles as signaling molecules in vertebrate development and differentiation. Two nuclear receptor families are involved in retinoid signaling: the retinoic acid receptor family (RARs), which includes RAR $\alpha$ , RAR $\beta$ , and RAR $\gamma$  and the retinoid X receptors (RXRs), which includes RXR $\alpha$ , RXR $\beta$ , and RXR $\gamma$ . Members of the RAR family are retinoic acid-inducible enhancer factors that have high affinity for all-trans retinoic acids. They belong to the superfamily of steroid/thyroid nuclear receptors. The RAR $\alpha$  and RAR $\beta$  genes are more homologous to the two related thyroid hormone receptors THRA and THRB, than to any other member of the nuclear receptor family, indicating that the thyroid hormone and retinoic acid receptors evolved from a common ancestor. The ligand binding domains of the RARs are highly conserved and RAR isoforms are expressed in distinct patterns throughout developing and mature organisms. The RXR family members are closely related to each other in their DNA- and ligand-binding domains but are very divergent from the retinoic acid receptor (RAR) subfamily in both structure and ligand specificity. RXRs are activated by 9-cis retinoic acid, a stereo and photoisomer of all-trans-RA.<sup>1-3</sup> Retinoid X receptors act as cellular coregulators that form heterodimers by binding to the receptors for retinoic acid (RAR), thyroid hormone (TR), vitamin D3 (VDR), or peroxisome proliferators (PPAR). These heterodimers then bind to their cognate DNA response elements and regulate gene expression.<sup>4-6</sup>

## Product Information

In mouse, alternative splicing of the gene for RXR $\beta$  results in two isoforms. It has been found in all mouse tissue tested including brain, thymus, spleen and liver.<sup>7</sup>

### Reagent

Monoclonal Anti-Retinoid X Receptor  $\beta$  is supplied as a solution in phosphate buffered saline, pH 7.4, with 0.08% sodium azide as a preservative.

### Precautions and Disclaimer

Due to the sodium azide content, a material safety data sheet (MSDS) for this product has been sent to the attention of the safety officer of your institution. Consult the MSDS for information regarding hazards and safe handling practices.

### Storage/Stability

Store at  $-20^{\circ}\text{C}$ . Upon initial thawing freeze the solution in working aliquots for extended storage. Avoid repeated freezing and thawing to prevent denaturing the antibody. Do not store in a frost-free freezer. The antibody is stable for at least 12 months when stored appropriately. Working dilutions should be discarded if not used within 12 hours.

### Product Profile

A recommended working concentration for immunoblotting is 1  $\mu\text{g}/\text{ml}$ . The immunoblotting was performed with NIH/3T3 cells.

Note: In order to obtain best results using different techniques and preparations we recommend determining optimal working concentration by titration.

### References

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4. Zhang, X.K., and Pfahl, M., Hetero- and homodimeric receptors in thyroid hormone and vitamin A action. *Receptor*, **3**, 183-191 (1993).

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7. Nagata T., et al., The mouse Rxrb gene encoding RXR  $\beta$ : genomic organization and two mRNA isoforms generated by alternative splicing of transcripts initiated from CpG island promoters. *Gene*, **142**, 183-189 (1994).

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