

## Product Information

### Anti-LXR $\beta$ (C-terminal)

produced in rabbit, IgG fraction of antiserum

Product Number **L5169**

### Product Description

Anti-LXR $\beta$  (C-terminal) is produced in rabbit using as the immunogen a synthetic peptide corresponding to a sequence at the C-terminal of human LXR $\beta$  (GenelD 7376), conjugated to KLH. The corresponding sequence is highly conserved (single amino acid substitution) in rat and mouse LXR $\beta$ . Whole antiserum is purified using protein A immobilized on agarose to provide the IgG fraction of antiserum.

Anti-LXR $\beta$  (C-terminal) specifically recognizes human LXR $\beta$ . The antibody can be used for immunoblotting (~45 kDa). Detection of the LXR $\beta$  band by immunoblotting is specifically inhibited by the LXR $\beta$  immunizing peptide.

The liver X receptors (LXRs) are oxysterol-activated nuclear receptors that play an important role in the control cholesterol homeostasis.<sup>1</sup> Two different genes have been described, LXR $\alpha$  (NR1H3) and LXR $\beta$  (NR1H2). LXR $\alpha$  expression is restricted to macrophages and tissues involved in lipid metabolism, whereas LXR $\beta$  is more ubiquitous. LXRs heterodimerize with the retinoid X receptor (RXR) and bind to the LXR response element (LXRE). LXRs regulate cholesterol homeostasis by modulating the transcription of genes involved in its catabolism, storage, absorption and transport.<sup>1</sup> Activated LXRs are also potent inhibitors of inflammatory responses in macrophages, and reduce inflammation *in vivo*.<sup>2</sup> LXR expression and activation with LXRs ligands have also been shown to modulate atherosclerotic lesions.<sup>3</sup> LXR $\alpha/\beta$ -deficient mice show enhanced lipid-loaded foam cell accumulation. LXRs have been suggested to play an important role in Alzheimer's Disease (AD) pathogenesis.<sup>4,5</sup> The initiation and progression of AD has been linked to cholesterol metabolism and inflammation, processes that can be modulated by LXRs. Genetic knockout of either LXR $\alpha$  or LXR $\beta$  in APP/PS1 transgenic mice results in increased amyloid plaque load. Ligand activation of LXRs has been shown to attenuate the inflammatory response of primary mixed glial cultures to fibrillar amyloid  $\beta$  peptide (fA $\beta$ ).

### Reagent

Supplied as a solution in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide as a preservative.

### Precautions and Disclaimer

For R&D use only. Not for drug, household, or other uses. Please consult the Safety Data Sheet for information regarding hazards and safe handling practices.

### Storage/Stability

For continuous use, store at 2–8 °C for up to one month. For extended storage, freeze in working aliquots. Repeated freezing and thawing, or storage in "frost-free" freezers, is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilutions should be discarded if not used within 12 hours.

### Product Profile

**Immunoblotting:** a working dilution of 1:1,000-1:2,000 is recommended using COS7 cells expressing human LXR $\beta$ .

**Note:** In order to obtain best results in various techniques and preparations, it is recommended to determine optimal working dilutions by titration.

### References

1. Zelcer, N., and Tontonoz, P., *J. Clin. Investig.*, **116**, 607-614 (2006).
2. Joseph, S. et al., *Nat. Med.*, **9**, 213-219 (2003).
3. Tangirala, R.K. et al., *Proc. Natl. Acad. Sci. USA*, **99**, 11896-11901 (2002).
4. Zelcer, N. et al., *Proc. Natl. Acad. Sci. USA*, **104**, 10601-10606 (2007).
5. Sun, Y. et al., *J. Biol. Chem.*, **278**, 27688–27694 (2003).

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