DB134: PPARγ (C15)

Background:

Peroxisome proliferator-activated receptors (PPAR) are members of the steroid nuclear receptor superfamily (1). The mammalian PPARs include three subtypes PPARα, PPARβ, and PPARγ. PPARs have been found to regulate fatty acid oxidation, fat cell development, lipoprotein metabolism, and glucose homeostasis (2). Recent studies have found that PPARs can affect the pathogenesis and development of tumors (3). PPARγ and PPARβ have been specifically implicated in tumorigenesis (3). PPARα is the focus of research studying its function in fatty acid beta-oxidation, lipid metabolism, and vascular inflammation (4). PPARβ also plays a key role in the activation of keratinocytes during the inflammatory reaction associated with a skin injury (5). Of the three subtypes of PPARs, PPARγ is the most widely studied. PPARγ has been implicated in various human chronic diseases such as diabetes mellitus, atherosclerosis, rheumatoid arthritis, inflammatory bowel disease, and Alzheimer's disease (6).

Origin:

PPARγ (C15) is provided as an affinity purified rabbit polyclonal antibody, raised against a peptide mapping near the carboxy terminus of human PPARγ.

Product Details:

Each vial contains 200 µg/ml of affinity purified rabbit IgG, PPARγ (C15) DB134, in 1 ml PBS containing 0.1 % sodium azide and 0.2% gelatin.

Competition Studies:

A blocking peptide is also available, DB134P, for use in competition studies. Each vial contains 100 µg of peptide in 0.5 ml PBS with 0.1% sodium azide and 100 µg BSA.

Specificity:

PPARγ (C15) DB134 will recognize mouse, rat, human, hamster, chicken, dog, bovine, and pig PPARγ1 and PPARγ2 by western blotting. Recommended western blotting starting dilution 1:400.
Immunohistochemistry: not yet tested
Immunoprecipitation: not yet tested

Storage:

Store this product at 4º C, do not freeze. The product is stable for one year from the date of shipment.

References:

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2. Li AC, and Glass CK. 2004. PPAR and LXR-dependent pathways controlling lipid metabolism and development of atherosclerosis. J Lipid Res.
3. Nahle Z. 2004. PPAR trilogy from metabolism to cancer. Curr Opin Clin Nutr Metab Care. 7(4):397-402.
4. van Raalte DH, Li M, Pritchard PH, Wasan KM. 2004. Peroxisome proliferator-activated receptor (PPAR)-alpha: a pharmacological target with a promising future. Pharm Res. 21(9): 1531-1538.
5. Tan NS, Michalik L, Desvergne B, Wahli W. 2003. Peroxisome proliferator-activated receptor (PPAR)-beta as a target for wound healing drugs: what is possible? Am J Clin Dermatol. 4(8): 523-530.
6. Takano H, Hasegawa H, Zou Y, Komuro I. 2004. Pleiotropic actions of PPAR gamma activators thiazolidinediones in cardiovascular diseases. Curr Pharm Des. 10(22): 2779-2786.

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