Product Information Sheet

Polyclonal Anti- Peroxisome Proliferator-activated Receptor Gamma, *PPAR-G (Magnetic Bead Conjugate)*

Catalogue No. PA1320-M
Lot No. 09J01
Ig type rabbit IgG
Size 100µg/vial
Specificity
Human.
No cross reactivity with other proteins.

Recommended application
ImmunoPrecipitation (IP)

Immunogen
A synthetic peptide corresponding to a sequence at the N-terminal of human PPAR-G, different from the rat and mouse sequence by two amino acids.

Purity
Immunogen affinity purified.

Contents
Each vial contains 1mg/ml Magnetic Bead in PBS, pH 7.2, 0.05mg NaN_3.

Storage
Store at 4˚C for frequent use.

Description
This Antagene antibody is immobilized by the covalent reaction of hydrazinonicotinamide-modified antibody with formylbenzamide-modified magnetic beads. It is useful for immunoprecipitation

BACKGROUND

The peroxisome proliferator-activated receptors (PPARs) are a group of three nuclear receptor isoforms, PPAR gamma, PPAR alpha, and PPAR delta, encoded by different genes. PPARs are ligand-regulated transcription factors that control gene expression by binding to specific response elements (PPREs) within promoters.[1] PPAR gamma is a transcription factor that has a pivotal role in adipocyte differentiation and expression of adipocyte-specific genes. The PPAR gamma1 and gamma2 isoforms result from alternative splicing and have ligand-dependent and -independent activation domains.[2] PPAR gamma is a member of a family of nuclear receptors/ligand-dependent transcription factors, which bind to hormone response elements on target gene promoters. Ameshima et al. (2003) found that PPAR gamma is abundantly expressed in normal lung tissues, especially in endothelial cells, but that its expression is reduced or absent in the angiogenic plexiform lesions of pulmonary hypertensive lungs and in the vascular lesions of a rat model of severe pulmonary hypertension. And they conclude that fluid shear stress decreases the expression of PPARgamma in endothelial cells and that loss of PPARgamma expression characterizes an abnormal, proliferating, apoptosis-resistant endothelial cell phenotype.[3]

REFERENCE

1. Berger J, Moller DE (2002). "The mechanisms of action of PPARs". Annu. Rev. Med. 53: 409–35.
2. Deeb, S. S.; Fajas, L.; Nemoto, M.; Pihlajamaki, J.; Mykkanen, L.; Kuusisto, J.; Laakso, M.; Fujimoto, W.; Auwerx, J. : A pro12ala substitution in PPAR-gamma-2 associated with decreased receptor activity, lower body mass index and improved insulin sensitivity. Nature Genet. 20: 284-287, 1998.
3. Ameshima, S.; Golpon, H.; Cool, C. D.; Chan, D.; Vandivier, R. W.; Gardai, S. J.; Wick, M.; Nemenoff, R. A.; Geraci, M. W.; Voelkel, N. F. : Peroxisome proliferator-activated receptor gamma (PPAR-gamma) expression is decreased in pulmonary hypertension and affects endothelial cell growth. Circ. Res. 92: 1162-1169, 2003.

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