



Product Information Sheet

Polyclonal Anti- Peroxisome Proliferator-activated Receptor Gamma, **PPAR-G**

Catalogue No. PA1320

Lot No. 09J01

Ig type rabbit IgG

Size 100µg/vial

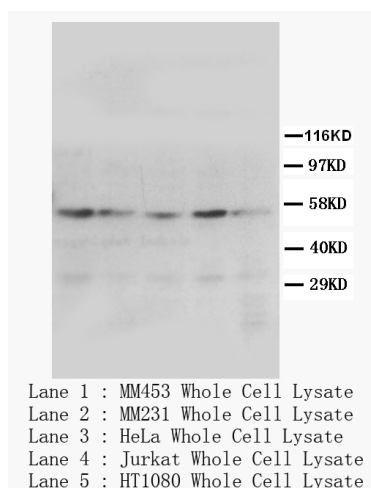
Specificity

Human.

No cross reactivity with other proteins.

Recommended application

Western blot



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.

Application

	Concentration	Tested Species	Concluded Species	Antigen Retrieval
WB	1µg/ml	Hu	-	-
IHC-P	-	-	-	-
IHC-F	-	-	-	-
ICC	-	-	-	-

Other applications have not been tested.

Optimal dilutions should be determined by end user.

Contents

Each vial contains 5mg BSA, 0.9mg NaCl, 0.2mg Na₂HPO₄, 0.05mg Thimerosal, 0.05mg NaN₃.

Reconstitution

0.2ml of distilled water will yield a concentration of 500µg/ml.

Storage

At -20°C for one year. After reconstitution, at 4°C for one month. It can also be aliquotted and stored frozen at -20°C for longer time.

To reorder contact us at:

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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.