bs-3614R **[Primary Antibody]**

PPAR alpha Rabbit pAb

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DATASHEET

Host: Rabbit **Isotype:** IgG

Clonality: Polyclonal

GeneID: 5465 **SWISS:** Q07869

Target: PPAR alpha

Immunogen: KLH conjugated synthetic peptide derived from human PPAR alpha: 301-400/468.

Purification: affinity purified by Protein A

Concentration: 1mg/ml

Storage: 0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
Shipped at 4°C. Store at -20°C for one year. Avoid repeated freeze/thaw cycles.

Background: Peroxisome proliferators are nongenotoxic carcinogens which are purported to exert their effect on cells through their interaction with members of the nuclear hormone receptor family, termed Peroxisome Proliferator Activated Receptors (PPARs). Nuclear hormone receptors are ligand dependent intracellular proteins that stimulate transcription of specific genes by binding to specific DNA sequences following activation by the appropriate ligand. Studies indicate that PPARs are activated by peroxisome proliferators such as clofibric acid, nafenopin, and WY-14,643, as well as by some fatty acids. It has also been shown that PPARs can induce transcription of acyl coenzyme A oxidase and cytochrome P450 A6 (CYP450 A6) through interaction with specific response elements. PPAR alpha is activated by free fatty acids including linoleic, arachidonic, and oleic acids. Induction of peroxisomes by this mechanism leads to a reduction in blood triglyceride levels. PPAR alpha is expressed mainly in skeletal muscle, heart, liver, and kidney and is thought to regulate many genes involved in the beta-oxidation of fatty acids. Activation of rat liver PPAR alpha has been shown to suppress hepatocyte apoptosis. PPAR alpha, like several other nuclear hormone receptors, heterodimerizes with retinoic X receptor (RXR) alpha to form a transcriptionally competent complex.

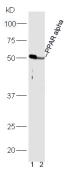
Applications: **WB** (1:500-2000)
IHC-P (1:100-500)
IHC-F (1:100-500)
IF (1:100-500)
Flow-Cyt (1μg /test)

Reactivity: Human, Mouse, Rat (predicted: Rabbit, Pig, Cow, Chicken, Horse)

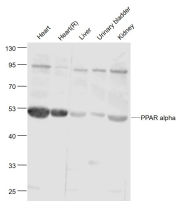
Predicted MW.: 51 kDa

Subcellular Location: Nucleus

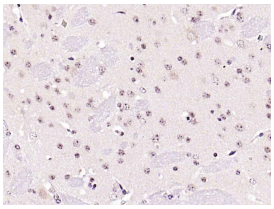
VALIDATION IMAGES



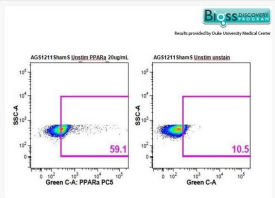
Sample: Lane1: Heart(Mouse) Lysate at 30 ug Lane2: Liver(Mouse) Cell Lysate at 30 ug Primary: Anti-PPAR alpha (bs-3614R) at 1:300 dilution; Secondary: HRP conjugated Goat-Anti-rabbit IgG(bs-0295G-HRP) at 1: 5000dilution; Predicted band size: 51 kD Observed band size: 51 kD



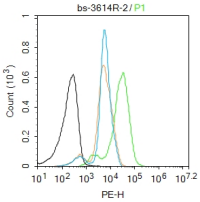
Sample: Heart (Mouse) Lysate at 40 ug Heart (Rat) Lysate at 40 ug Liver (Mouse) Lysate at 40 ug Urinary bladder (Mouse) Lysate at 40 ug Kidney (Mouse) Lysate at 40 ug Primary: Anti-PPAR alpha (bs-3614R) at 1/1000 dilution Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution Predicted band size: 52/19 kD Observed band size: 52 kD



Paraformaldehyde-fixed, paraffin embedded (rat brain); Antigen retrieval by boiling in sodium citrate buffer (pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 20 minutes; Blocking buffer (normal goat serum) at 37°C for 30min; Antibody incubation with (PPAR alpha) Polyclonal Antibody, Unconjugated (bs-3614R) at 1:200 overnight at 4°C, followed by operating according to SP Kit(Rabbit) (sp-0023) instructionsand DAB staining.



Rat splenocytes stained with Anti-PPAR alpha Polyclonal Antibody, PE-CY5 Conjugated (bs-3614R-PE-Cy5) at 1:50.



Blank control: Jurkat. Primary Antibody (green line): Rabbit Anti-PPAR alpha antibody (bs-3614R) Dilution: 1µg /10^6 cells; Isotype Control Antibody (orange line): Rabbit IgG . Secondary Antibody : Goat anti-rabbit IgG-FITC Dilution: 1µg /test. Protocol The cells were fixed with 4% PFA (10min at room temperature)and then permeabilized with 90% ice-cold methanol for 20 min at-20°C.The cells were then incubated in 5%BSA to block non-specific protein-protein interactions for 30 min at room temperature .Cells stained with Primary Antibody for 30 min at room temperature. The secondary antibody used for 40 min at room temperature. Acquisition of 20,000 events was performed.

SELECTED CITATIONS

- **[IF=15.304]** Lili Chang. et al. Regulating T-cell metabolic reprogramming and blocking PD-1 co-promote personalized postoperative autologous nanovaccines. BIOMATERIALS. 2023 Jun;297:122104 FCM ;Mouse. 37058898
- **[IF=8.1]** Zou Dongmei. et al. Impaired SUMOylation of FoxA1 promotes nonalcoholic fatty liver disease through down-regulation of Sirt6. CELL DEATH DIS. 2024 Sep;15(9):1-14 IHC,WB ;Mouse. 39277582
- **[IF=8.3]** Yuwei Bai. et al. Mult-omics Analysis Reveals the Lipid-lowering Effects of Sea Buckthorn and Milk Thistle Solid Beverage in Hyperlipidemic Rats. PHYTOMEDICINE. 2025 May;:156920 WB ;Rat. 40472616
- **[IF=8.4]** Wang Zhen-chuan. et al. Targeting PPARα activation sensitizes glioblastoma cells to temozolomide and reverses acquired resistance by inhibiting H3K18 lactylation. ACTA PHARMACOL SIN. 2025 Jun;:1-16 IF,IHC,WB ;Mouse,Human. 40500345
- **[IF=6.7]** Yan Zhang. et al. Pterosin B improves cognitive dysfunction by promoting microglia M1/M2 polarization through inhibiting Klf5/Parp14 pathway. PHYTOMEDICINE. 2024 Oct;:156152 WB ;Mouse. 39413455