

NIACR1 Antibody (Center)

Affinity Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP12667C

Specification

NIACR1 Antibody (Center) - Product Information

Application WB,E
Primary Accession Q8TDS4

Other Accession <u>P49019</u>, <u>Q9EP66</u>, <u>NP_808219.1</u>

Reactivity
Predicted
Host
Clonality
Isotype
Antigen Region

Human
Mouse
Rabbit
Polyclonal
Rabbit IgG
200-228

NIACR1 Antibody (Center) - Additional Information

Gene ID 338442

Other Names

Hydroxycarboxylic acid receptor 2, G-protein coupled receptor 109A, G-protein coupled receptor HM74A, Niacin receptor 1, Nicotinic acid receptor, HCAR2, GPR109A, HCA2, HM74A, NIACR1

Target/Specificity

This NIACR1 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 200-228 amino acids of human NIACR1.

Dilution

WB~~1:1000

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

NIACR1 Antibody (Center) is for research use only and not for use in diagnostic or therapeutic procedures.

NIACR1 Antibody (Center) - Protein Information

Name HCAR2

Synonyms GPR109A, HCA2, HM74A, NIACR1



Function Acts as a high affinity receptor for both nicotinic acid (also known as niacin) and (D)-beta-hydroxybutyrate and mediates increased adiponectin secretion and decreased lipolysis through G(i)- protein-mediated inhibition of adenylyl cyclase. This pharmacological effect requires nicotinic acid doses that are much higher than those provided by a normal diet. Mediates nicotinic acid-induced apoptosis in mature neutrophils. Receptor activation by nicotinic acid results in reduced cAMP levels which may affect activity of cAMP-dependent protein kinase A and phosphorylation of target proteins, leading to neutrophil apoptosis. The rank order of potency for the displacement of nicotinic acid binding is 5-methyl pyrazole-3-carboxylic acid = pyridine-3-acetic acid > acifran > 5-methyl nicotinic acid = acipimox >> nicotinuric acid = nicotinamide.

Cellular Location

Cell membrane; Multi-pass membrane protein

Tissue Location

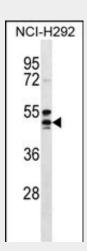
Expression largely restricted to adipose tissue and spleen. Expressed on mature neutrophils but not on immature neutrophils or eosinophils.

NIACR1 Antibody (Center) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- <u>Immunohistochemistry</u>
- <u>Immunofluorescence</u>
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

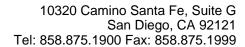
NIACR1 Antibody (Center) - Images



NIACR1 Antibody (Center) (Cat. #AP12667c) western blot analysis in NCI-H292 cell line lysates (35ug/lane). This demonstrates the NIACR1 antibody detected the NIACR1 protein (arrow).

NIACR1 Antibody (Center) - Background

NIACR1 acts as a high affinity receptor for both nicotinic acid (also known as niacin) and (D)-beta-hydroxybutyrate and mediates increased adiponectin secretion and decreased lipolysis





through G(i)-protein-mediated inhibition of adenylyl cyclase. This pharmacological effect requires nicotinic acid doses that are much higher than those provided by a normal diet. Mediates nicotinic acid-induced apoptosis in mature neutrophils. Receptor activation by nicotinic acid results in reduced cAMP levels which may affect activity of cAMP-dependent protein kinase A and phosphorylation of target proteins, leading to neutrophil apoptosis. The rank order of potency for the displacement of nicotinic acid binding is 5-methyl pyrazole-3-carboxylic acid = pyridine-3-acetic acid > acifran > 5-methyl nicotinic acid = acipimox >> nicotinuric acid = nicotinamide.

NIACR1 Antibody (Center) - References

Li, X., et al. Biochem. Pharmacol. 80(9):1450-1457(2010) Bailey, S.D., et al. Diabetes Care 33(10):2250-2253(2010) Li, G., et al. J. Biol. Chem. 285(29):22605-22618(2010) Mandrika, I., et al. Biochem. Biophys. Res. Commun. 395(2):281-287(2010) Shen, H.C., et al. J. Med. Chem. 53(6):2666-2670(2010)