SANTA CRUZ BIOTECHNOLOGY, INC.

DGAT1 (G-20): sc-26173

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Glucose and Insulin are anabolic signals which upregulate the transcriptions of a series of lipogenic enzymes to convert excess carbohydrate into triglycerides for efficient energy storage. Acyl-Coenzyme A:diacylglycerol acyltransferase, also known as DGAT1 and ARGP1, is a microsomal enzyme that assists in the synthesis of fatty acids into triglycerides. DGAT catalyzes the terminal and only committed step in triacylglycerol synthesis by using diacylglycerol (DAG) and fatty acyl CoA as substrates. DGAT plays a fundamental role in the metabolism of cellular diacylglycerol and is important in higher eukaryotes for physiologic processes involving triacylglycerol metabolism such as intestinal fat absorption, lipoprotein assembly, adipose tissue formation, and lactation. DGAT1 is involved in fat absorption in the intestine and in basal level triglyceride synthesis in adipose tissue, where it is more highly expressed. Mice lacking DGAT1 have increased energy expenditure and therefore are resistant to obesity. In addition, mice lacking both copies of DGAT1 are completely devoid of milk secretion, most likely because of deficient triglyceride synthesis in the mammary gland.

CHROMOSOMAL LOCATION

Genetic locus: Dgat1 (mouse) mapping to 15 D3.

SOURCE

DGAT1 (G-20) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the N-terminus of DGAT1 of mouse origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-26173 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

APPLICATIONS

DGAT1 (G-20) is recommended for detection of DGAT1 of mouse and rat origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for DGAT1 siRNA (m): sc-40488, DGAT1 siRNA (r): sc-72066, DGAT1 shRNA Plasmid (m): sc-40488-SH, DGAT1 shRNA Plasmid (r): sc-72066-SH, DGAT1 shRNA (m) Lentiviral Particles: sc-40488-V and DGAT1 shRNA (r) Lentiviral Particles: sc-72066-V.

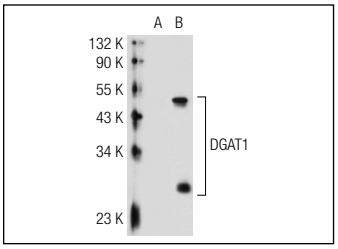
Molecular Weight of DGAT1: 55 kDa.

Positive Controls: DGAT1 (m): 293T Lysate: sc-119756.

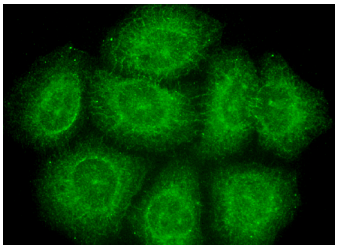
RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Western Blotting: use donkey anti-goat IgG-HRP: sc-2020 (dilution range: 1:2000-1:100,000) or Cruz Marker™ compatible donkey anti-goat IgG-HRP: sc-2033 (dilution range: 1:2000-1:5000), Cruz Marker™ Molecular Weight Standards: sc-2035, TBS Blotto A Blocking Reagent: sc-2333 and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml). 3) Immunofluorescence: use donkey anti-goat IgG-FITC: sc-2024 (dilution range: 1:100-1:400) or donkey anti-goat IgG-TR: sc-2783 (dilution range: 1:100-1:400) with UltraCruz™ Mounting Medium: sc-24941.

DATA



DGAT1 (G-20): sc-26173. Western blot analysis of DGAT1 expression in non-transfected: sc-117752 (**A**) and mouse DGAT1 transfected: sc-119756 (**B**) 293T whole cell lysates.



DGAT1 (G-20): sc-26173. Immunofluorescence staining of methanol-fixed HeLa cells showing membrane and cytoplasmic localization.

SELECT PRODUCT CITATIONS

1. Petridou, A., et al. 2007. Long-term exercise increases the DNA binding activity of peroxisome proliferator-activated receptor γ in rat adipose tissue. Metab. Clin. Exp. 56: 1029-1036.
2. Yang, Z., et al. 2009. Evidence for an effect of clozapine on the regulation of fat-cell derived factors. Clin. Chim. Acta 408: 98-104.
3. Chen, Y., et al. 2015. Inhibition of ERK1/2 and activation of LXR synergistically reduce atherosclerotic lesions in ApoE-deficient mice. Arterioscler. Thromb. Vasc. Biol. 35: 948-959.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.