SANTA CRUZ BIOTECHNOLOGY, INC.

AdipoR2 (N-16): sc-46755



BACKGROUND

Adiponectin is a circulating hormone secreted by adipocytes that improves the metabolism of glucose and lipids, and is expressed at low levels in those with obesity and diabetes. Adiponectin receptors AdipoR1 and AdipoR2, also designated progestin and AdipoQ receptor family members I and II, respectively, regulate fatty acid oxidation and the uptake of glucose by adiponectin. Each receptor activates a unique set of signaling molecules including AMPK, p38 MAPK and PPAR α . AdipoR1 has a high affinity for globular adiponectin and low-affinity for full-length adiponectin, while AdipoR2 has an intermediate affinity for both forms. AdipoR1 and AdipoR2 are mainly expressed in liver and muscle. Adiponectin, AdipoR1 and AdipoR2 are all associated with body composition, Insulin sensitivity and metabolic parameters. Physical training increases circulating adiponectin and mRNA expression of AdipoR1 and AdipoR2 in muscle, which may mediate the improvement of Insulin resistance and the metabolic syndrome in response to exercise.

REFERENCES

- Kadowaki, T. and Yamauchi, T. 2005. Adiponectin and adiponectin receptors. Endocr. Rev. 26: 439-451.
- Bluher, M., et al. 2005. Regulation of adiponectin receptor R1 and R2 gene expression in adipocytes of C57BL/6 mice. Biochem. Biophys. Res. Commun. 329: 1127-1132.
- Nilsson, L., et al. 2005. Prolactin and growth hormone regulate adiponectin secretion and receptor expression in adipose tissue. Biochem. Biophys. Res. Commun. 331: 1120-1126.
- Kaltenbach, S., et al. 2005. Adiponectin receptor gene expression in human skeletal muscle cells is not regulated by fibrates and thiazolidinediones. Int. J. Obes. Relat. Metab. Disord. 29: 760-765.
- Chen, M., et al. 2005. Impaired activation of AMP-kinase and fatty acid oxidation by globular adiponectin in cultured human skeletal muscle of obese type 2 diabetics. J. Clin. Endocrinol. Metab. 90: 3665-3672.

CHROMOSOMAL LOCATION

Genetic locus: Adipor2 (mouse) mapping to 6 F1.

SOURCE

AdipoR2 (N-16) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the N-terminus of AdipoR2 of mouse origin.

PRODUCT

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

Blocking peptide available for competition studies, sc-46755 P, (100 μ g peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

STORAGE

Store at 4° C, **D0 NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

APPLICATIONS

AdipoR2 (N-16) is recommended for detection of AdipoR2 of mouse and rat origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 μ g per 100-500 μ g 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 AdipoR2 siRNA (m): sc-60126, AdipoR2 siRNA (r): sc-156025, AdipoR2 shRNA Plasmid (m): sc-60126-SH, AdipoR2 shRNA Plasmid (r): sc-156025-SH, AdipoR2 shRNA (m) Lentiviral Particles: sc-60126-V and AdipoR2 shRNA (r) Lentiviral Particles: sc-156025-V.

Molecular Weight of AdipoR2: 44 kDa.

Positive Controls: mouse liver extract: sc-2256, rat liver extract: sc-2395 or F9 cell lysate: sc-2245.

DATA





AdipoR2 (N-16): sc-46755. Western blot analysis of AdipoR2 expression in mouse liver (A) and rat liver (B) tissue extracts.

AdipoR2 (N-16): sc-46755. Western blot analysis of AdipoR2 expression in F9 whole cell lysate (\pmb{A}) and mouse liver tissue extract (\pmb{B}).

SELECT PRODUCT CITATIONS

- Coope, A., et al. 2008. AdipoR1 mediates the anorexigenic and Insulin/ leptin-like actions of adiponectin in the hypothalamus. FEBS Lett. 582: 1471-1476.
- Massip-Salcedo, M., et al. 2008. Activation of peroxisome proliferatoractivated receptor-α inhibits the injurious effects of adiponectin in rat steatotic liver undergoing ischemia-reperfusion. Hepatology 47: 461-472.
- Du, R.H., et al. 2014. Fumigaclavine C activates PPARγ pathway and attenuates atherogenesis in ApoE-deficient mice. Atherosclerosis 234: 120-128.

RESEARCH USE

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

