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LEPR Antibody (C-term) Blocking Peptide

Synthetic peptide
Catalog # BP17169b

Specification

LEPR Antibody (C-term) Blocking Peptide - Product Information

Primary Accession P48357

LEPR Antibody (C-term) Blocking Peptide - Additional Information

Gene ID 3953

Other Names
Leptin receptor, LEP-R, HuB219, OB receptor, OB-R, CD295, LEPR, DB, OBR

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

LEPR Antibody (C-term) Blocking Peptide - Protein Information

Name LEPR

Synonyms DB, OBR

Function
Receptor for hormone LEP/leptin (Probable) (PubMed:22405007). On ligand binding, mediates LEP central and peripheral effects through the activation of different signaling pathways such as JAK2/STAT3 and MAPK cascade/FOS. In the hypothalamus, LEP acts as an appetite- regulating factor that induces a decrease in food intake and an increase in energy consumption by inducing anorexinogenic factors and suppressing orexigenic neuropeptides, also regulates bone mass and secretion of hypothalamo-pituitary-adrenal hormones (By similarity) (PubMed:9537324). In the periphery, increases basal metabolism, influences reproductive function, regulates pancreatic beta-cell function and insulin secretion, is pro-angiogenic and affects innate and adaptive immunity (PubMed:25060689, PubMed:12504075, PubMed:8805376). Control of energy homeostasis and melanocortin production (stimulation of POMC and full repression of AgRP transcription) is mediated by STAT3 signaling, whereas distinct signals regulate NPY and the control of fertility, growth and glucose homeostasis.

Involved in the regulation of counter-regulatory response to hypoglycemia by inhibiting neurons of the parabrachial nucleus. Has a specific effect on T lymphocyte responses, differentially regulating the proliferation of naive and memory T -ells. Leptin increases Th1 and suppresses Th2 cytokine production (By similarity).

Cellular Location
Cell membrane; Single-pass type I membrane protein. Basolateral cell membrane

Tissue Location
Isoform A is expressed in fetal liver and in hematopoietic tissues and choroid plexus. In adults highest expression in heart, liver, small intestine, prostate and ovary. Low level in lung and kidney. Isoform B is highly expressed in hypothalamus, but also in skeletal muscle. Detected in fundic and antral epithelial cells of the gastric mucosa (PubMed:19159218). Isoform B and isoform A are expressed by NK cells (at protein level) (PubMed:12504075)

LEPR Antibody (C-term) Blocking Peptide - Protocols

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

- Blocking Peptides

LEPR Antibody (C-term) Blocking Peptide - Images

LEPR Antibody (C-term) Blocking Peptide - Background

The protein encoded by this gene belongs to the gp130family of cytokine receptors that are known to stimulate genetranscription via activation of cytosolic STAT proteins. Thisprotein is a receptor for leptin (an adipocyte-specific hormonethat regulates body weight), and is involved in the regulation offat metabolism, as well as in a novel hematopoietic pathway that isrequired for normal lymphopoiesis. Mutations in this gene have beenassociated with obesity and pituitary dysfunction. Alternativelyspliced transcript variants encoding different isoforms have beendescribed for this gene. It is noteworthy that this gene and LEPROTgene (GeneID:54741) share the same promoter and the first 2 exons,however, encode distinct proteins (PMID:9207021).

LEPR Antibody (C-term) Blocking Peptide - References

Hu, M., et al. Pharmacogenet. Genomics 20(10):634-637(2010)Bailey, S.D., et al. Diabetes Care 33(10):2250-2253(2010)de Luis, D.A., et al. Ann. Nutr. Metab. 57(2):89-94(2010)Louis, G.W., et al. J. Neurosci. 30(34):11278-11287(2010)Sarzynski, M.A., et al. Int J Obes (Lond) (2010) In press :