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Leptin Receptor (LEPR) Antibody (N-term) Blocking peptide

Synthetic peptide
Catalog # BP6151a

Specification

Leptin Receptor (LEPR) Antibody (N-term) Blocking peptide - Product Information

Primary Accession	P48357
Other Accession	NP_002294

Leptin Receptor (LEPR) Antibody (N-term) Blocking peptide - Additional Information

Gene ID 3953

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

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP6151a was selected from the N-term region of human LEPR . A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

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.

Leptin Receptor (LEPR) Antibody (N-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)

Leptin Receptor (LEPR) Antibody (N-term) Blocking peptide - Protocols

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

- Blocking Peptides

Leptin Receptor (LEPR) Antibody (N-term) Blocking peptide - Images

Leptin Receptor (LEPR) Antibody (N-term) Blocking peptide - Background

Leptin, an adipocyte-specific hormone, regulates adipose-tissue mass through hypothalamic effects on satiety and energy expenditure by acting through the leptin receptor (LEPR). LEPR is a single-transmembrane-domain receptor of the cytokine receptor family that is identical to the mouse diabetes (db) gene product. During weight loss, leptin levels decrease, whereas soluble LEPR levels and the receptor bound fraction of leptin increases. The presence of LEPR in the absorptive cells of the small intestine suggests that leptin may have a physiological role in the regulation of nutrient absorption.

Leptin Receptor (LEPR) Antibody (N-term) Blocking peptide - References

Schroth, M., et al., J. Clin. Endocrinol. Metab. 88(11):5497-5501 (2003).Couturier, C., et al., J. Biol. Chem. 278(29):26604-26611 (2003).Gavrila, A., et al., J. Clin. Endocrinol. Metab. 88(6):2838-2843 (2003).Yannakoulia, M., et al., J. Clin. Endocrinol. Metab. 88(4):1730-1736 (2003).Kado, N., et al., Hum. Reprod. 18(4):715-720 (2003).