Product datasheet

Anti-IGF1 Receptor antibody [alphaIR3] ab16890

Overview

Product name  Anti-IGF1 Receptor antibody [alphaIR3]
Description  Mouse monoclonal [alphaIR3] to IGF1 Receptor
Host species  Mouse
Specificity  Recognizes the human IGF-I Receptor. IGF-I Receptor (Ab-1) immunoprecipitates the α and β subunits of the IGF-I receptor. IGF-I receptor (ab16890) blocks IGF-I binding to its receptor and may bind weakly to the insulin receptor (see application references). It also inhibits the growth of MCF-7 cells in culture suggesting the IGF-I receptor may be involved in autocrine regulation of cell growth (see application references). This antibody is not recommended for Western blotting or paraffin sections.

Tested applications  
Suitable for: Flow Cyt, IP, Neutralising, ICC/IF
Unsuitable for: IHC-P or WB

Species reactivity  Reacts with: Hamster, Human

Immunogen  Tissue, cells or virus corresponding to IGF1 Receptor. Partially purified receptor from human placenta.


General notes  This product was changed from ascites to tissue culture supernatant on 17 May 2019. Please note that the dilutions may need to be adjusted accordingly. If you have any questions, please do not hesitate to contact our scientific support team.

Properties

Form  Liquid
Storage instructions  Shipped at 4°C. Store at +4°C.
Storage buffer  pH: 7.40
Constituent: PBS

Purity  Tissue culture supernatant

Purification notes  Purified from TCS.

Clonality  Monoclonal
Clone number  alphaIR3
Isotype  IgG1
Function

Receptor tyrosine kinase which mediates actions of insulin-like growth factor 1 (IGF1). Binds IGF1 with high affinity and IGF2 and insulin (INS) with a lower affinity. The activated IGF1R is involved in cell growth and survival control. IGF1R is crucial for tumor transformation and survival of malignant cell. Ligand binding activates the receptor kinase, leading to receptor autophosphorylation, and tyrosines phosphorylation of multiple substrates, that function as signaling adapter proteins including, the insulin-receptor substrates (IRS1/2), Shc and 14-3-3 proteins. Phosphorylation of IRSs proteins lead to the activation of two main signaling pathways: the PI3K-AKT/PKB pathway and the Ras-MAPK pathway. The result of activating the MAPK pathway is increased cellular proliferation, whereas activating the PI3K pathway inhibits apoptosis and stimulates protein synthesis. Phosphorylated IRS1 can activate the 85 kDa regulatory subunit of PI3K (PIK3R1), leading to activation of several downstream substrates, including protein AKT/PKB. AKT phosphorylation, in turn, enhances protein synthesis through mTOR activation and triggers the antiapoptotic effects of IGF1R through phosphorylation and inactivation of BAD. In parallel to PI3K-driven signaling, recruitment of Grb2/SOS by phosphorylated IRS1 or Shc leads to recruitment of Ras and activation of the ras-MAPK pathway. In addition to these two main signaling pathways IGF1R signals also through the Janus kinase/signal transducer and activator of transcription pathway (JAK/STAT). Phosphorylation of JAK proteins can lead to phosphorylation/activation of signal transducers and activators of transcription (STAT) proteins. In particular activation of STAT3, may be essential for the transforming activity of IGF1R. The JAK/STAT pathway activates gene transcription and may be responsible for the transforming activity. JNK kinases can also be activated by the IGF1R. IGF1 exerts inhibiting activities on JNK activation via phosphorylation and inhibition of MAP3K5/ASK1, which is able to directly associate with the IGF1R.

When present in a hybrid receptor with INSR, binds IGF1. PubMed:12138094 shows that hybrid receptors composed of IGF1R and INSR isoform Long are activated with a high affinity by IGF1, with low affinity by IGF2 and not significantly activated by insulin, and that hybrid receptors composed of IGF1R and INSR isoform Short are activated by IGF1, IGF2 and insulin. In contrast, PubMed:16831875 shows that hybrid receptors composed of IGF1R and INSR isoform Long and hybrid receptors composed of IGF1R and INSR isoform Short have similar binding characteristics, both bind IGF1 and have a low affinity for insulin.
**Tissue specificity**

Found as a hybrid receptor with INSR in muscle, heart, kidney, adipose tissue, skeletal muscle, hepatoma, fibroblasts, spleen and placenta (at protein level). Expressed in a variety of tissues. Overexpressed in tumors, including melanomas, cancers of the colon, pancreas prostate and kidney.

**Involvement in disease**

Insulin-like growth factor 1 resistance

**Sequence similarities**

Belongs to the protein kinase superfamily. Tyr protein kinase family. Insulin receptor subfamily. Contains 4 fibronectin type-III domains. Contains 1 protein kinase domain.

**Post-translational modifications**

Autophosphorylated on tyrosine residues in response to ligand binding. Autophosphorylation occurs in trans, i.e. one subunit of the dimeric receptor phosphorylates tyrosine residues on the other subunit. Autophosphorylation occurs in a sequential manner; Tyr-1165 is predominantly phosphorylated first, followed by phosphorylation of Tyr-1161 and Tyr-1166. While every single phosphorylation increases kinase activity, all three tyrosine residues in the kinase activation loop (Tyr-1165, Tyr-1161 and Tyr-1166) have to be phosphorylated for optimal activity. Can be autophosphorylated at additional tyrosine residues (in vitro). Autophosphorylated is followed by phosphorylation of juxtamembrane tyrosines and C-terminal serines. Phosphorylation of Tyr-980 is required for IRS1- and SHC1-binding. Phosphorylation of Ser-1278 by GSK-3beta restrains kinase activity and promotes cell surface expression, it requires a priming phosphorylation at Ser-1282. Dephosphorylated by PTPN1. Polyubiquitinated at Lys-1168 and Lys-1171 through both 'Lys-48' and 'Lys-29' linkages, promoting receptor endocytosis and subsequent degradation by the proteasome. Ubiquitination is facilitated by pre-existing phosphorylation. Sumoylated with SUMO1. Controlled by regulated intramembrane proteolysis (RIP). Undergoes metalloprotease-dependent constitutive ectodomain shedding to produce a membrane-anchored 52 kDa C-Terminal fragment which is further processed by presenilin gamma-secretase to yield an intracellular 50 kDa fragment.

**Cellular localization**

Cell membrane.

**Images**

ICC/IF image of ab16890 stained HEK-293 (Human epithelial cell line from embryonic kidney) cells. The cells were 4% formaldehyde fixed (10 minutes) and then incubated in 1% BSA / 10% normal goat serum / 0.3M glycine in 0.1% PBS-Tween for 1 hour to permeabilize the cells and block non-specific protein-protein interactions. The cells were then incubated with the antibody (ab16890, 1 µg/ml) overnight at +4°C. The secondary antibody (green) was Alexa Fluor® 488 goat anti-mouse IgG (H+L) used at a 1/1000 dilution for 1 hour. Alexa Fluor® 594 WGA was used to label plasma membranes (red) at a 1/200 dilution for 1h. DAPI was used to stain the cell nuclei (blue) at a concentration of 1.43 µM.

This image was generated using the ascites version of the product.
Overlay histogram showing LoVo (Human colorectal adenocarcinoma cell line) cells stained with ab16890 (red line). The cells were fixed with 4% paraformaldehyde (10 minutes) and incubated in 1x PBS / 10% normal goat serum / 0.3M glycine to block non-specific protein-protein interactions. The cells were then incubated with the antibody (ab16890, 1/20 dilution) for 30 minutes at 22°C. The secondary antibody used was DyLight® 488 goat anti-mouse IgG (H+L) (ab96879) at 1/500 dilution for 30 minutes at 22°C. Isotype control antibody (black line) was mouse IgG1 [ICIGG1] (ab91353, 2 µg/1x10^6 cells) used under the same conditions. Acquisition of >5,000 events was performed. This antibody gave a positive signal in LoVo cells fixed with methanol (5 minutes) used under the same conditions.

Please note that Abcam do not have data for use of this antibody on non-fixed cells. We welcome any customer feedback. This image was generated using the ascites version of the product.

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