

Anti-IGF1R/CD221 Antibody (7Q293)

Product Details

Ig Type:	Mouse IgG2a
Reactivity:	Human
Conjugation:	Unconjugated
Clone:	7Q293
Purification:	Affinity-chromatography

Applications

Verified Activity:	Overlay Peak curve showing Hela cells surface stained with TMAH-00599 (red line) at 1:50. Then 10% normal goat serum was incubated to block non-specific protein-protein interactions followed by the antibody (1 μ g/1*10 ⁶ cells) for 45 min at 4°C. The secondary antibody used was FITC-conjugated Goat Anti-Mouse IgG(H+L) at 1/200 dilution for 35 min at 4°C. Isotype control antibody (green line) was mouse IgG1 (1 μ g/1*10 ⁶ cells) used under the same conditions. Acquisition of >10,008 events was performed.
Application:	ELISA, FCM
Recommended	FCM:1:20-1:500.

Properties

Stability & Storage:	Store at -20°C or -80°C for 12 months. Avoid repeated freeze-thaw cycles.
Shipping:	Shipping with blue ice.

Antigen Details

Immunogen:	Recombinant Protein: Human IGF1R Protein
Antigen Species:	Human
Gene ID:	3480
Uniprot ID:	P08069
Synonyms:	insulin-like growth factor 1 receptor;IGF-I R;CD221;JTK13;IGFIR;IGFR;IGF1 Receptor
Biology Area:	Cancer, Developmental biology, Signal transduction

Research Background

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.

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