

Product datasheet for **TA384486**

IGF1 Receptor (IGF1R) Rabbit Monoclonal Antibody [Clone ID: R06-6B1]

Product data:

Product Type:	Primary Antibodies
Clone Name:	R06-6B1
Applications:	IP, WB
Recommended Dilution:	WB: 1/1000 IP: 1/20
Reactivity:	Human, Mouse, Rat
Host:	Rabbit
Isotype:	IgG
Clonality:	Monoclonal
Immunogen:	A synthetic phosphopeptide corresponding to residues surrounding Tyr1166 of human INSR/IGF-1R (Phosphorylated)
Formulation:	50mM Tris-Glycine(pH 7.4), 0.15M NaCl, 40% Glycerol, 0.01% Sodium azide and 0.05% BSA
Concentration:	lot specific
Purification:	Affinity Purified
Conjugation:	Unconjugated
Storage:	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles.
Stability:	1 year
Predicted Protein Size:	Calculated MW: 155 kDa; Observed MW: 95 kDa
Gene Name:	insulin like growth factor 1 receptor
Database Link:	Entrez Gene 3480 Human P08069



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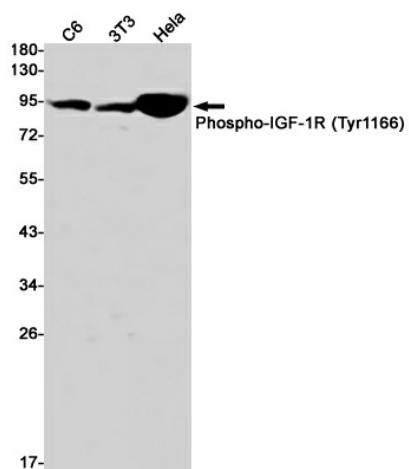
Background:

Swiss-Prot Acc.P08069.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.

Synonyms:

CD221; IGFI; IGFIR; JTK13; MGC18216; MGC142170; MGC142172

Product images:



Western blot analysis of Phospho-IGF1R (Tyr1166) in C6, 3T3, HeLa lysates using Phospho-IGF1 Receptor (Tyr1166) antibody.