

IGF1R (Ab-1346) Conjugated Antibody

Catalog No: #C33217



Package Size: #C33217-AF350 100ul #C33217-AF405 100ul #C33217-AF488 100ul
 #C33217-AF555 100ul #C33217-AF594 100ul #C33217-AF647 100ul
 #C33217-AF680 100ul #C33217-AF750 100ul #C33217-Biotin 100ul

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Description

Product Name	IGF1R (Ab-1346) Conjugated Antibody
Host Species	Rabbit
Clonality	Polyclonal
Species Reactivity	Hu
Specificity	The antibody detects endogenous levels of total IGF1R protein.
Immunogen Description	Synthesized non-phosphopeptide derived from human IGF1R around the phosphorylation site of tyrosine 1346 (Q-P-Y(p)-A-H).
Conjugates	Biotin AF350 AF405 AF488 AF555 AF594 AF647 AF680 AF750
Other Names	CD221;EC 2.7.10.1;IGF1R;Insulin-like growth factor I receptor precursor;kinase IGF1R
Accession No.	Swiss-Prot#:P08069NCBI Gene ID:3480
Uniprot	P08069
GeneID	3480;
Excitation Emission	AF350: 346nm/442nm AF405: 401nm/421nm AF488: 493nm/519nm AF555: 555nm/565nm AF594: 591nm/614nm AF647: 651nm/667nm AF680: 679nm/702nm AF750: 749nm/775nm
Calculated MW	145
Formulation	0.01M Sodium Phosphate, 0.25M NaCl, pH 7.6, 5mg/ml Bovine Serum Albumin, 0.02% Sodium Azide
Storage	Store at 4°C in dark for 6 months

Application Details

Suggested Dilution:

AF350 conjugated: most applications: 1: 50 - 1: 250
 AF405 conjugated: most applications: 1: 50 - 1: 250
 AF488 conjugated: most applications: 1: 50 - 1: 250
 AF555 conjugated: most applications: 1: 50 - 1: 250
 AF594 conjugated: most applications: 1: 50 - 1: 250
 AF647 conjugated: most applications: 1: 50 - 1: 250
 AF680 conjugated: most applications: 1: 50 - 1: 250
 AF750 conjugated: most applications: 1: 50 - 1: 250

Product Description

The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.

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. shows that hybrid receptors composed of IGF1R and INSR isoform Longare 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 Shortare activated by IGF1, IGF2 and insulin. In contrast, shows that hybrid receptors composed of IGF1R and INSR isoform Longand hybrid receptors composed of IGF1R and INSR isoform Shorthave similar binding characteristics, both bind IGF1 and have a low affinity for insulin.

Note: This product is for in vitro research use only