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Anti-IGF1R / CD221 Reference Antibody (teprotumumab)

Recombinant Antibody
Catalog # APR10191

Product Information

Application	Functional assay, Kinetics (SPR), Kinetics (BLI), FACS, E, FTA
Primary Accession	P08069
Reactivity	Human, Mouse
Clonality	Monoclonal
Isotype	IgG1
Calculated MW	154793

Additional Information

Target/Specificity	IGF1R / CD221
Endotoxin	
Conjugation	Unconjugated
Expression system	CHO
Format	Purified monoclonal antibody supplied in PBS, pH6.0, without preservative.This antibody is purified through a protein A column.

Protein Information

Name	IGF1R
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 IGFIR 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.

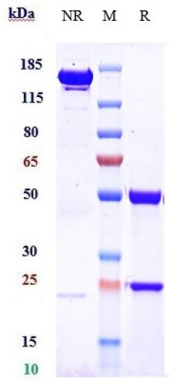
Cellular Location

Cell membrane; Single-pass type I membrane protein

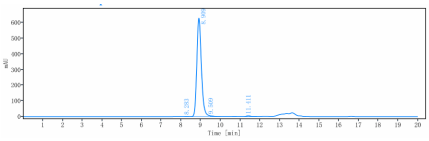
Tissue Location

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.

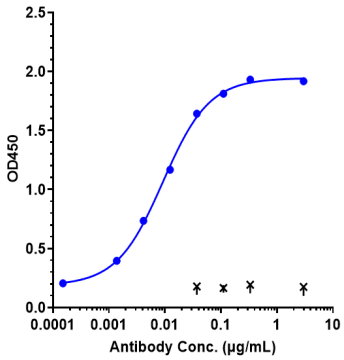
Images



Anti-IGF1R / CD221 Reference Antibody (teprotumumab) on SDS-PAGE under reducing (R) condition. The purity of the protein is greater than 95%.

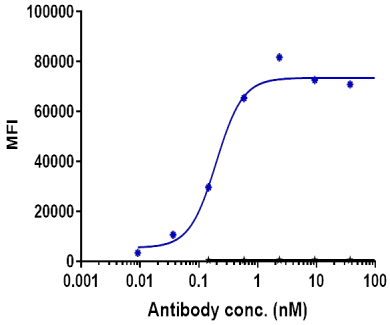


The purity of Anti-IGF1R / CD221 Reference Antibody (teprotumumab) is 96.97%, determined by SEC-HPLC.



Immobilized human IGF1R His at 2 ug/mL can bind Anti-IGF1R / CD221 Reference Antibody (teprotumumab), EC50=0.009199 ug/mL.

Human IGF1R CHO-K1 cells were stained with Anti-IGF1R / CD221 Reference Antibody (teprotumumab) and negative control protein respectively, washed and then followed by APC and analyzed with FACS, EC50=0.1968 nM.



Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.