

IGF-1R(Phospho-Tyr1161) Antibody

Catalog No: #11087

Package Size: #11087-1 50ul #11087-2 100ul

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Description

Product Name	IGF-1R(Phospho-Tyr1161) Antibody
Host Species	Rabbit
Clonality	Polyclonal
Purification	Antibodies were produced by immunizing rabbits with synthetic phosphopeptide and KLH conjugates. Antibodies were purified by affinity-chromatography using epitope-specific phosphopeptide. Non-phospho specific antibodies were removed by chromatography using non-phosphopeptide.
Applications	WB IHC IF
Species Reactivity	Hu Ms Rt
Specificity	The antibody detects endogenous level of IGF-1R only when phosphorylated at tyrosine 1161.
Immunogen Type	Peptide-KLH
Immunogen Description	Peptide sequence around phosphorylation site of tyrosine 1161(D-I-Y(p)-E-T)derived from Human IGF-1R.
Target Name	IGF-1R
Modification	Phospho
Other Names	IGFR; CD221; IGFI; JTK13
Accession No.	Swiss-Prot: P08069NCBI Gene ID: 3480NCBI mRNA: NM_000875.3NCBI Protein: NP_000866.1
Uniprot	P08069
GeneID	3480;
Concentration	1.0mg/ml
Formulation	Supplied at 1.0mg/mL in phosphate buffered saline (without Mg ²⁺ and Ca ²⁺), pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol.
Storage	Store at -20°C

Application Details

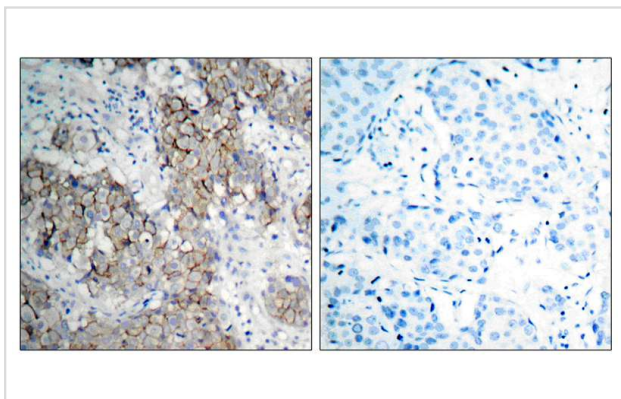
Predicted MW: 95oO 200kd

Western blotting: 1:500~1:1000

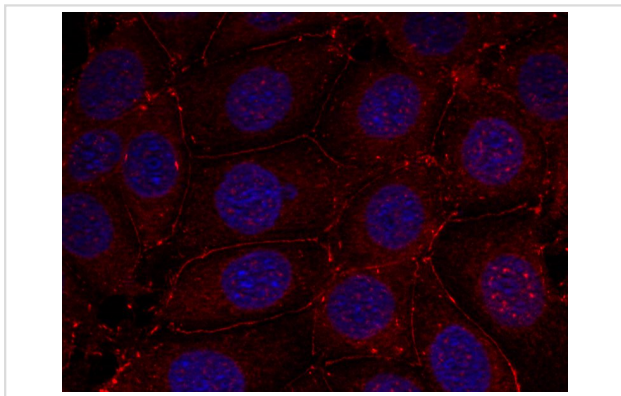
Immunohistochemistry: 1:50~1:100

Immunofluorescence: 1:100~1:200

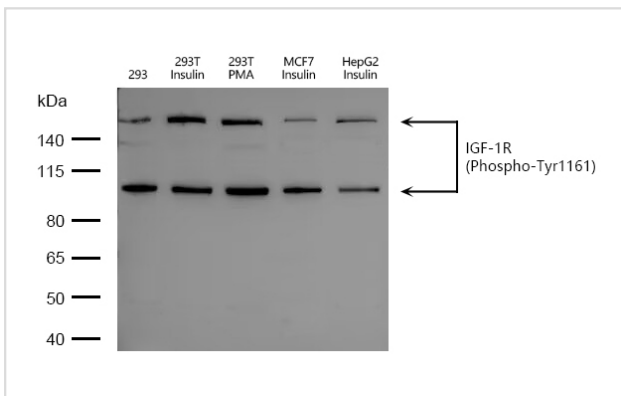
Images



Immunohistochemical analysis of paraffin- embedded human breast carcinoma tissue using IGF-1R (phospho-Tyr1161) antibody (#11087).



Immunofluorescence staining of methanol-fixed MCF7 cells using IGF-1R (phospho-Tyr1161) antibody (#11087, Red).



Western blot analysis of IGF-1R(Phospho-Tyr1161) expression in 293,293T Insulin,293T PMA,MCF7 Insulin,HepG2 Insulin

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 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, 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.

Kasuya J., Paz I.B., Maddux B.A., Goldfine I.D., Hefta S.A., Fujita-Yamaguchi Y. *Biochemistry* 32:13531-13536(1993)

Slaaby R., Schaeffer L., Lautrup-Larsen I., Andersen A.S., Shaw A.C., Mathiasen I.S., Brandt J.J. *Biol. Chem.* 281:25869-25874(2006)

Wu J., Li W., Craddock B.P., Foreman K.W., Mulvihill M.J., Ji Q.S., Miller W.T., Hubbard S.R. *EMBO J.* 27:1985-1994(2008)

Note: This product is for in vitro research use only