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IGF1R Antibody (N-term K66) Blocking Peptide
Synthetic peptide
Catalog # BP7649d

Specification

IGF1R Antibody (N-term K66) Blocking Peptide - Product Information

Primary Accession P08069

IGF1R Antibody (N-term K66) Blocking Peptide - Additional Information

Gene ID 3480

Other Names
Insulin-like growth factor 1 receptor, Insulin-like growth factor I receptor, IGF-I receptor, CD221, Insulin-like growth factor 1 receptor alpha chain, Insulin-like growth factor 1 receptor beta chain, IGF1R

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

IGF1R Antibody (N-term K66) Blocking Peptide - 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.

IGF1R Antibody (N-term K66) Blocking Peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides

IGF1R Antibody (N-term K66) Blocking Peptide - Images

IGF1R Antibody (N-term K66) Blocking Peptide - Background

The IGF1R receptor binds insulin-like growth factor with a high affinity and plays a critical role in transformation events. Cleavage of the precursor generates alpha and beta subunits. It is highly overexpressed in most malignant tissues where it functions as an anti-apoptotic agent by enhancing cell survival. The protein possess tyrosine kinase activity.

IGF1R Antibody (N-term K66) Blocking Peptide - References

Song, R.X., et al., Proc. Natl. Acad. Sci. U.S.A. 101(7):2076-2081 (2004).Zhao, H., et al., Oncogene 23(3):786-794 (2004).Lu, Y., et al., Biochem. Biophys. Res. Commun. 313(3):709-715 (2004).Hakam, A., et al., Dig. Dis. Sci. 48(10):1972-1978 (2003).Li, Y., et al., Arterioscler. Thromb. Vasc. Biol. 23(12):2178-2184 (2003).