

IGF1R Antibody (N-term K66)
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP7649d

Specification

IGF1R Antibody (N-term K66) - Product Information

| | |
|-------------------|---|
| Application | IF, WB, IHC-P, FC,E |
| Primary Accession | P08069 |
| Other Accession | P24062 , Q60751 |
| Reactivity | Human |
| Predicted | Mouse, Rat |
| Host | Rabbit |
| Clonality | Polyclonal |
| Isotype | Rabbit IgG |
| Antigen Region | 51-77 |

IGF1R Antibody (N-term K66) - 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

Target/Specificity

This IGF1R antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 51-77 amino acids from the N-terminal region of human IGF1R.

Dilution

IF~~1:10~50
WB~~1:1000
IHC-P~~1:50~100
FC~~1:10~50

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

IGF1R Antibody (N-term K66) is for research use only and not for use in diagnostic or therapeutic procedures.

IGF1R Antibody (N-term K66) - 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 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.

Cellular Location

Cell membrane; Single-pass type I membrane protein

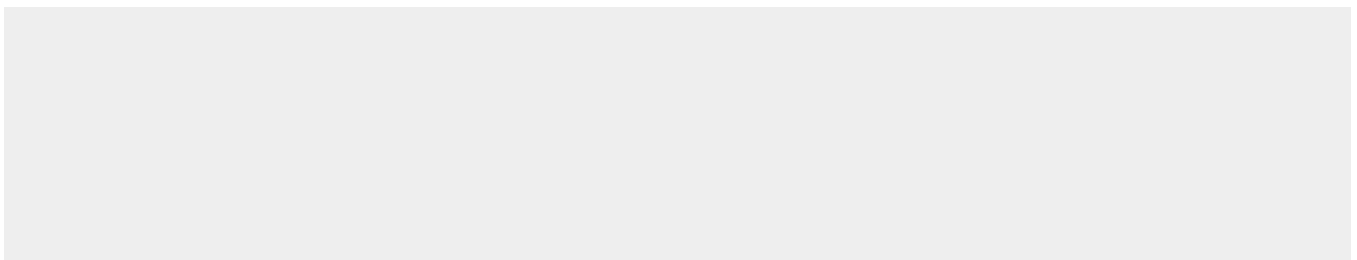
Tissue Location

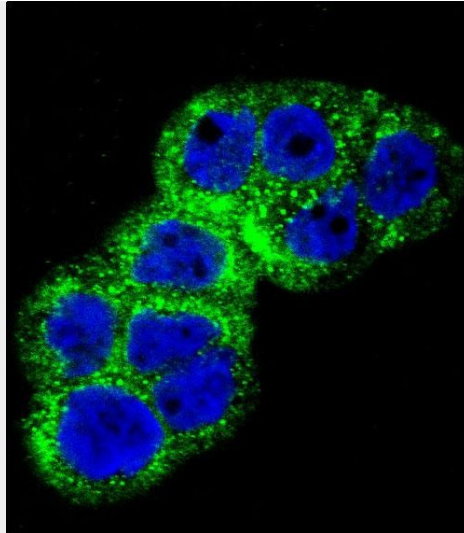
Found as a hybrid receptor with INSR in muscle, heart, kidney, adipose tissue, skeletal muscle, hepatoma, fibroblasts, spleen and placenta (at protein level). Expressed in a variety of tissues. Overexpressed in tumors, including melanomas, cancers of the colon, pancreas prostate and kidney.

IGF1R Antibody (N-term K66) - Protocols

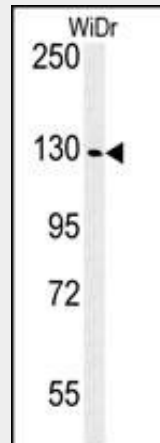
Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

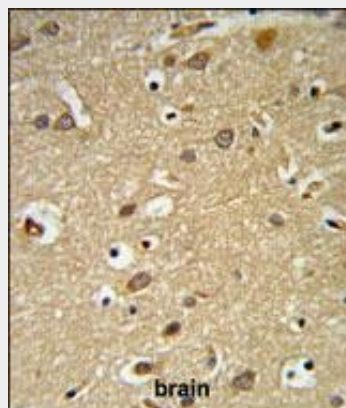
IGF1R Antibody (N-term K66) - Images



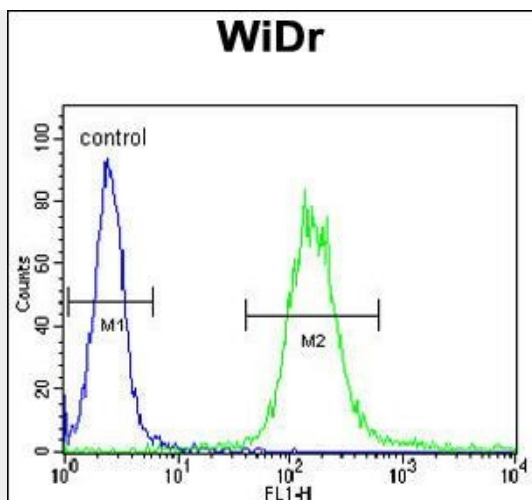
Confocal immunofluorescent analysis of IGF1R Antibody (N-term K66)(Cat#AP7649d) with WiDr cell followed by Alexa Fluor 488-conjugated goat anti-rabbit IgG (green). DAPI was used to stain the cell nuclear (blue).



Western blot analysis of IGF1R Antibody (N-term K66) (Cat. #AP7649d) in WiDr cell line lysates (35ug/lane).IGF1R (arrow) was detected using the purified Pab.



IGF1R Antibody (N-term K66) (Cat. #AP7649d) IHC analysis in formalin fixed and paraffin embedded brain tissue followed by peroxidase conjugation of the secondary antibody and DAB staining. This data demonstrates the use of the IGF1R Antibody (N-term K66) for immunohistochemistry. Clinical relevance has not been evaluated.



IGF1R Antibody (N-term K66) (Cat. #AP7649d) flow cytometric analysis of WiDr cells (right histogram) compared to a negative control (PBS alone) (left histogram). FITC-conjugated goat-anti-rabbit secondary antibodies were used for the analysis.

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