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# Mouse Igf1r Antibody (Center) Blocking Peptide

**Synthetic peptide**
**Catalog # BP14622c**

## Specification

### Mouse Igf1r Antibody (Center) Blocking Peptide - Product Information

Primary Accession Q60751

### Mouse Igf1r Antibody (Center) Blocking Peptide - Additional Information

**Gene ID** 16001

**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.

### Mouse Igf1r Antibody (Center) 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 (By similarity). When present in a hybrid receptor with INSR, binds IGF1 (By similarity).

**Cellular Location**
Cell membrane; Single-pass type I membrane protein

## Mouse Igf1r Antibody (Center) Blocking Peptide - Protocols

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

- Blocking Peptides

## Mouse Igf1r Antibody (Center) Blocking Peptide - Images

## Mouse Igf1r Antibody (Center) Blocking Peptide - Background

This receptor binds insulin-like growth factor 1 (IGF1) with a high affinity and IGF2 with a lower affinity. It has a tyrosine-protein kinase activity, which is necessary for the activation of the IGF1-stimulated downstream signaling cascade. When present in a hybrid receptor with INSR, binds IGF1 (By similarity).

## Mouse Igf1r Antibody (Center) Blocking Peptide - References

Heron-Milhavet, L., et al. J. Cell. Physiol. 225(1):1-6(2010)Dinchuk, J.E., et al. Endocrinology 151(9):4123-4132(2010)Divall, S.A., et al. J. Clin. Invest. 120(8):2900-2909(2010)Boucher, J., et al. J. Biol. Chem. 285(22):17235-17245(2010)Tomizawa, M., et al. World J. Gastroenterol. 16(15):1854-1858(2010)