



# Insulin R Monoclonal Antibody

<b>Catalog No</b>	YP-mAb-13382
<b>Isotype</b>	IgG
<b>Reactivity</b>	Human;Rat;Mouse;;Pig
<b>Applications</b>	WB
<b>Gene Name</b>	INSR
<b>Protein Name</b>	Insulin receptor
<b>Immunogen</b>	The antiserum was produced against synthesized peptide derived from human IR. AA range:1326-1375
<b>Specificity</b>	Insulin R Monoclonal Antibody detects endogenous levels of Insulin R protein.
<b>Formulation</b>	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
<b>Source</b>	Monoclonal, Mouse,IgG
<b>Purification</b>	The antibody was affinity-purified from mouse antiserum by affinity-chromatography using epitope-specific immunogen.
<b>Dilution</b>	WB 1:500-1:2000
<b>Concentration</b>	1 mg/ml
<b>Purity</b>	≥90%
<b>Storage Stability</b>	-20°C/1 year
<b>Synonyms</b>	INSR; Insulin receptor; IR; CD antigen CD220
<b>Observed Band</b>	155kD
<b>Cell Pathway</b>	Cell membrane ; Single-pass type I membrane protein . Late endosome . Lysosome . Binding of insulin to INSR induces internalization and lysosomal degradation of the receptor, a means for down-regulating this signaling pathway after stimulation. In the presence of SORL1, internalized INSR molecules are redirected back to the cell surface, thereby preventing their lysosomal catabolism and strengthening insulin signal reception. .
<b>Tissue Specificity</b>	Isoform Long and isoform Short are predominantly expressed in tissue targets of insulin metabolic effects: liver, adipose tissue and skeletal muscle but are also expressed in the peripheral nerve, kidney, pulmonary alveoli, pancreatic acini, placenta vascular endothelium, fibroblasts, monocytes, granulocytes, erythrocytes and skin. Isoform Short is preferentially expressed in fetal cells such as fetal fibroblasts, muscle, liver and kidney. Found as a hybrid receptor with IGF1R in muscle, heart, kidney, adipose tissue, skeletal muscle, hepatoma, fibroblasts, spleen and placenta (at protein level). Overexpressed in several tumors, including breast, colon, lung, ovary, and thyroid carcinomas.
<b>Function</b>	catalytic activity:ATP + a [protein]-L-tyrosine = ADP + a [protein]-L-tyrosine phosphate.;disease:Defects in INSR are the cause of familial hyperinsulinemic hypoglycemia 5 (HHF5) [MIM:609968]. Familial hyperinsulinemic hypoglycemia [MIM:256450], also referred to as congenital hyperinsulinism, nesidioblastosis, or



persistent hyperinsulinemic hypoglycemia of infancy (PPHI), is the most common cause of persistent hypoglycemia in infancy and is due to defective negative feedback regulation of insulin secretion by low glucose levels.,disease:Defects in INSR are the cause of insulin resistance (Ins resistance) [MIM:125853].,disease:Defects in INSR are the cause of insulin-resistant diabetes mellitus with acanthosis nigricans type A (IRAN type A) [MIM:610549]. This syndrome is characterized by the association of severe insulin resistance (manifested by marked hyperinsulinemia and a failure to r

**Background**

This gene encodes a member of the receptor tyrosine kinase family of proteins. The encoded preproprotein is proteolytically processed to generate alpha and beta subunits that form a heterotetrameric receptor. Binding of insulin or other ligands to this receptor activates the insulin signaling pathway, which regulates glucose uptake and release, as well as the synthesis and storage of carbohydrates, lipids and protein. Mutations in this gene underlie the inherited severe insulin resistance syndromes including type A insulin resistance syndrome, Donohue syndrome and Rabson-Mendenhall syndrome. Alternative splicing results in multiple transcript variants. [provided by RefSeq, Oct 2015],

**matters needing attention**

Avoid repeated freezing and thawing!

**Usage suggestions**

This product can be used in immunological reaction related experiments. For more information, please consult technical personnel.

## Products Images