



# IR (Phospho Tyr999) mouse mAb

Catalog No	BYmab-13123
Isotype	IgG
Reactivity	Human;Mouse;Rat
Applications	WB
Gene Name	INSR
Protein Name	IR (Phospho Tyr999)
Immunogen	Synthesized peptide derived from human IR (Phospho Tyr999)
Specificity	This antibody detects endogenous levels of Human,Mouse,Rat IR (Phospho Tyr999)
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Source	Monoclonal, Mouse,IgG
Purification	The antibody was affinity-purified from mouse antiserum by affinity-chromatography using epitope-specific immunogen.
Dilution	WB 1:500-2000
Concentration	1 mg/ml
Purity	≥90%
Storage Stability	-20°C/1 year
Synonyms	Insulin receptor (IR;EC 2.7.10.1;CD antigen CD220) [Cleaved into: Insulin receptor subunit alpha; Insulin receptor subunit beta]
Observed Band	95kD
Cell Pathway	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. .
Tissue Specificity	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.

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<b>Function</b>	<p>MAPKKK cascade, activation of MAPK activity, regulation of protein amino acid phosphorylation, negative regulation of protein amino acid phosphorylation, positive regulation of protein amino acid phosphorylation, reproductive developmental process, heart morphogenesis, regulation of glycogen biosynthetic process, regulation of carbohydrate metabolic process, regulation of glycolysis, regulation of DNA replication, regulation of transcription, DNA-dependent, protein complex assembly, protein amino acid phosphorylation, phosphorus metabolic process, phosphate metabolic process, regulation of mitosis, cell surface receptor linked signal transduction, enzyme linked receptor protein signaling pathway, transmembrane receptor protein tyrosine kinase signaling pathway, G-protein coupled receptor protein signaling pathway, intracellular signaling cascade, protein kinase cascade, regulation of mito</p>
<b>Background</b>	<p>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 respond to exogenous insulin) with the skin lesion acanthosis nigricans and ovarian hyperandrogenism in adolescent female subjects. Women frequently present with hirsutism, acne, amenorrhea or oligomenorrhea, and virilization. This syndrome is different from the type B that has been demonstrated to be secondary to the presence of circulating autoantibodies against the insulin receptor.,disease:Defects in INSR are the cause of leprechaunism [MIM:246200]; also known as Donohue syndrome. Leprechaunism represents the most severe form of insulin resistance syndrome, characterized by intrauterine and postnatal growth retardation and death in early infancy. Inheritance is autosomal recessive.,disease:Defects in INSR are the cause of Rabson-Mendenhall syndrome [MIM:262190]; also known as Mendenhall syndrome. It is a severe insulin resistance syndrome characterized by insulin-resistant diabetes mellitus with pineal hyperplasia and somatic abnormalities. Typical features include coarse, senile-appearing facies, dental and skin abnormalities, abdominal distension, and phallic enlargement. Inheritance is autosomal recessive.,disease:Defects in INSR may be associated with noninsulin-dependent diabetes mellitus (NIDDM) [MIM:125853]; also known as diabetes mellitus type 2.,enzyme regulation:Autophosphorylation activates the kinase activity.,function:This receptor binds insulin and has a tyrosine-protein kinase activity. Isoform Short has a higher affinity for insulin. Mediates the metabolic functions of insulin. Binding to insulin stimulates association of the receptor with downstream mediators including IRS1 and phosphatidylinositol 3'-kinase (PI3K). Can activate PI3K either directly by binding to the p85 regulatory subunit, or indirectly via IRS1. When present in a hybrid receptor with IGF1R, binds IGF1. PubMed:12138094 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, PubMed:16831875 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.,online information:Insulin receptor entry,PTM:After being transported from the endoplasmic reticulum to the Golgi apparatus, the single glycosylated precursor is further glycosylated and then cleaved, followed by its transport to the plasma membrane.,PTM:Autophosphorylated on tyrosine residues in response to insulin.,PTM:Phosphorylation of Tyr-999 is required for IRS1- and SHC1-binding.,similarity:Belongs to the protein kinase superfamily. Tyr protein kinase family. Insulin receptor subfamily.,similarity:Contains 1 protein kinase domain.,similarity:Contains 3 fibronectin type-III domains.,subunit:Tetramer of 2 alpha and 2 beta chains linked by disulfide bonds. The alpha chains contribute to the formation of the ligand-binding domain, while the beta chains carry the kinase domain. Interacts with SORBS1 but dissociates</p>

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from it following insulin stimulation. Binds SH2B2. Interacts with the PTB/PID domains of IRS1 and SHC1 in vitro when autophosphorylated on tyrosine residues. The sequences surrounding the phosphorylated NPXY motif contribute differentially to either IRS1 or SHC1 recognition. Interacts with the SH2 domains of the 85 kDa regulatory subunit of PI3K (PIK3R1) in vitro, when autophosphorylated on tyrosine residues. Interacts with SOCS7. Forms a hybrid receptor with IGF1R, the hybrid is a tetramer consisting of 1 alpha chain and 1 beta chain of INSR and 1 alpha chain and 1 beta chain of IGF1R.,tissue specificity:Found as a hybrid receptor with IGF1R in muscle, heart, kidney, adipose tissue, skeletal muscle, hepatoma, fibroblasts, spleen and placenta (at protein level). Isoform Long and isoform Short are expressed in the peripheral nerve, kidney, liver, striated muscle, fibroblasts and skin. Isoform Short is expressed also in the spleen and lymphoblasts.,

**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

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