



Insulin Receptor β Rabbit mAb

Catalog No	YP-rAb-17590
Isotype	IgG
Reactivity	Human,Mouse,Rat,Pig
Applications	WB,IHC,IF,IP,ELISA
Gene Name	INSR
Protein Name	Insulin Receptor β
Purification Process	Protein A
Specificity	Endogenous
Formulation	PBS, 50% glycerol, 0.05% Proclin 300, 0.05%BSA
Source	Monoclonal, Rabbit,IgG
Dilution	IHC 1:200-1:1000; WB 1:2000-1:10000; IF 1:200-1:1000; ELISA 1:5000-1:20000; IP 1:50-1:200; Note: For IHC, we suggest antigen retrieval with TE buffer pH 9.0
Concentration	0.5 mg/ml
Purity	$\geq 90\%$
Storage Stability	-15°C to -25°C /1 year(Do not lower than -25°C)
Synonyms	Insulin receptor ; IR ; CD antigen CD220 ; [Cleaved into: Insulin receptor subunit alpha ; Insulin receptor subunit beta]
Observed Band	95kD
Calculated Molecular Weight	156kD
Cell Pathway	Membrane
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.
Function	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

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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 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.,

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

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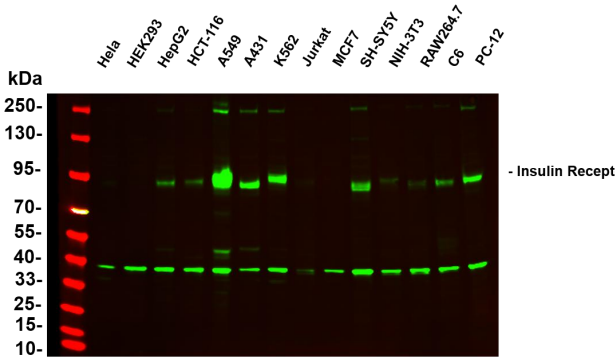
syndrome, Donohue syndrome and Rabson-Mendenhall syndrome. Alternative splicing results in multiple transcript variants. [provided by RefSeq, Oct 2015],

matters needing attention

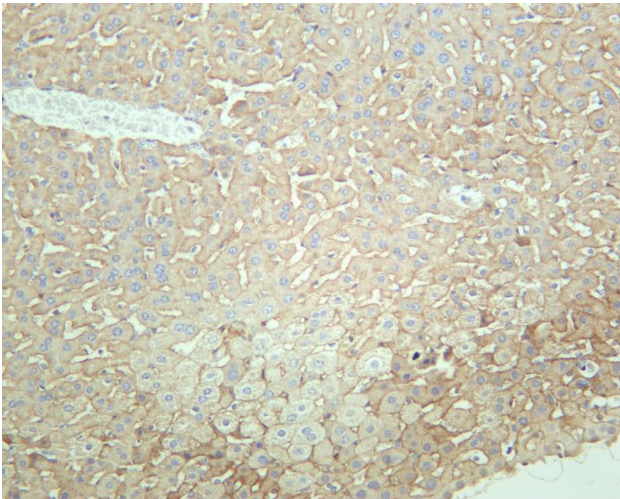
Avoid repeated freezing and thawing!

Usage suggestions

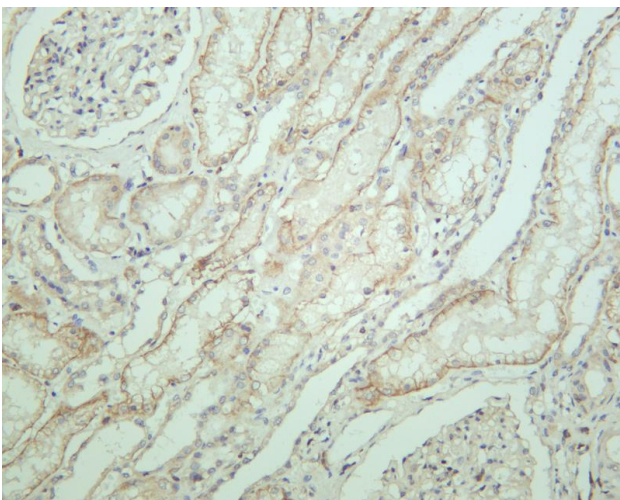
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Various whole cell lysates were separated by 4-20% SDS-PAGE, and the primary antibody was used at 4~C, over night with a 1:5000 dilution. The Dylight 800-conjugated Goat anti-Rabbit antibody

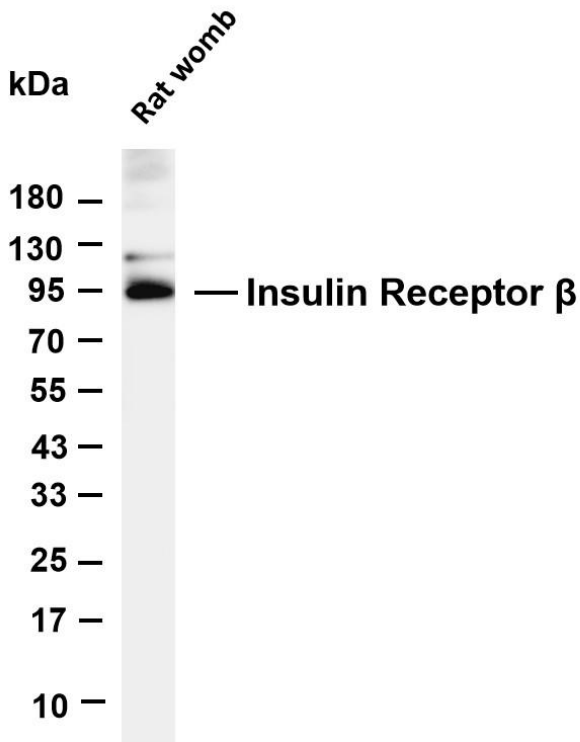


Mouse liver was stained with anti-Insulin Receptor β rabbit antibody



Human kidney was stained with anti-Insulin Receptor β rabbit antibody





Various whole cell lysates were separated by 4-20% SDS-PAGE, and the membrane was blotted with anti-Insulin Receptor β antibody. The HRP-conjugated Goat anti-Rabbit IgG(H + L) antibody was used to detect the antibody. Lane 1: Rat womb Predicted band size: 156kDa Observed band size: 95kDa

