



Insulin Receptor β (phospho-Tyr1345) rabbit pAb

Catalog No	YP-Ab-13112
Isotype	IgG
Reactivity	Human;Rat;Mouse;
Applications	WB
Gene Name	INSR
Immunogen	Synthesized phosho peptide around human Insulin Receptor β (Tyr1345)
Specificity	This antibody detects endogenous levels of Human Insulin Receptor β (phospho-Tyr1345)
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Source	Polyclonal, Rabbit,IgG
Purification	The antibody was affinity-purified from rabbit serum by affinity-chromatography using specific immunogen.
Dilution	WB 1:1000-2000
Concentration	1 mg/ml
Purity	$\geq 90\%$
Storage Stability	-20°C/1 year
Synonyms	Insulin R;INSR;Insulin Receptor β (Tyr1345);Insulin receptor (IR) (EC 2.7.10.1) (CD antigen CD220) [Cleaved into: Insulin receptor subunit alpha;Insulin receptor subunit beta]
Observed Band	95kD
Calculated Molecular Weight	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.

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 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.

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