



Insulin R Polyclonal Antibody

Catalog No	BYab-13382	
Isotype	IgG	
Reactivity	Human;Rat;Mouse;;Pig	
Applications	IHC;IF;WB;ELISA	
Gene Name	INSR	
Protein Name	Insulin receptor	
Immunogen	The antiserum was produced against synthesized peptide derived from human IR AA range:1326-1375	
Specificity	Insulin R Polyclonal Antibody detects endogenous levels of Insulin R protein.	
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 antiserum by affinity-chromatography using epitope-specific immunogen.	
Dilution	WB 1:500-2000 Immunohistochemistry: 1/100 - 1/300. Immunofluorescence: 1/200 - 1/1000. ELISA: 1/5000. Not yet tested in other applications.	
Concentration	1 mg/ml	
Purity	≥90%	
Storage Stability	-20°C/1 year	
Synonyms	INSR; Insulin receptor; IR; CD antigen CD220	
Observed Band	155kD	
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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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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Products Images			
(b) IR- α 165 kDa IR- β 95 kDa P-IR (Y1361) 100 kDa β -actin 43 kDa $c_{control m} r_{1100} r_{B} r_{B} r_{110} r_{B} r_{B} r_{110} r_{B} r_{$	Han, Xiaojuan, et al. "Insulin attenuates beta-amyloid-associated insulin/Akt/EAAT signaling perturbations in human astrocytes." Cellular and molecular neurobiology 36.6 (2016): 851-864.		
	Immunofluorescence analysis of HUVEC cells, using IR Antibody. The picture on the right is blocked with the synthesized peptide.		
	Immunohistochemistry analysis of paraffin-embedded human breast carcinoma tissue, using IR Antibody. The picture on the right is blocked with the synthesized peptide.		

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