

Rabbit IR Polyclonal Antibody | anti-IR antibody

Phospho-IR (Tyr1361) Antibody

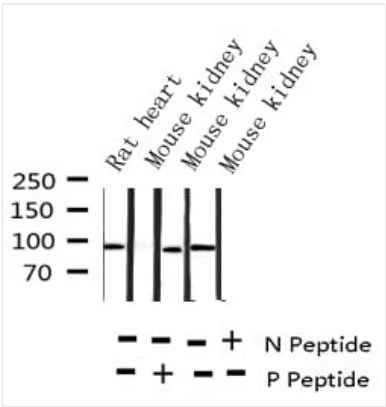
CATALOG # AAA321480	HOST Rabbit	REACTIVITY Human, Mouse, Rat	APPLICATIONS ELISA, Immunohistochemistry, Western Blot
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PRODUCT SYNONYMS

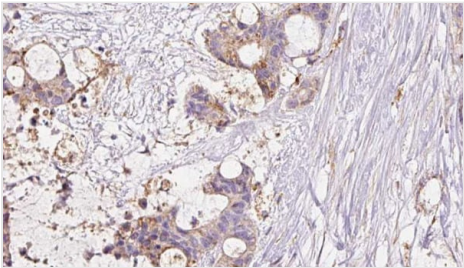
IR; Polyclonal Antibody; Phospho-IR (Tyr1361) Antibody; CD220; HHF5; HIR B; INSR; Insulin receptor; Insulin receptor subunit beta; anti-IR antibody

PRODUCT SPECIFICATIONS

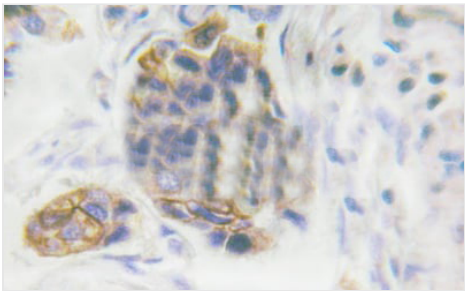
Host	Rabbit
Reactivity	Human, Mouse, Rat
Clonality	Polyclonal
Isotype	IgG
Specificity	Phospho-IR (Tyr1361) antibody detects endogenous levels of IR only when phosphorylated at Tyrosine 1361
Purity/Purification	From purified rabbit serum by affinity purification via sequential chromatography on phospho-and non-phospho-peptide affinity columns.
Form/Format	Liquid Phosphate buffered saline, pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol.
Concentration	1mg/ml (varies by lot)
Sequence Length	1382
Applicable Applications for anti-IR antibody	ELISA, IHC (Immunohistochemistry), WB (Western Blot)
Immunogen	A synthesized peptide derived from human IR around the phosphorylation site of Tyrosine 1361
Subcellular Location	Membrane; Single-Pass Type I Membrane Protein.
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.
Predicted Cross Reactivity	Pig, Bovine, Sheep, Rabbit, Dog, Chicken, Xenopus
Similarity	Pig (92%), Bovine (92%), Sheep (92%), Rabbit (92%), Dog (100%), Chicken (100%), Xenopus (100%)
Conjugation	Unconjugated
Epitope	Phospho Tyr1361
Preparation and Storage	Store at -20 degree C. Stable for 12 months from date of receipt.



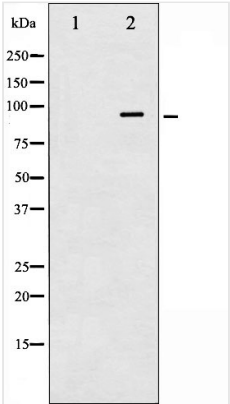
WB (Western Blot)
Western blot analysis of Phospho-IR (Tyr1361) expression in various lysates



IHC (Immunohistochemistry)
AAA321480 at 1/100 staining Human liver cancer tissue by IHC-P. The sample was formaldehyde fixed and a heat mediated antigen retrieval step in citrate buffer was performed. The sample was then blocked and incubated with the antibody for 1.5 hours at 22 degree C. An HRP conjugated goat anti-rabbit antibody was used as the secondary.



IHC (Immunohistochemistry)
AAA321480 at 1/100 staining human breast carcinoma tissues sections by IHC-P. The tissue was formaldehyde fixed and a heat mediated antigen retrieval step in citrate buffer was performed. The tissue was then blocked and incubated with the antibody for 1.5 ho



WB (Western Blot)
Western blot analysis of IR phosphorylation expression in Heatshock treated 293 whole cell lysates, The lane on the left is treated with the antigen-specific peptide.

Related Product Information for anti-IR antibody

Description: After removal of the precursor signal peptide, the insulin receptor precursor is post-translationally cleaved into two chains (alpha and beta) that are covalently linked. Binding of insulin to the insulin receptor (INSR) stimulates glucose uptake.

Function: Receptor tyrosine kinase which mediates the pleiotropic actions of insulin. Binding of insulin leads to phosphorylation of several intracellular substrates, including, insulin receptor substrates (IRS1, 2, 3, 4), SHC, GAB1, CBL and other signaling intermediates. Each of these phosphorylated proteins serve as docking proteins for other signaling proteins that contain Src-homology-2 domains (SH2 domain) that specifically recognize different phosphotyrosine residues, including the p85 regulatory subunit of PI3K and SHP2. Phosphorylation of IRSs proteins lead to the activation of two main signaling pathways: the PI3K-AKT/PKB pathway, which is responsible for most of the metabolic actions of insulin, and the Ras-MAPK pathway, which regulates expression of some genes and cooperates with the PI3K pathway to control cell growth and differentiation. Binding of the SH2 domains of PI3K to phosphotyrosines on IRS1 leads to the activation of PI3K and the generation of phosphatidylinositol-(3, 4, 5)-triphosphate (PIP3), a lipid second messenger, which activates several PIP3-dependent serine/threonine kinases, such as PDK1 and subsequently AKT/PKB. The net effect of this pathway is to produce a translocation of the glucose transporter SLC2A4/GLUT4 from cytoplasmic vesicles to the cell membrane to facilitate glucose transport. Moreover, upon insulin stimulation, activated AKT/PKB is responsible for: anti-apoptotic effect of insulin by inducing phosphorylation of BAD; regulates the expression of gluconeogenic and lipogenic enzymes by controlling the activity of the winged helix or forkhead (FOX) class of transcription factors. Another pathway regulated by PI3K-AKT/PKB activation is mTORC1 signaling pathway which regulates cell growth and metabolism and integrates signals from insulin. AKT mediates insulin-stimulated protein synthesis by phosphorylating TSC2 thereby activating mTORC1 pathway. The Ras/RAF/MAP2K/MAPK pathway is mainly involved in mediating cell growth, survival and cellular differentiation of insulin. Phosphorylated IRS1 recruits GRB2/SOS complex, which triggers the activation of the Ras/RAF/MAP2K/MAPK pathway. In addition to binding insulin, the insulin receptor can bind insulin-like growth factors (IGFI and IGFI). Isoform Short has a higher affinity for IGFI binding. 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.

Subunit Structure: Tetramer of 2 alpha and 2 beta chains linked by disulfide bonds. The alpha chains carry the insulin-binding regions, while the beta chains carry the kinase domain. 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. Interacts with SORBS1 but dissociates from it following insulin stimulation. Binds SH2B2. Activated form of INSR interacts (via Tyr-999) with the PTB/PID domains of IRS1 and SHC1. The sequences surrounding the phosphorylated NPXY motif contribute differentially to either IRS1 or SHC1 recognition. Interacts (via tyrosines in the C-terminus) with IRS2 (via PTB domain and 591-786 AA); the 591-786 would be the primary anchor of IRS2 to INSR while the PTB domain would have a stabilizing action on the interaction with INSR. Interacts with the SH2 domains of the 85 kDa regulatory subunit of PI3K (PIK3R1) in vitro, when autophosphorylated on tyrosine residues. Interacts with SOCS7. Interacts (via the phosphorylated Tyr-999), with SOCS3. Interacts (via the phosphorylated Tyr-1185, Tyr-1189, Tyr-1190) with SOCS1. Interacts with CAV2 (tyrosine-phosphorylated form); the interaction is increased with 'Tyr-27' phosphorylation of CAV2 (By similarity). Interacts with ARRB2 (By similarity). Interacts with GRB10; this interaction blocks the association between IRS1/IRS2 and INSR, significantly reduces insulin-stimulated tyrosine phosphorylation of IRS1 and IRS2 and thus decreases insulin signaling. Interacts with GRB7. Interacts with PDK1. Interacts (via Tyr-1190) with GRB14 (via BPS domain); this interaction protects the tyrosines in the activation loop from dephosphorylation, but promotes dephosphorylation of Tyr-999, this results in decreased interaction with, and phosphorylation of, IRS1. Interacts (via subunit alpha) with ENPP1 (via 485-599 AA); this interaction blocks autophosphorylation. Interacts with PTPRE; this interaction is dependent of Tyr-1185, Tyr-1189 and Tyr-1190 of the INSR. Interacts with STAT5B (via SH2 domain). Interacts with PTPRF. Interacts with ATIC; ATIC together with PRKAA2/AMPK2 and HACD3/PTPLAD1 is proposed to be part of a signaling network regulating INSR autophosphorylation and endocytosis (By similarity). Interacts with the cone snail venom insulin Con-Ins G1 (PubMed:27617429).

Post-translational Modifications: 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. Autophosphorylated on tyrosine residues in response to insulin. Phosphorylation of Tyr-999 is required for binding to IRS1, SHC1 and STAT5B. Dephosphorylated by PTPRE at Tyr-999, Tyr-1185, Tyr-1189 and Tyr-1190. Dephosphorylated by PTPRF and PTPN1. Dephosphorylated by PTPN2; down-regulates insulin-induced signaling.

Similarity: The tetrameric insulin receptor binds insulin via non-identical regions from two alpha chains, primarily via the C-terminal region of the first INSR alpha chain. Residues from the leucine-rich N-terminus of the other INSR alpha chain also contribute to this insulin binding site. A secondary insulin-binding site is formed by residues at the junction of fibronectin type-III domain 1 and 2. Belongs to the protein kinase superfamily. Tyr protein kinase family. Insulin receptor subfamily.

References	Geng S, Zhu W, Wang S, Xie C, Li X, Wu J, Li Y, Chen Y, Wang X, Meng Y, Zhang Q, Chen J, Zhong C; Journal: Biochem Biophys Res Commun. P53 modulates hepatic insulin sensitivity through NF-kB and p38/ERK MAPK pathways. Geng S, Wang S, Zhu W, Xie C, Li X, Wu J, Zhu J, Jiang Y, Yang X, Li Y, Chen Y, Wang X, Meng Y, Zhong C; Journal: Biomed Pharmacother. Curcumin suppresses JNK pathway to attenuate BPA-induced insulin resistance in LO2 cells. Geng S, Wang S, Zhu W, Xie C, Li X, Wu J, Zhu J, Jiang Y, Yang X, Li Y, Chen Y, Wang X, Meng Y, Zhu M, Wu R, Huang C, Zhong C; Journal: Toxicol Lett. Curcumin attenuates BPA-induced insulin resistance in HepG2 cells through suppression of JNK/p38 pathways.
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NCBI AND UNIPROT INFORMATION

NCBI GI #	119395736
NCBI GeneID	3643
NCBI Accession #	NP_000199.2
NCBI GenBank Nucleotide #	NM_000208.3
UniProt Accession #	P06213; Q17RW0; Q59H98; Q9UCB7; Q9UCB8; Q9UCB9
Molecular Weight	Observed: 95 kDa Predicted: 157 kDa
NCBI Official Full Name	insulin receptor isoform Long preproprotein
NCBI Official Synonym Full Names	insulin receptor
NCBI Official Symbol	INSR
NCBI Official Synonym Symbols	HHF5; CD220
NCBI Protein Information	insulin receptor
UniProt Protein Name	Insulin receptor
UniProt Gene Name	INSR
UniProt Synonym Gene Names	IR

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