

Rabbit Phospho-INSR (Y1185) Polyclonal Antibody | anti-INSR antibody

Phospho-INSR (Y1185) Antibody

CATALOG #	HOST	REACTIVITY	APPLICATIONS
AAA285280	Rabbit	Human (Predicted Reactivity: Bovine, Mouse, Rat, Xenopus, Drosophila)	ELISA, Dot Blot

PRODUCT SYNONYMS

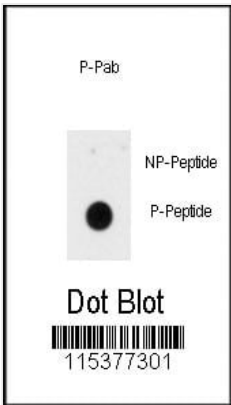
Phospho-INSR (Y1185); Polyclonal Antibody; Phospho-INSR (Y1185) Antibody; Insulin receptor; IR; CD220; Insulin receptor subunit alpha; Insulin receptor subunit beta; INSR; anti-INSR antibody

PRODUCT SPECIFICATIONS

Host	Rabbit
Reactivity	Human (Predicted Reactivity: Bovine, Mouse, Rat, Xenopus, Drosophila)
Clonality	Polyclonal
Isotype	Rabbit Ig
Specificity	This INSR Antibody is generated from rabbits immunized with a KLH conjugated synthetic phosphopeptide corresponding to amino acid residues surrounding Y1185 of human INSR.
Purity/Purification	Peptide Affinity Purified Rabbit Polyclonal Antibody (Pab)
Form/Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.
Sequence Length	1,370
Applicable Applications for anti-INSR antibody	ELISA, DB (Dot Blot)

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 phosphotyrosines 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: antiapoptotic 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 IGFII). Isoform Short has a higher affinity for IGFII binding. When present in a hybrid receptor with IGF1R, binds IGF1. PubMed: 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: 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.
Cellular Location	Cell membrane; Single-pass type I membrane protein
Tissue Location	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. IsoformShort is preferentially expressed in fetal cellssuch as fetal fibroblasts, muscle, liver andkidney. Found as a hybrid receptor withIGF1R in muscle, heart, kidney, adiposetissue, skeletal muscle, hepatoma,fibroblasts, spleen and placenta (at proteinlevel). Overexpressed in several tumors,including breast, colon, lung, ovary, and thyroid carcinomas.
Preparation and Storage	Maintain refrigerated at 2-8 degree C for up to 2 weeks. For long term storage store at -20 degree C in small aliquots to prevent freeze-thaw cycles.

IMAGES



DB (Dot Blot)
 Dot blot analysis of anti-Phospho-INSR-pY1185 Antibody on nitrocellulose membrane. 50ng of Phospho-peptide or Non Phospho-peptide per dot were adsorbed. Antibody working concentrations are 0.5ug per ml.

ADDITIONAL INFORMATION

Related Product Information for anti-INSR antibody	Binding of insulin to the insulin receptor (INSR) stimulates glucose uptake, thereby mediating the metabolic functions of insulin. Binding to insulin stimulates association of the receptor with downstream mediators including IRS1 and phosphatidylinositol 3'-kinase (PI3K). This protein can activate PI3K either directly by binding to the p85 regulatory subunit, or indirectly via IRS1. 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.
Product Categories/Family for anti-INSR antibody	Cancer; Cardiovascular; Metabolism; Neuroscience; Signal Transduction; Phospho Antibodies
References	Nakamaru, K., et al., Biochem. Biophys. Res. Commun. 328(2):449-454 (2005). Diaz, E., et al., Clin. Biochem. 38(3):243-247 (2005). McGettrick, A.J., et al., J. Biol. Chem. 280(8):6441-6446 (2005). Schmitt, T.L., et al., J. Biol. Chem. 280(5):3795-3801 (2005). Denley, A., et al., Mol. Endocrinol. 18(10):2502-2512 (2004).

NCBI AND UNIPROT INFORMATION

NCBI GI #	119395736
NCBI GeneID	3643
NCBI Accession #	P06213; NP_001073285.1
NCBI GenBank Nucleotide #	NM_000208.2
UniProt Accession #	Q9PVZ4; Q17RW0; Q59H98; Q9UCB7; Q9UCB8; Q9UCB9; P06213
Molecular Weight	156333
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
UniProt Entry Name	INSR_HUMAN

For Research Use Only. Not for use in diagnostic or therapeutic procedures. Small volumes of product may become entrapped in the seal of the vial during shipment and storage; if necessary, briefly centrifuge the vial on a tabletop centrifuge to dislodge any liquid in the cap.

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Online datasheet: <https://stage.aaabiotech.com/polyclonal/phospho-insr-y1185-antibody-dot-blot-elisa/285280>