IR (Phospho-Tyr1361) Antibody

Catalog No: #12015

Package Size: #12015-1 50ul #12015-2 100ul

Orders: order@signalwayantibody.com
Support: tech@signalwayantibody.com

Description

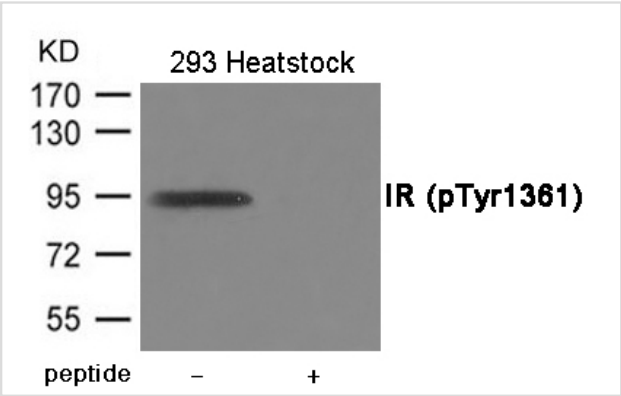
Product Name	IR (Phospho-Tyr1361) Antibody
Host Species	Rabbit
Clonality	Polyclonal
Purification	Antibodies were produced by immunizing rabbits with synthetic phosphopeptide and KLH conjugates. Antibodies were purified by affinity-chromatography using epitope-specific phosphopeptide. Non-phospho specific antibodies were removed by chromatogramphy using non-phosphopeptide.
Applications	WB
Species Reactivity	Human;Mouse;Rat
Specificity	The antibody detects endogenous level of IR only when phosphorylated at Tyrosine 1361.
Immunogen Type	Peptide-KLH
Immunogen Description	Peptide sequence around phosphorylation site of Tyrosine 1361 (I-P-Y(p)-T-H) derived from Human IR.
Conjugates	Unconjugated
Target Name	IR
Modification	Phospho
Other Names	HHF5, CD220
Accession No.	Swiss-Prot#: P06213; NCBI Gene#: 3643; NCBI Protein#: NP_000199.2
SDS-PAGE MW	95kd
Concentration	1.0mg/ml
Formulation	Supplied at 1.0mg/mL in phosphate buffered saline (without Mg2+ and Ca2+), pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol.
Storage	Store at -20°C/1 year

Application Details

Predicted MW: 95kd

Western blotting: 1:500~1:1000

Images



Western blot analysis of extracts from 293 cells treated with Heatstock using IR (Phospho-Tyr1361) Antibody #12015.The lane on the right is treated with the antigen-specific peptide.

Background

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 PDPK1 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 IGFII). Isoform Short has a higher affinity for IGFII binding. When present in a hybrid receptor with IGF1R, binds IGF1. Ref.40 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, Ref.46 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.

Note: This product is for in vitro research use only and is not intended for use in humans or animals.