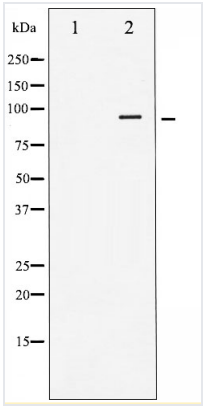
IR Antibody

PRODUCT OVERVIEW

Product Name	IR, Antibody
Full Product Name	Phospho-IR (Tyr1355) Antibody
Product Gene Name	anti-IR antibody
Product Synonym Names	CD220 antigen; EC 2.7.10.1; IR; insulin receptor; kinase InsR
Species Reactivity	Human
Form/Format	In phosphate buffered saline, pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol.
Purity/Purification	Immunogen affinity purified
Specificity	Phospho-IR (Tyr1355) Antibody detects endogenous levels of IR only when phosphorylated at Tyrosine 1355
Preparation and Storage	Store at-20 degree C for 1 year
Molecular Weight	95kDa
OMIM	125853
3D Structure	ModBase 3D Structure for P06213
Immunogen	A synthesized peptide derived from human IR around the phosphorylation site of Tyrosine 1355
ISO Certification	Manufactured in an ISO 9001:2015 Certified Laboratory.
Applications Tested/Suitable for anti-IR antibody	Western Blot (WB), Immunohistochemistry (IHC)
Application Notes for anti-IR antibody	WB: 1:500-1:2000 IHC: 1:50-1:200
Related Product Information for anti-IR antibody	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.
Other Notes	Small volumes of anti-IR antibody vial(s) may occasionally become entrapped in the seal of the product vial during shipment and storage. If necessary, briefly centrifuge the vial on a tabletop centrifuge to dislodge any liquid in the container`s cap. Certain products may require to ship with dry ice and additional dry ice fee may apply.
Searchable Terms for IR purchase	MBS8511282 is an antibody targeting IR. Also known as Phospho-IR (Tyr1355) Antibody. Reactive in Human.



Western Blot (WB) of anti-IR antibody
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.

ADDITIONAL DETAILS

NCBI/Uniprot data below describe general gene information for IR. It may not necessarily be applicable to this product.

NCBI GI #	119395736
NCBI GeneID	3643
NCBI Accession #	NP_000199.2
UniProt Primary Accession #	P06213
UniProt Secondary Accession #	Q17RW0; Q59H98; Q9UCB7; Q9UCB8; Q9UCB9
UniProt Related Accession #	P06213
GenBank Nucleotide #	NM_000208.3
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
UniProt Protein Name	Insulin receptor
UniProt Synonym Protein Names	CD_antigen: CD220
Protein Family	Irditoxin
UniProt Gene Name	INSR
UniProt Synonym Gene Names	IR
NCBI Protein Information	insulin receptor
NCBI Summary for IR	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]

UniProt Comments for IR	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 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. 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.
Research Articles on IR	**1.** Findings demonstrate that, in human breast cancer cells, DDR1 regulates IR expression and ligand dependent biological actions. This novel functional crosstalk is likely clinically relevant.
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