Product Datasheet

INSR (Ab-1375) Conjugated Antibody

Catalog No: #C33230



 Package Size:
 #C33230-AF350 100ul
 #C33230-AF405 100ul
 #C33230-AF488 100ul

 #C33230-AF555 100ul
 #C33230-AF594 100ul
 #C33230-AF647 100ul

 #C33230-AF680 100ul
 #C33230-AF750 100ul
 #C33230-Biotin 100ul

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

Description

Product Name	INSR (Ab-1375) Conjugated Antibody
Host Species	Rabbit
Clonality	Polyclonal
Species Reactivity	Hu
Specificity	The antibody detects endogenous levels of total INSR protein.
Immunogen Description	Synthesized non-phosphopeptide derived from human Stathmin around the phosphorylation site of threonine
	1375 (I-L-T(p)-L-P).
Conjugates	Biotin AF350 AF405 AF488 AF555 AF594 AF647 AF680 AF750
Other Names	CD220 antigen;EC 2.7.10.1;insulin receptor;IR;kinase InsR
Accession No.	Swiss-Prot#:P06213NCBI Gene ID:3643
Uniprot	P06213
GeneID	3643;
Excitation Emission	AF350: 346nm/442nm
	AF405: 401nm/421nm
	AF488: 493nm/519nm
	AF555: 555nm/565nm
	AF594: 591nm/614nm
	AF647: 651nm/667nm
	AF680: 679nm/702nm
	AF750: 749nm/775nm
Calculated MW	130
Formulation	0.01M Sodium Phosphate, 0.25M NaCl, pH 7.6, 5mg/ml Bovine Serum Albumin, 0.02% Sodium Azide
Storage	Store at 4°C in dark for 6 months

Application Details

Suggested Dilution:
AF350 conjugated: most applications: 1: 50 - 1: 250
AF405 conjugated: most applications: 1: 50 - 1: 250
AF488 conjugated: most applications: 1: 50 - 1: 250
AF555 conjugated: most applications: 1: 50 - 1: 250
AF594 conjugated: most applications: 1: 50 - 1: 250
AF647 conjugated: most applications: 1: 50 - 1: 250
AF680 conjugated: most applications: 1: 50 - 1: 250
AF750 conjugated: most applications: 1: 50 - 1: 250

Product Description

The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.

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. 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, 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