SunLong Biotech Co.,LTD
Tel: 0086-571-56623320 Fax:0086-571-56623318
E-mail:sales@sunlongbiotech.com
www.sunlongbiotech.com

Rabbit Anti-Insulin Receptor Beta antibody

SL0290R

Product Name	Insulin Receptor Beta
Chinese Name	胰岛素受体β抗体
Alias	CD 220; CD220; CD220 antigen; HHF 5; HHF5; HIR B; INSR; INSR; Insulin receptor; Insulin re beta; IR; INSR_HUMAN.
Research Area	Neurobiology Signal transduction Apoptosis Kinases and Phosphatases Diabetes Endocrinopath
Immunogen Species	Rabbit
Clonality	Polyclonal
React Species	Human, (predicted: Mouse, Rat,)
Applications	WB=1:500-2000,IHC-P=1:100-500,IHC-F=1:100-500,ICC/IF=1:100-500,IF=1:100-500,Flow-C （Paraffin sections need antigen repair） not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Theoretical molecular weight	68/152kDa
Cellular localization	The cell membrane
Form	Liquid
Concentration	1mg/ml
immunogen	KLH conjugated synthetic peptide derived from human Insulin Receptor Beta: 1001-1100/1382
Lsotype	IgG
Purification	affinity purified by Protein A
Buffer Solution	1M TBS(pH7.4) with 1% BSA, 3% Proclin300 and 50% Glycerol.
Storage	Shipped at 4℃. Store at -20 °C for one year. Avoid repeated freeze/thaw cycles.
Attention	This product as supplied is intended for research use only, not for use in human, therapeutic or d applications.
PubMed	PubMed
Product	The human insulin receptor is a heterotetrameric membrane glycoprotein consisting of disulfide l

Detail

in a beta-alpha-alpha-beta configuration. The beta subunit (95 kDa) possesses a single transmem
whereas the alpha subunit (135 kDa) is completely extracellular. The insulin receptor exhibits re
kinase (RTK) activity. RTKs are single pass transmembrane receptors that possess intrinsic cyto
enzymatic activity, catalyzing the transfer of the gamma phosphate of ATP to tyrosine residues i
substrates. RTKs are essential components of signal transduction pathways that affect cell prolif
differentiation, migration and metabolism.
Included in this large protein family are the insulin receptor and the receptors for growth factors
epidermal growth factor, fibroblast growth factor and vascular endothelial growth factor. Recept
occurs through ligand binding, which facilitates receptor dimerization and autophosphorylation
tyrosine residues in the cytoplasmic portion. The interaction of insulin with the alpha subunit of
receptor activates the protein tyrosine kinase of the beta subunit, which then undergoes an autop
that increases its tyrosine kinase activity. Three adapter proteins, IRS1, IRS2 and Shc, become p
on tyrosine residues following insulin receptor activation. These three phosphorylated proteins t
with SH2 domain containing signaling proteins.

Function:
Receptor tyrosine kinase which mediates the pleiotropic actions of insulin. Binding of insulin lea
phosphorylation of several intracellular substrates, including, insulin receptor substrates (IRS1, 2
GAB1, CBL and other signaling intermediates. Each of these phosphorylated proteins serve as do
for other signaling proteins that contain Src-homology-2 domains (SH2 domain) that specifically
different phosphotyrosines residues, including the p85 regulatory subunit of PI3K and SHP2. Ph
of IRSs proteins lead to the activation of two main signaling pathways: the PI3K-AKT/PKB path
responsible for most of the metabolic actions of insulin, and the Ras-MAPK pathway, which reg
expression of some genes and cooperates with the PI3K pathway to control cell growth and diffe
Binding of the SH2 domains of PI3K to phosphotyrosines on IRS1 leads to the activation of PI3
generation of phosphatidylinositol-(3, 4, 5)-triphosphate (PIP3), a lipid second messenger, which
several PIP3-dependent serine/threonine kinases, such as PDPK1 and subsequently AKT/PKB. T
this pathway is to produce a translocation of the glucose transporter SLC2A4/GLUT4 from cytopl
to the cell membrane to facilitate glucose transport. Moreover, upon insulin stimulation, activate
responsible for: anti-apoptotic effect of insulin by inducing phosphorylation of BAD; regulates the
gluconeogenic and lipogenic enzymes by controlling the activity of the winged helix or forkhead
transcription factors. Another pathway regulated by PI3K-AKT/PKB activation is mTORC1 sign
which regulates cell growth and metabolism and integrates signals from insulin. AKT mediates
insulin-stimulated protein synthesis by phosphorylating TSC2 thereby activating mTORC1 pathw
Ras/RAF/MAP2K/MAPK pathway is mainly involved in mediating cell growth, survival and ce
differentiation of insulin. Phosphorylated IRS1 recruits GRB2/SOS complex, which triggers the
the Ras/RAF/MAP2K/MAPK pathway. In addition to binding insulin, the insulin receptor can bi
growth factors (IGFI and IGFII). Isoform Short has a higher affinity for IGFII binding. When pres
receptor with IGF1R, binds IGF1. PubMed:12138094 shows that hybrid receptors composed of
INSR isoform Long are activated with a high affinity by IGF1, with low affinity by IGF2 and no
activated by insulin, and that hybrid receptors composed of IGF1R and INSR isoform Short are
IGF1, IGF2 and insulin. In contrast, PubMed:16831875 shows that hybrid receptors composed o
INSR isoform Long and hybrid receptors composed of IGF1R and INSR isoform Short have sim
characteristics, both bind IGF1 and have a low affinity for insulin.

Subunit:
Tetramer of 2 alpha and 2 beta chains linked by disulfide bonds. The alpha chains contribute to th the ligand-binding domain, while the beta chains carry the kinase domain. Forms a hybrid recepto the hybrid is a tetramer consisting of 1 alpha chain and 1 beta chain of INSR and 1 alpha chain ar of IGF1R. Interacts with SORBS1 but dissociates from it following insulin stimulation. Binds SH Activated form of INSR interacts (via Tyr-999) with the PTB/PID domains of IRS1 and SHC1. T surrounding the phosphorylated NPXY motif contribute differentially to either IRS1 or SHC1 re Interacts (via tyrosines in the C-terminus) with IRS2 (via PTB domain and 591-786 AA); the 591 the primary anchor of IRS2 to INSR while the PTB domain would have a stabilizing action on th with INSR. Interacts with the SH2 domains of the 85 kDa regulatory subunit of PI3K (PIK3R1) autophosphorylated on tyrosine residues. Interacts with SOCS7. Interacts (via the phosphorylate with SOCS3. Interacts (via the phosphorylated Tyr-1185, Tyr-1189, Tyr-1190) with SOCS1. Inte CAV2 (tyrosine-phosphorylated form); the interaction is increased with 'Tyr-27'phosphorylation similarity). Interacts with ARRB2 (By similarity). Interacts with GRB10; this interaction blocks t between IRS1/IRS2 and INSR, significantly reduces insulin-stimulated tyrosine phosphorylation IRS2 and thus decreases insulin signaling. Interacts with GRB7 (By similarity). Interacts with PD (via Tyr-1190) with GRB14 (via BPS domain); this interaction protects the tyrosines in the activa dephosphorylation, but promotes dephosphorylation of Tyr-999, this results in decreased interac phosphorylation of, IRS1. Interacts (via subunit alpha) with ENPP1 (via 485-599 AA); this inter autophosphorylation. Interacts with PTPRE; this interaction is dependent of Tyr-1185, Tyr-1189 of the INSR. Interacts with STAT5B (via SH2 domain). Interacts with PTPRF.

Subcellular Location:
Membrane; Single-pass type I membrane protein.

Tissue Specificity:
Isoform Long and isoform Short are predominantly expressed in tissue targets of insulin metaboli adipose tissue and skeletal muscle but are also expressed in the peripheral nerve, kidney, pulmon pancreatic acini, placenta vascular endothelium, fibroblasts, monocytes, granulocytes, erythrocyt Isoform Short is preferentially expressed in fetal cells such as fetal fibroblasts, muscle, liver and as a hybrid receptor with IGF1R in muscle, heart, kidney, adipose tissue, skeletal muscle, hepatoc spleen and placenta (at protein level). Overexpressed in several tumors, including breast, colon, lu thyroid carcinomas.

Post-translational modifications:
After being transported from the endoplasmic reticulum to the Golgi apparatus, the single glycos precursor is further glycosylated and then cleaved, followed by its transport to the plasma memb Autophosphorylated on tyrosine residues in response to insulin. Phosphorylation of Tyr-999 is re IRS1-, SHC1-, and STAT5B-binding. Dephosphorylated by PTPRE on Tyr-999, Tyr-1185, Tyr- Tyr-1190 residues. Dephosphorylated by PTPRF.

DISEASE:
Defects in INSR are the cause of Rabson-Mendenhall syndrome (RMS) [MIM:262190]; also kno

Mendenhall syndrome. RMS is a severe insulin resistance syndrome characterized by insulin-res
mellitus with pineal hyperplasia and somatic abnormalities. Typical features include coarse, seni
facies, dental and skin abnormalities, abdominal distension, and phallic enlargement. Inheritance
recessive.
Defects in INSR are the cause of leprechaunism (LEPRCH) [MIM:246200]; also known as Dono
Leprechaunism represents the most severe form of insulin resistance syndrome, characterized by
and postnatal growth retardation and death in early infancy. Inheritance is autosomal recessive.
Defects in INSR may be associated with noninsulin-dependent diabetes mellitus (NIDDM) [MIM
known as diabetes mellitus type 2.
Defects in INSR are the cause of familial hyperinsulinemic hypoglycemia type 5 (HHF5) [MIM:
Familial hyperinsulinemic hypoglycemia [MIM:256450], also referred to as congenital hyperins
nesidioblastosis, or persistent hyperinsulinemic hypoglycemia of infancy (PPHI), is the most cor
persistent hypoglycemia in infancy and is due to defective negative feedback regulation of insuli
low glucose levels.
Defects in INSR are the cause of insulin-resistant diabetes mellitus with acanthosis nigricans type
A) [MIM:610549]. This syndrome is characterized by the association of severe insulin resistance (
marked hyperinsulinemia and a failure to respond to exogenous insulin) with the skin lesion acar
nigricans and ovarian hyperandrogenism in adolescent female subjects. Women frequently prese
hirsutism, acne, amenorrhea or oligomenorrhea, and virilization. This syndrome is different from
has been demonstrated to be secondary to the presence of circulating autoantibodies against the in

Similarity:
Belongs to the protein kinase superfamily. Tyr protein kinase family. Insulin receptor subfamily.
Contains 3 fibronectin type-III domains.
Contains 1 protein kinase domain.

SWISS:
P06213

Gene ID:
3643

Database links:

Entrez Gene: 3643 Human

Entrez Gene: 16337 Mouse

Entrez Gene: 24954 Rat

Entrez Gene: 484990 Dog

Omim: 147670 Human

SwissProt: P06213 Human

SwissProt: P15208 Mouse

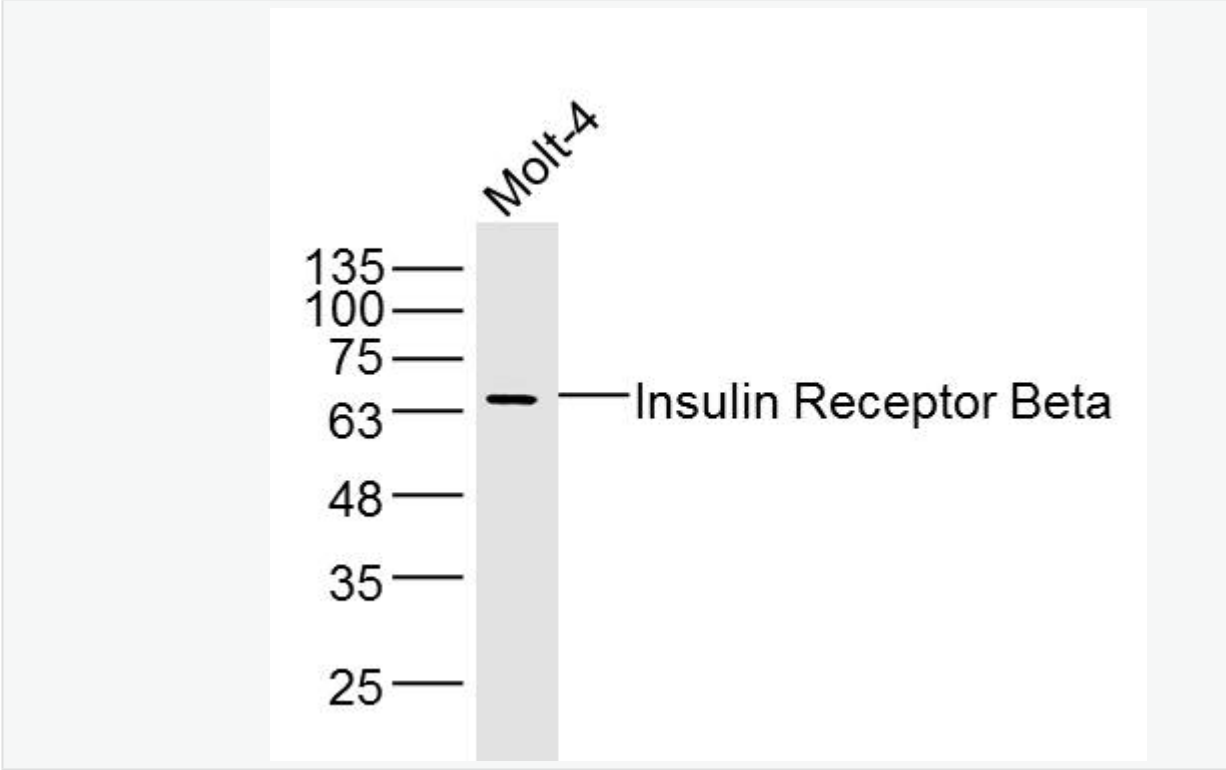
SwissProt: P15127 Rat

Unigene: 465744 Human

Unigene: 9876 Rat

胰岛素受体是一个四聚体，由两个 α 亚基和两个 β 亚基通过二硫键连接。两个 α 亚基位于外侧，其上有胰岛素的结合位点；两个 β 亚基是 Transmembrane protein，起 Signal transdu 无胰岛素结 合时，受体的酪氨酸蛋白激酶没有活性。当胰岛素与受体的 α 亚基结合并改变 构型后，酪氨酸蛋白激酶才被激活，激活后可催化两个反应： ①使四聚体复合物中 β 亚基 酪氨酸残基磷酸化,这种过程称为自我磷酸化（autophosphorylation)； ②将胰岛素受体底物 receptor substrate，IRSs)上具有重要作用的十几个酪氨酸残基磷酸化，磷酸化的 IRSs 能够 游效应物。

Product Picture



Sample:Molt-4 Cell (Human) Lysate at 40 ug

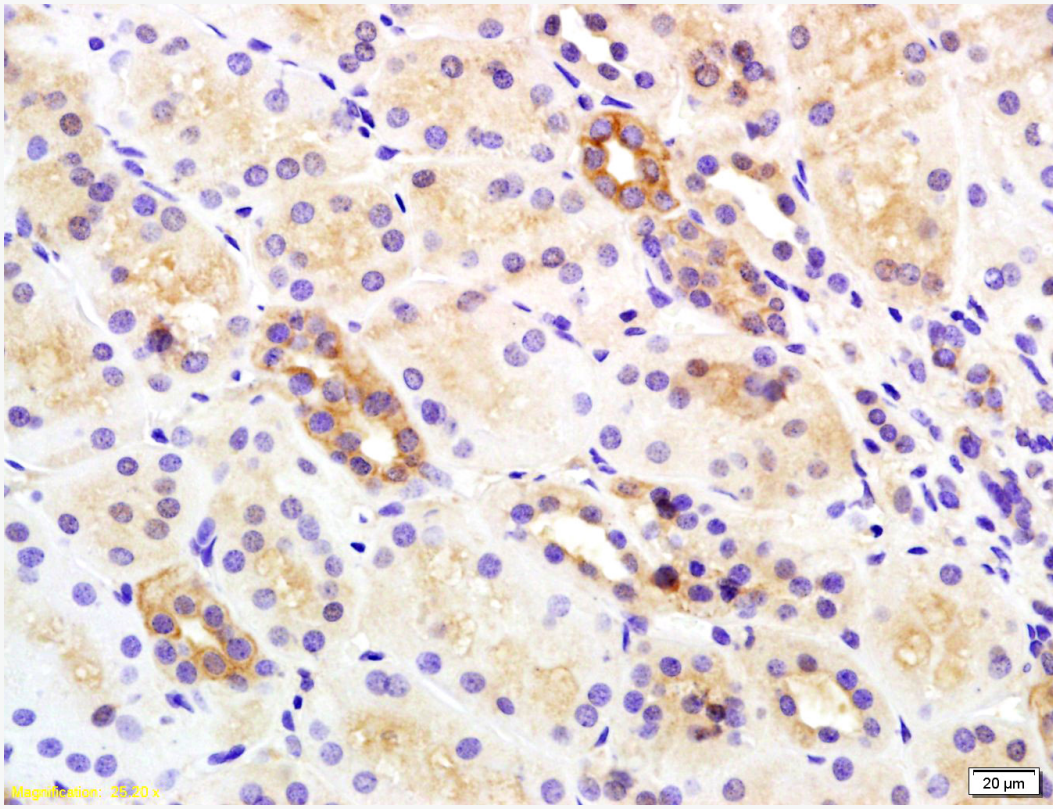
SunLong Biotech Co.,LTD
Tel: 0086-571-56623320 Fax:0086-571-56623318
E-mail:sales@sunlongbiotech.com
www.sunlongbiotech.com

Primary: Anti- Insulin Receptor Beta (SL0290R) at 1/300 dilution

Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000 dilution

Predicted band size: 68 kD

Observed band size: 68 kD



Tissue/cell: human kidney carcinoma; 4% Paraformaldehyde-fixed and paraffin-embedded;

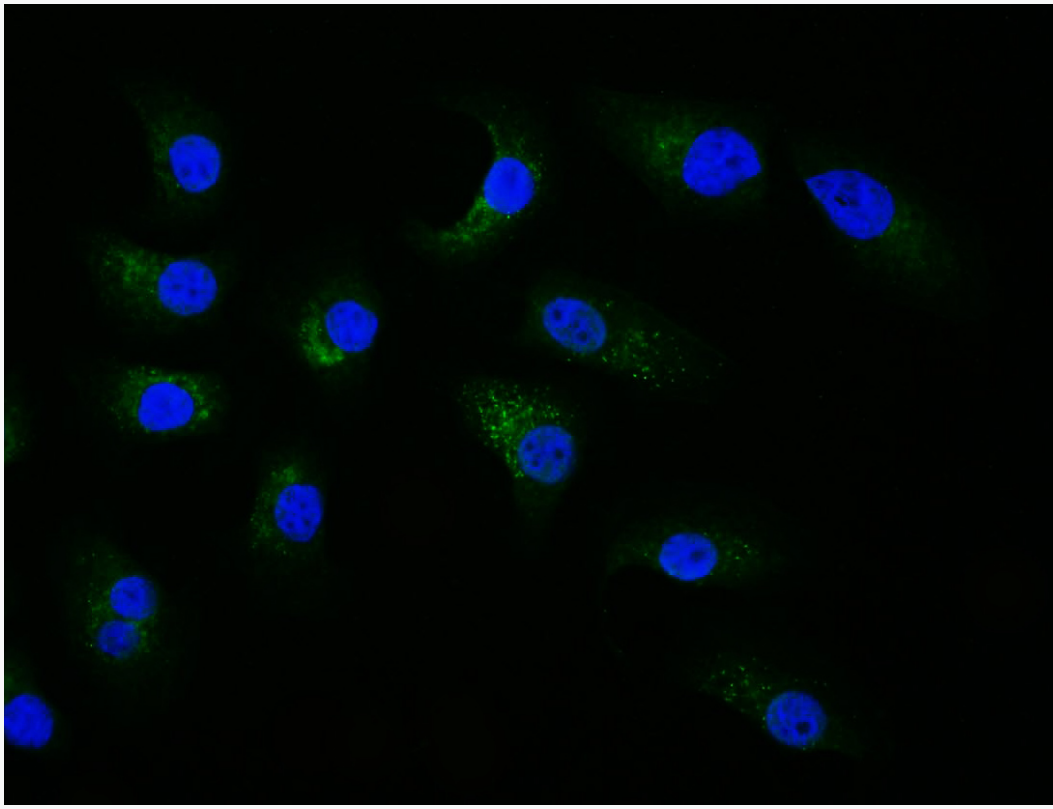
Antigen retrieval: citrate buffer (1M, pH 6.0), Boiling bathing for 15min; Block endogenous p

3% Hydrogen peroxide for 30min; Blocking buffer (normal goat serum,C-0005) at 37℃ for 2

Incubation: Anti-Insulin Receptor Beta Polyclonal Antibody, Unconjugated(SL0290R) 1:200,

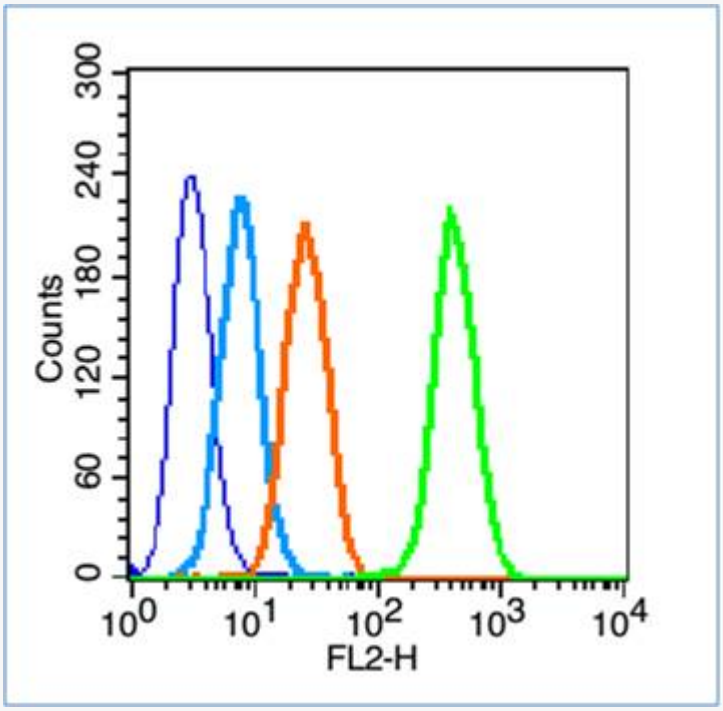
4°C, followed by conjugation to the secondary antibody(SP-0023) and DAB(C-0010) staining

SunLong Biotech Co.,LTD
Tel: 0086-571-56623320 Fax:0086-571-56623318
E-mail:sales@sunlongbiotech.com
www.sunlongbiotech.com



HepG2 cell; 4% Paraformaldehyde-fixed; Triton X-100 at room temperature for 20 min; Bloc
(normal goat serum, C-0005) at 37°C for 20 min; Antibody incubation with (Insulin Receptor
polyclonal Antibody, Unconjugated (SL0290R) 1:100, 90 minutes at 37°C; followed by a con
Anti-Rabbit IgG antibody at 37°C for 90 minutes, DAPI (blue, C02-04002) was used to stain th

SunLong Biotech Co.,LTD
Tel: 0086-571-56623320 Fax:0086-571-56623318
E-mail:sales@sunlongbiotech.com
www.sunlongbiotech.com



Blank control (blue line): HL60(blue).

Primary Antibody (green line): Rabbit Anti-Insulin Receptor alpha antibody (SL0290R)

Dilution: 0.2µg /10^6 cells;

Isotype Control Antibody (orange line): Rabbit IgG .

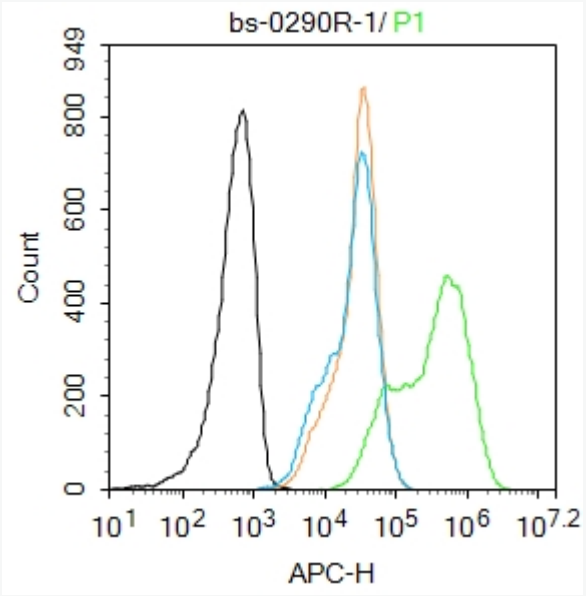
Secondary Antibody (white blue line): Goat anti-rabbit IgG-PE

Dilution: 1µg /test.

Protocol

The cells were fixed with 70% ethanol Overnight at 4°C. Cells stained with Primary Antibody room temperature. The cells were then incubated in 1 X PBS/2%BSA/10% goat serum to block protein-protein interactions followed by the antibody for 15 min at room temperature. The sec antibody used for 40 min at room temperature. Acquisition of 20,000 events was performed.

SunLong Biotech Co.,LTD
Tel: 0086-571-56623320 Fax:0086-571-56623318
E-mail:sales@sunlongbiotech.com
www.sunlongbiotech.com



Blank control:Molt4.

Primary Antibody (green line): Rabbit Anti-Insulin Receptor Beta antibody (SL0290R)

Dilution: 1µg /10^6 cells;

Isotype Control Antibody (orange line): Rabbit IgG .

Secondary Antibody : Goat anti-rabbit IgG-AF647

Dilution: 1µg /test.

Protocol

The cells were fixed with 4% PFA (10min at room temperature)and then permeabilized with 0.

20 min at room temperature. The cells were then incubated in 5%BSA to block non-specific pr

interactions for 30 min at room temperature .Cells stained with Primary Antibody for 30 min a

temperature. The secondary antibody used for 40 min at room temperature. Acquisition of 20,

was performed.