

Insulin Receptor R Antibody (C-term)
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP7654b

Specification

Insulin Receptor R Antibody (C-term) - Product Information

Application	WB, IHC-P,E
Primary Accession	P14616
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit Ig
Calculated MW	143720
Antigen Region	1256-1287

Insulin Receptor R Antibody (C-term) - Additional Information

Gene ID 3645

Other Names

Insulin receptor-related protein, IRR,
IR-related receptor, Insulin receptor-related
protein alpha chain, Insulin receptor-related
protein beta chain, INSRR, IRR

Target/Specificity

This Insulin Receptor R antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 1256-1287 amino acids from the C-terminal region of human Insulin Receptor R.

Dilution

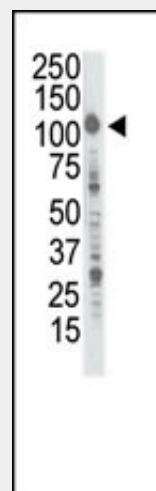
WB~~1:1000
IHC-P~~1:50~100

Format

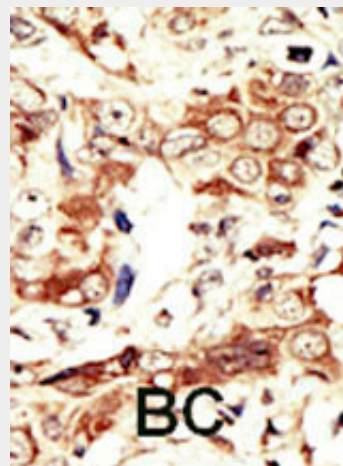
Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.



Western blot analysis of anti-InsRR Pab (Cat. #AP7654b) in A375 cell lysate. Lane A: preimmune, Lane B: purified antibody. InsRR (Arrow) was detected using purified Pab. Secondary HRP-anti-rabbit was used for signal visualization with chemiluminescence.



Formalin-fixed and paraffin-embedded human cancer tissue reacted with the primary antibody, which was peroxidase-conjugated to the secondary antibody, followed by AEC staining. This data demonstrates the use of this antibody for immunohistochemistry; clinical relevance has not been evaluated. BC = breast carcinoma; HC = hepatocarcinoma.

Precautions

Insulin Receptor R Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

Insulin Receptor R Antibody (C-term) - Protein Information

Name INSRR

Synonyms IRR

Function

Receptor with tyrosine-protein kinase activity. Functions as a pH sensing receptor which is activated by increased extracellular pH. Activates an intracellular signaling pathway that involves IRS1 and AKT1/PKB.

Cellular Location

Membrane; Single-pass type I membrane protein.

Insulin Receptor R Antibody (C-term) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

Insulin Receptor R Antibody (C-term) - Background

Protein kinases are enzymes that transfer a phosphate group from a phosphate donor, generally the γ phosphate of ATP, onto an acceptor amino acid in a substrate protein. By this basic mechanism, protein kinases mediate most of the signal transduction in eukaryotic cells, regulating cellular metabolism, transcription, cell cycle progression, cytoskeletal rearrangement and cell movement, apoptosis, and differentiation. With more than 500 gene products, the protein kinase family is one of the largest families of proteins in eukaryotes. The family has been classified in 8 major groups based on sequence comparison of their tyrosine (PTK) or serine/threonine (STK) kinase catalytic domains. The tyrosine kinase (TK) group is mainly involved in the regulation of cell-cell interactions such as differentiation, adhesion, motility and death. There are currently about 90 TK genes sequenced, 58 are of receptor protein TK (e.g. EGFR, EPH, FGFR, PDGFR, TRK, and VEGFR families), and 32 of cytosolic TK (e.g. ABL, FAK, JAK, and SRC families).

Insulin Receptor R Antibody (C-term) - References

Shier, P., et al., J. Biol. Chem. 264(25):14605-14608 (1989).
Whitmore, T.E., et al., Cytogenet. Cell Genet. 87 (1-2), 93-94 (1999).
Hanze, J., et al., Horm. Metab. Res. 31 (2-3), 77-79 (1999).
Shier, P., et al., Cytogenet. Cell Genet. 54 (1-2), 80-81 (1990).
Elmlinger, M.W., et al., Regul. Pept. 84 (1-3), 37-42 (1999).