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ProductInformation

Anti-phospho-Insulin Receptor/Insulin-Like Growth Factor-1 Receptor (pTyr$^{1162/1163}$)
produced in rabbit, affinity isolated antibody

Catalog Number **I2158**

Product Description

Anti-phospho-Insulin Receptor/Insulin-Like Growth Factor-1 Receptor (IR/IGF1R) (pTyr$^{1162/1163}$) was produced in rabbit using a synthetic phosphorylated peptide derived from the region of IR that contains tyrosines 1162 and 1163. The corresponding residues in the IGFR are tyrosines 1135 and 1136. The antibody is preadsorbed to remove any reactivity towards a non-phosphorylated IR/IGF1R. The final product is generated by affinity chromatography using an IR-derived peptide that is phosphorylated at tyrosines 1162 and 1163. Anti-phospho-IR/IGF1R (pTyr$^{1162/1163}$) specifically recognizes human (Gene ID: 25718) and rat (Gene ID: 3480) insulin receptor and insulin-like growth factor-1 receptor phosphorylated at tyrosines 1162 and 1163. Cross reactivity with mouse IR/IGF1R (100% homologous) has not been tested. It is used in immunoblotting [1] and immunocytochemistry applications.

The IR and IGF1R are heterotetrameric proteins consisting of two ligand-binding α subunits and two β subunits that each contains a tyrosine kinase domain. Biological actions of insulin and IGF1 are mediated by their respective cell surface receptors, both of which are receptor tyrosine kinases that regulate multiple signaling pathways through activation of a series of phosphorylation cascades. Insulin/IGF1 binding to the extracellular domain leads to autophosphorylation of the receptor and activation of intrinsic tyrosine kinase activity, which allows appropriate substrates to be phosphorylated. These two receptors differ in sequence in regions that confer specificity for the designated ligand as well as in certain intracellular signaling domains. These differences allow insulin and IGF1 to regulate different physiological functions through receptors that share a very similar structure. Phosphorylation sites that are unique to each receptor presumably play a key role in these signaling differences. The catalytic loops within the tyrosine kinase domains of the IR/IGF1R contain a three tyrosine motif corresponding to Tyr1158, 1162 and 1163 (for the IR) and Tyr1131, 1135 and 1136 (for the IGF1R). [2-4]

It is generally believed that autophosphorylation within the activation loop proceeds in a progressive manner initiating at the second tyrosine (1162 or 1135), followed by phosphorylation at the first tyrosine (1158 or 1131), then the last (1163 or 1136), upon which the IR or IGF1R becomes fully active.[4]

Reagent

Supplied as a solution in Dulbecco's phosphate buffered saline (without Mg^{2+} and Ca^{2+}), pH 7.3, with 50% glycerol, 1.0 mg/mL BSA (IgG, protease free) as a carrier and 0.05% sodium azide.

The amount of reagent is sufficient for 10 blots.

Precautions and Disclaimer

This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

Store at –20 °C. Upon initial thawing freeze the solution in working aliquots for extended storage. Avoid repeated freezing and thawing to prevent denaturing the antibody. Do not store in frost-free freezers. Working dilution samples should be discarded if not used within 12 hours. The antibody is stable for at least 6 months when stored appropriately.

Product Profile

Immunoblotting: a recommended working dilution of 1:1000 is determined using CHO-T cells transfected with a vector containing insulin receptor and stimulated with insulin.

Note: In order to obtain best results in different techniques and preparations we recommend determining optimal working concentration by titration test.

References

1. Pender, C., et. al., Regulation of insulin receptor function by a small molecule insulin activator. *J. Biol. Chem*., **277**, 43565-43571 (2002).
2. Bevan, P., Insulin signaling. *J. Cell. Sci*., **114**, 1429-1430 (2001).
3. Ottensmeyer, F. P., et. al., Mechanism of transmembrane signaling: insulin binding and the insulin receptor. *Biochemistry*, **39**, 12103-12112 (2000).
4. Motley, E. D., et al., Lysophosphatidylcholine inhibits insulin-induced akt activation through protein kinase C-alpha in vascular smooth muscle cells. *Hypertension*, **3**, 508-512 (2002).

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