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Commentary

Insulin Receptor Signaling Platform: A New Approach

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ABSTRACT

Insulin Receptor (IR) signaling is important for maintaining glucose homeostasis. Insulin resistance, type 2 diabetes, obesity, cancer, hypertension, and cardiovascular problems have all been linked to a malfunctioning and/or uncontrolled IR activation. The molecular mechanisms that mediate IR activation have become a popular topic of study in both science and medicine. The present state of knowledge about IR structure, function and signaling is summarized below, with a focus on the role of glycosylation and sialylation in IR activation.

Keywords: Insulin; G-protein-coupled receptor; Tyrosine kinase; Glycosylation; Matrix

DESCRIPTION

The Insulin Receptor (IR) is a transmembrane Receptor Tyrosine Kinase (RTK) complex with a high affinity that is required for maintaining body glucose homeostasis. Although the structure controlling RTK signal transduction is well established, the mechanism controlling IR activation is indeed not. The inhibition of receptor activation is thought to be dependent on glycosylation and sialylation regulation. Glycosylation of IR is an essential alteration that permits the receptor to be processed, hormonally regulated, and have binding activity. The precise factors that mediate IR activation, on the other hand, are less well understood. We give an overview of the structure and function of insulin receptors, as well as the role of glycosylation in receptor activation and cell signaling.

IR signaling

The IR is a preformed disulfide bond-linked dimer with two chains (and) produced from the same gene product in each promoter. A single molecule of the ligand insulin binds to the two extracellular domains of the receptor dimer in distinct ways. Insulin binding causes reciprocal phosphorylation of Tyrosine residues in intracellular domains, as well as interactions with a variety of intracellular proteins, some of which are Tyr-phosphorylated by the IR. The signaling pathways induced by insulin binding to its receptor have been thoroughly examined previously. In brief, the signal from IR activation is transmitted either through the Insulin Receptor Substrates (IRSs) family to Phosphatidylinositol-4,5-bisphosphate-3-kinase (PI3K), resulting in largely short-term metabolic effects in classical insulin target tissues, or through IRS and/or Src homology 2 domain containing family members to Extracellular Signal-Regulated Kinases (ERK), resulting in mainly mitotic effects in those tissues.

Both of these mechanisms are thought to be involved in insulin's synaptic actions. Insulin-induced increases in synaptic density may necessitate PI3K rather than ERK activity, whereas insulin-induced Ca2+ oscillation dampening necessitates ERK rather than PI3K activity. Real-time observations of insulin effect on synaptic IR at physiological concentrations could help to elucidate the significance of the many downstream pathways.

Several Alzheimer's preclinical researchers have concentrated on reversing downstream signaling problems in the IRS pathway, which is the one that is most heavily damaged in peripheral insulin resistance. Although the clinical benefit has not been linked to alterations in insulin signaling, the agonist that lowers IRS-1 abnormalities does show therapeutic promise in Alzheimer's disease patients. It's vital to remember that downstream insulin signaling in the brain differs from that in the periphery in key ways.

CONCLUSION

The GPCR signal integration in IR activation provides an overview of current insulin receptor structure including role of receptor glycosylation alterations and the major intermediates

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involved in insulin IR receptor molecular activation and subsequent cell signalling. Insulin-induced IR intimate experiences a negative imbalance of this unique IR-signaling platform, which may contribute to insulin resistance and type 2 diabetes.