1. Phylogeny  
   The insulin receptor (INSR; UniProt P06213) is a highly conserved receptor tyrosine kinase that belongs to the insulin/IGF receptor subfamily, sharing an evolutionary lineage with the insulin‐like growth factor 1 receptor (IGF‐1R) and the insulin receptor–related receptor (IRR) (belfiore2009insulinreceptorisoforms pages 1-2, sarfstein2015theinsrigf1rreceptor pages 1-4). Orthologs of INSR have been identified throughout metazoans, with conserved domain architectures present in invertebrates such as Drosophila as well as vertebrates, indicating that the ancestral receptor appeared early in evolution and has been maintained due to its central role in metabolic regulation (payankaulam2019transcriptionalregulationof pages 1-3, du2017acomprehensivesurvey pages 27-29). Sequence analyses and kinome classifications place INSR within the receptor tyrosine kinase superfamily, and the alternative splicing that gives rise to two major isoforms (IR-A and IR-B) appears to be a feature that evolved to allow tissue-specific control of both metabolic and growth‐promoting signals (belfiore2009insulinreceptorisoforms pages 1-2, galal2023insulinreceptorisoforms pages 23-24).
2. Reaction Catalyzed  
   INSR catalyzes the transfer of the γ-phosphate from ATP to specific tyrosine residues on itself (in an autophosphorylation reaction) and on intracellular substrates, primarily insulin receptor substrates (IRS1–IRS4) and other adaptor proteins, thereby generating phosphotyrosine sites that act as docking sites for downstream effectors (saltiel2011mechanismsofinsulin pages 14-17, belfiore2009insulinreceptorisoforms pages 5-6). In chemical terms, the receptor mediates the reaction:  
     ATP + [protein]-tyrosine → ADP + [protein]-phosphotyrosine + H⁺  
   where “protein” may be either the receptor itself (autophosphorylation) or specific substrate proteins (belfiore2009insulinreceptorisoforms pages 5-6, saltiel2011mechanismsofinsulin pages 39-41).
3. Cofactor Requirements  
   The catalytic activity of the insulin receptor requires ATP as a phosphate donor and is dependent on the presence of divalent cations such as Mg²⁺, which are essential cofactors for stabilizing the binding of ATP in its kinase domain (du2017acomprehensivesurvey pages 27-29, belfiore2009insulinreceptorisoforms pages 6-7).
4. Substrate Specificity  
   INSR exhibits substrate specificity toward polypeptides containing tyrosine residues embedded within specific sequence contexts that function as docking sites for SH2 and phosphotyrosine-binding (PTB) domain proteins. In particular, the receptor phosphorylates IRS proteins and other substrates (e.g., Shc, GAB1, CBL) at tyrosine residues that conform to its recognition requirements, thereby facilitating subsequent recruitment of signaling components such as the p85 regulatory subunit of PI3K and SHP2 (belfiore2009insulinreceptorisoforms pages 5-6, hubbard2013theinsulinreceptor pages 9-10).
5. Structure  
   INSR is synthesized as a single polypeptide chain that is proteolytically processed into two subunits and assembled into a disulfide-linked heterotetramer composed of two extracellular α-subunits and two transmembrane β-subunits (belfiore2009insulinreceptorisoforms pages 6-7). The extracellular portion of the α-subunits contains several distinct domains: two leucine-rich domains (L1 and L2) flanking a cysteine-rich (CR) region and three fibronectin type III (FnIII) domains; these regions mediate high-affinity ligand binding (belfiore2009insulinreceptorisoforms pages 5-6, galal2023insulinreceptorisoforms pages 2-5). The β-subunits comprise a short extracellular segment, a single-pass transmembrane helix, and a long intracellular domain that harbors the juxtamembrane region, the catalytic tyrosine kinase domain, and a C-terminal tail; within the kinase domain, key features include the activation loop, whose autophosphorylation stabilizes the active conformation, and other structural motifs such as the hydrophobic spine and C-helix that are crucial for kinase function (hubbard2013theinsulinreceptor pages 1-2, belfiore2009insulinreceptorisoforms pages 6-7). Unique structural characteristics include the folded-over (“Λ” or inverted V) conformation of the ectodomain that undergoes conformational rearrangements upon insulin binding to bring the kinase domains of the β-subunits into proximity, facilitating trans-autophosphorylation (belfiore2009insulinreceptorisoforms pages 5-6, hubbard2013theinsulinreceptor pages 9-10).
6. Regulation  
   INSR is regulated at multiple levels. At the post-transcriptional level, alternative splicing of exon 11 produces two isoforms, IR-A and IR-B, which differ by a 12–amino acid sequence; IR-A, which lacks exon 11, is more mitogenic and exhibits higher affinity for IGF-II, while IR-B is more metabolically active and predominantly expressed in tissues such as liver, muscle, and adipose tissue (belfiore2009insulinreceptorisoforms pages 1-2, galal2023insulinreceptorisoforms pages 6-7). At the post-translational level, insulin binding triggers conformational changes and rapid autophosphorylation of tyrosine residues within the activation loop, an event that is critical for full catalytic activation; subsequent phosphorylation of additional tyrosine residues in the juxtamembrane and C-terminal regions creates docking sites for downstream adaptor proteins (belfiore2009insulinreceptorisoforms pages 5-6, saltiel2011mechanismsofinsulin pages 39-41). In addition, serine phosphorylation, receptor ubiquitination, and interactions with inhibitory proteins (e.g., Grb10, Grb14) further modulate INSR activity and stability, while protein tyrosine phosphatases such as PTP1B dephosphorylate key phosphotyrosines to attenuate signaling (youngren2007regulationofinsulin pages 5-6, saltiel2011mechanismsofinsulin pages 14-17). Transcriptional regulation of the INSR gene is mediated by a complex interplay of cis-regulatory elements and transcription factors such as Sp1, FOXO, and p53, which govern tissue-specific expression and may be altered in disease states (payankaulam2019transcriptionalregulationof pages 1-3, payankaulam2019transcriptionalregulationof pages 16-18).
7. Function  
   INSR is a receptor tyrosine kinase that mediates the pleiotropic actions of insulin. Upon insulin binding, the receptor undergoes autophosphorylation and triggers two major cascades: the phosphoinositide 3-kinase (PI3K)-AKT/PKB pathway, which is responsible for most of insulin’s metabolic effects (including glucose uptake, glycogen synthesis, and lipid metabolism), and the Ras-MAPK pathway, which regulates gene expression and supports cell growth and differentiation (belfiore2009insulinreceptorisoforms pages 5-6, saltiel2011mechanismsofinsulin pages 14-17). Expression of INSR is broad, with IR-B predominantly found in classic insulin-responsive tissues such as liver, muscle, adipose tissue, and kidney, whereas IR-A is more common in fetal tissues, the brain, and certain cancers, reflecting its higher affinity for IGF-II and mitogenic properties (belfiore2009insulinreceptorisoforms pages 1-2, galal2023insulinreceptorisoforms pages 6-7). By phosphorylating its primary substrates, such as IRS proteins, Shc, and others, INSR orchestrates the assembly of multiprotein signaling complexes that lead to the activation of downstream kinases and the regulation of diverse physiological processes including cellular metabolism, growth, differentiation, and survival (belfiore2009insulinreceptorisoforms pages 5-6, hubbard2013theinsulinreceptor pages 9-10).
8. Other Comments  
   Alterations in INSR expression or function have been implicated in various pathological states; overexpression of the IR-A isoform and formation of hybrid receptors with IGF-1R are associated with cancer progression and chemoresistance in several tumor types, while aberrant INSR signaling is a central feature of insulin resistance and type 2 diabetes mellitus (belfiore2009insulinreceptorisoforms pages 26-27, galal2023insulinreceptorisoforms pages 27-28). In addition, several pharmacological approaches targeting INSR’s kinase activity or its downstream signaling pathways are under investigation to improve insulin sensitivity or counteract tumor growth (galal2023insulinreceptorisoforms pages 24-25, youngren2007regulationofinsulin pages 14-14). The receptor’s complex regulation by alternative splicing, post-translational modifications, and protein–protein interactions has made it an attractive target for therapeutic intervention in metabolic diseases as well as in oncology (belfiore2009insulinreceptorisoforms pages 3-5, saltiel2011mechanismsofinsulin pages 35-36).
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