1. Phylogeny  
   IGF1R is a member of the receptor tyrosine kinase family and belongs to the insulin receptor subfamily, which also includes the insulin receptor (IR) and the insulin‐like growth factor 2 receptor. Its amino acid sequence and overall domain organization are highly conserved across vertebrates, with orthologs present in mammals, birds, amphibians, and fish, indicative of an ancestral gene duplication event that gave rise to both IGF1R and IR (adams2000structureandfunction pages 1-2, baserga2000thecontradictionsof pages 1-2). IGF1R shares approximately 70% sequence homology with the insulin receptor, situating it within an evolutionary core set of receptors that emerged early in metazoan evolution, as supported by studies tracing receptor tyrosine kinases from simple multicellular organisms to vertebrates (adams2000structureandfunction pages 2-5, belfiore2009insulinreceptorisoforms pages 1-2). Evolutionary studies indicate that the insulin receptor family, including IGF1R, can be traced back to gene duplications that occurred prior to the divergence of vertebrates, with further diversification achieved through alternative splicing events and the evolution of receptor hybrids (belfiore2009insulinreceptorisoforms pages 25-26, adams2000structureandfunction pages 36-37).
2. Reaction Catalyzed  
   IGF1R functions as a receptor tyrosine kinase that catalyzes the transfer of the γ-phosphate group from adenosine triphosphate (ATP) to specific tyrosine residues on its substrate proteins. The chemical reaction can be formally written as:  
   ATP + [protein]-(L-tyrosine) → ADP + [protein]-(L-tyrosine)-phosphate + H⁺  
   Upon ligand binding, IGF1R undergoes autophosphorylation on its intracellular tyrosine residues, which then serves to phosphorylate downstream substrates such as the insulin receptor substrates (IRS1/2), Shc, and proteins from the 14-3-3 family, thereby propagating intracellular signaling cascades essential for cell growth and survival (adams2000structureandfunction pages 25-27, dupont2004insulinandinsulinlike pages 1-2).
3. Cofactor Requirements  
   The kinase activity of IGF1R is dependent on the presence of divalent cations, particularly magnesium ions (Mg²⁺), which are essential for ATP binding and proper catalytic function. This requirement for Mg²⁺ is a common feature among protein kinases and is critical for aligning the nucleotide substrate in the active site for efficient phosphoryl transfer (adams2000structureandfunction pages 1-2).
4. Substrate Specificity  
   IGF1R preferentially phosphorylates tyrosine residues located within specific linear motifs on its substrates. Although the precise consensus sequence for IGF1R substrates is not exhaustively defined in the available literature, the receptor is known to phosphorylate key tyrosine residues on insulin receptor substrates (IRS1/2), which contain docking sites for SH2 domain–containing regulatory proteins. The phosphorylation events create binding sites that facilitate the recruitment of downstream signaling molecules involved in the PI3K-AKT and Ras-MAPK pathways (adams2000structureandfunction pages 25-27, dupont2004insulinandinsulinlike pages 4-5). This substrate specificity is mediated in part by the structural organization of the IGF1R tyrosine kinase domain, which aligns its activation loop and catalytic residues to selectively transfer phosphate groups to target proteins that conform to its recognition pattern (li2009inhibitionofthe pages 4-5).
5. Structure  
   IGF1R is synthesized as a single-chain precursor of approximately 1367 amino acids that undergoes extensive post-translational modifications including glycosylation, proteolytic cleavage, and disulfide bond–mediated dimerization to form the mature heterotetramer consisting of two extracellular α-subunits and two transmembrane β-subunits. The extracellular region is organized into multiple domains: starting with an N-terminal leucine-rich (L1) domain, followed by a cysteine-rich region, an L2 domain, and then three fibronectin type III (FnIII) domains. The FnIII domains are arranged in series and play roles in ligand binding, receptor dimerization, and conformational regulation. The transmembrane domain spans residues approximately 906–929, anchoring the receptor in the plasma membrane, while the intracellular region contains the tyrosine kinase domain responsible for catalytic activity (adams2000structureandfunction pages 1-2, adams2000structureandfunction pages 5-7). Recent cryo-electron microscopy studies and crystallographic data have revealed that in its inactive state the extracellular portion adopts a Λ-shaped dimeric conformation stabilized by inter-protomer interactions between the L1 domain of one subunit and the FnIII-2 domain of the other. Ligand (IGF1) binding induces a dramatic conformational change to an asymmetric Γ-shaped active dimer, characterized by a reduction in the distance between the membrane-proximal FnIII-3 domains. This repositioning enables the intracellular kinase domains to come into close contact, facilitating trans-autophosphorylation on key tyrosine residues located in the activation loop, such as Tyr1131, Tyr1135, and Tyr1136, which are essential for full catalytic activation (adams2000structureandfunction pages 1-2, adams2000structureandfunction pages 35-36, li2019structuralbasisof pages 1-2, li2019structuralbasisof pages 4-5). Additional structural features include extensive N-linked glycosylation in the extracellular region, which is critical for proper folding, stability, and trafficking of the receptor, as well as disulfide bonds that provide structural integrity by linking the α- and β-subunits (adams2000structureandfunction pages 2-5, adams2000structureandfunction pages 34-35).
6. Regulation  
   Regulation of IGF1R is multifaceted and occurs at several post-translational levels. Ligand binding to the extracellular portion relieves autoinhibition, allowing the receptor to undergo autophosphorylation on specific tyrosine residues within its kinase domain. These phosphorylation events act as molecular switches that enhance the recruitment and phosphorylation of downstream substrates such as IRS proteins, which in turn activate the PI3K-AKT and Ras-MAPK signaling cascades (adams2000structureandfunction pages 25-27, pollak2008insulinandinsulinlike pages 10-11). The receptor is also regulated by glycosylation, which is essential for proper biosynthesis, membrane transport, and receptor dimerization; mutations that disrupt glycosylation sites can impair receptor expression and signaling (adams2000structureandfunction pages 2-5, adams2000structureandfunction pages 34-35). In addition, IGF1R exhibits negative cooperativity in ligand binding, a phenomenon attributed to the asymmetric binding of IGF1, where binding of a single IGF1 molecule to the receptor hinders subsequent binding of a second molecule, thus fine-tuning signaling intensity (li2019structuralbasisof pages 4-5). Receptor internalization and subsequent recycling or degradation further contribute to the regulation of its signaling output. Mutational studies, such as those involving the Arg59Ter mutation, have demonstrated that reduced receptor expression leads to diminished autophosphorylation and downstream signaling, underscoring the impact of gene dosage on IGF1R function (raile2006clinicalandfunctional pages 5-7, raile2006clinicalandfunctional pages 7-8). Finally, formation of hybrid receptors with the insulin receptor, particularly with the IR-A isoform, further modulates the receptor’s affinity for various ligands and its downstream signaling profile, contributing to tissue-specific and pathological activation patterns (belfiore2009insulinreceptorisoforms pages 25-26, belfiore2009insulinreceptorisoforms pages 37-38).
7. Function  
   IGF1R is a central mediator of insulin-like growth factor signaling and plays an essential role in regulating cellular growth, survival, and differentiation. Upon binding with its high-affinity ligand IGF1, as well as with IGF2 and insulin at lower affinities, IGF1R activates intracellular signaling pathways by inducing autophosphorylation and subsequent phosphorylation of adapter proteins such as IRS1/2 and Shc. These events lead to the activation of the PI3K-AKT pathway, which inhibits apoptosis and stimulates protein synthesis, and the Ras-MAPK pathway, which promotes cellular proliferation (adams2000structureandfunction pages 1-2, leroith2007mechanismsofdisease pages 1-2, dupont2004insulinandinsulinlike pages 1-2). IGF1R plays a crucial role in fetal growth and postnatal development; functional knockout studies in animal models have consistently demonstrated that loss of IGF1R leads to severe growth retardation and embryonic lethality, confirming its indispensable role in orchestrating normal development (laron2001insulinlikegrowthfactor pages 2-2, leroith2007mechanismsofdisease pages 6-8). In addition to its physiological roles, IGF1R signaling is implicated in oncogenesis through its mitogenic and anti-apoptotic effects. Overexpression or dysregulation of IGF1R is commonly observed in multiple tumor types, including breast, prostate, and ovarian cancers, where enhanced IGF1R signaling contributes to tumor transformation, survival, and resistance to chemo- and radiotherapy (amutha2017roleofinsulinlike pages 1-2, baserga2000thecontradictionsof pages 5-6, li2009inhibitionofthe pages 28-29). The receptor also participates in the formation of hybrid receptors with the insulin receptor; such hybrids often display altered ligand binding properties and contribute to the complexity of insulin/IGF signaling in both physiological and pathological contexts (belfiore2009insulinreceptorisoforms pages 25-26, belfiore2009insulinreceptorisoforms pages 37-38).
8. Other Comments  
   Several inhibitors targeting IGF1R have been developed, including small-molecule tyrosine kinase inhibitors and monoclonal antibodies that block ligand binding or receptor autophosphorylation. One notable example is picropodophyllin (PPP), a cyclolignan that selectively inhibits IGF1R phosphorylation without significantly affecting the insulin receptor, thereby representing a promising approach for cancer therapy (girnita2004cyclolignansasinhibitors pages 1-1, li2009inhibitionofthe pages 35-36). In addition to these experimental inhibitors, clinical trials have evaluated various IGF1R-targeted agents for their therapeutic potential in solid tumors and hematological malignancies (li2009inhibitionofthe pages 26-28). Disease associations of IGF1R include its role in oncogenesis, where dysregulated receptor signaling correlates with increased tumor cell proliferation, resistance to apoptosis, and enhanced metastatic potential. Rare mutations in IGF1R, such as the Arg59Ter mutation, have been linked to intrauterine and postnatal growth retardation, microcephaly, and developmental delays, further underscoring the receptor’s critical role in growth regulation and its sensitivity to gene dosage effects (raile2006clinicalandfunctional pages 5-7, raile2006clinicalandfunctional pages 7-8). Furthermore, the complex interplay between IGF1R and insulin receptor isoforms, including the formation of hybrid receptors, contributes to variations in ligand specificity and downstream signaling, which may influence both metabolic regulation and neoplastic transformation (belfiore2009insulinreceptorisoforms pages 29-31, singh2014insulinreceptor(ir) pages 1-2). Finally, extensive post-translational modifications such as glycosylation and disulfide bond formation are integral to IGF1R function and represent potential targets for therapeutic intervention in diseases characterized by aberrant receptor activity (adams2000structureandfunction pages 2-5, adams2000structureandfunction pages 34-35).
9. References
10. adams2000structureandfunction pages 1-2
11. adams2000structureandfunction pages 2-5
12. adams2000structureandfunction pages 5-7
13. adams2000structureandfunction pages 7-9
14. adams2000structureandfunction pages 9-10
15. adams2000structureandfunction pages 10-12
16. adams2000structureandfunction pages 21-22
17. adams2000structureandfunction pages 25-27
18. adams2000structureandfunction pages 32-33
19. adams2000structureandfunction pages 33-34
20. adams2000structureandfunction pages 34-35
21. adams2000structureandfunction pages 35-36
22. adams2000structureandfunction pages 36-37
23. adams2000structureandfunction pages 38-39
24. amutha2017roleofinsulinlike pages 1-2
25. baserga2000thecontradictionsof pages 1-2
26. baserga2000thecontradictionsof pages 4-5
27. baserga2000thecontradictionsof pages 5-6
28. belfiore2009insulinreceptorisoforms pages 1-2
29. belfiore2009insulinreceptorisoforms pages 25-26
30. belfiore2009insulinreceptorisoforms pages 27-28
31. belfiore2009insulinreceptorisoforms pages 28-29
32. belfiore2009insulinreceptorisoforms pages 29-31
33. belfiore2009insulinreceptorisoforms pages 36-37
34. bondy2004signalingbyinsulinlike pages 1-2
35. dupont2004insulinandinsulinlike pages 1-2
36. dupont2004insulinandinsulinlike pages 4-5
37. girnita2004cyclolignansasinhibitors pages 1-1
38. laron2001insulinlikegrowthfactor pages 2-2
39. leroith2007mechanismsofdisease pages 1-2
40. leroith2007mechanismsofdisease pages 6-8
41. li2009inhibitionofthe pages 1-2
42. li2009inhibitionofthe pages 2-4
43. li2009inhibitionofthe pages 4-5
44. li2009inhibitionofthe pages 5-6
45. li2009inhibitionofthe pages 6-7
46. li2009inhibitionofthe pages 7-9
47. li2009inhibitionofthe pages 8-9
48. li2009inhibitionofthe pages 9-10
49. li2009inhibitionofthe pages 21-22
50. li2009inhibitionofthe pages 22-24
51. li2009inhibitionofthe pages 26-28
52. li2009inhibitionofthe pages 28-29
53. li2009inhibitionofthe pages 35-36
54. li2019structuralbasisof pages 1-2
55. li2019structuralbasisof pages 4-5
56. pollak2008insulinandinsulinlike pages 2-3
57. pollak2008insulinandinsulinlike pages 3-4
58. pollak2008insulinandinsulinlike pages 8-9
59. pollak2008insulinandinsulinlike pages 10-11
60. raile2006clinicalandfunctional pages 5-7
61. raile2006clinicalandfunctional pages 7-8
62. raile2006clinicalandfunctional pages 8-8
63. singh2014insulinreceptor(ir) pages 1-2
64. tian2015quantitativeanalysisof pages 10-11
65. tsui2008evidenceforan pages 8-9

Each reference is cited exactly as provided by the context and supports the details presented in the respective sections.

References

1. (adams2000structureandfunction pages 1-2): T. E. Adams, V. C. Epa, T. P. J. Garrett, and C. W. Ward\*. Structure and function of the type 1 insulin-like growth factor receptor. Cellular and Molecular Life Sciences, 57:1050-1093, Jul 2000. URL: https://doi.org/10.1007/pl00000744, doi:10.1007/pl00000744. This article has 869 citations and is from a domain leading peer-reviewed journal.
2. (adams2000structureandfunction pages 2-5): T. E. Adams, V. C. Epa, T. P. J. Garrett, and C. W. Ward\*. Structure and function of the type 1 insulin-like growth factor receptor. Cellular and Molecular Life Sciences, 57:1050-1093, Jul 2000. URL: https://doi.org/10.1007/pl00000744, doi:10.1007/pl00000744. This article has 869 citations and is from a domain leading peer-reviewed journal.
3. (adams2000structureandfunction pages 21-22): T. E. Adams, V. C. Epa, T. P. J. Garrett, and C. W. Ward\*. Structure and function of the type 1 insulin-like growth factor receptor. Cellular and Molecular Life Sciences, 57:1050-1093, Jul 2000. URL: https://doi.org/10.1007/pl00000744, doi:10.1007/pl00000744. This article has 869 citations and is from a domain leading peer-reviewed journal.
4. (adams2000structureandfunction pages 25-27): T. E. Adams, V. C. Epa, T. P. J. Garrett, and C. W. Ward\*. Structure and function of the type 1 insulin-like growth factor receptor. Cellular and Molecular Life Sciences, 57:1050-1093, Jul 2000. URL: https://doi.org/10.1007/pl00000744, doi:10.1007/pl00000744. This article has 869 citations and is from a domain leading peer-reviewed journal.
5. (adams2000structureandfunction pages 32-33): T. E. Adams, V. C. Epa, T. P. J. Garrett, and C. W. Ward\*. Structure and function of the type 1 insulin-like growth factor receptor. Cellular and Molecular Life Sciences, 57:1050-1093, Jul 2000. URL: https://doi.org/10.1007/pl00000744, doi:10.1007/pl00000744. This article has 869 citations and is from a domain leading peer-reviewed journal.
6. (adams2000structureandfunction pages 33-34): T. E. Adams, V. C. Epa, T. P. J. Garrett, and C. W. Ward\*. Structure and function of the type 1 insulin-like growth factor receptor. Cellular and Molecular Life Sciences, 57:1050-1093, Jul 2000. URL: https://doi.org/10.1007/pl00000744, doi:10.1007/pl00000744. This article has 869 citations and is from a domain leading peer-reviewed journal.
7. (adams2000structureandfunction pages 34-35): T. E. Adams, V. C. Epa, T. P. J. Garrett, and C. W. Ward\*. Structure and function of the type 1 insulin-like growth factor receptor. Cellular and Molecular Life Sciences, 57:1050-1093, Jul 2000. URL: https://doi.org/10.1007/pl00000744, doi:10.1007/pl00000744. This article has 869 citations and is from a domain leading peer-reviewed journal.
8. (adams2000structureandfunction pages 35-36): T. E. Adams, V. C. Epa, T. P. J. Garrett, and C. W. Ward\*. Structure and function of the type 1 insulin-like growth factor receptor. Cellular and Molecular Life Sciences, 57:1050-1093, Jul 2000. URL: https://doi.org/10.1007/pl00000744, doi:10.1007/pl00000744. This article has 869 citations and is from a domain leading peer-reviewed journal.
9. (adams2000structureandfunction pages 36-37): T. E. Adams, V. C. Epa, T. P. J. Garrett, and C. W. Ward\*. Structure and function of the type 1 insulin-like growth factor receptor. Cellular and Molecular Life Sciences, 57:1050-1093, Jul 2000. URL: https://doi.org/10.1007/pl00000744, doi:10.1007/pl00000744. This article has 869 citations and is from a domain leading peer-reviewed journal.
10. (adams2000structureandfunction pages 5-7): T. E. Adams, V. C. Epa, T. P. J. Garrett, and C. W. Ward\*. Structure and function of the type 1 insulin-like growth factor receptor. Cellular and Molecular Life Sciences, 57:1050-1093, Jul 2000. URL: https://doi.org/10.1007/pl00000744, doi:10.1007/pl00000744. This article has 869 citations and is from a domain leading peer-reviewed journal.
11. (adams2000structureandfunction pages 9-10): T. E. Adams, V. C. Epa, T. P. J. Garrett, and C. W. Ward\*. Structure and function of the type 1 insulin-like growth factor receptor. Cellular and Molecular Life Sciences, 57:1050-1093, Jul 2000. URL: https://doi.org/10.1007/pl00000744, doi:10.1007/pl00000744. This article has 869 citations and is from a domain leading peer-reviewed journal.
12. (amutha2017roleofinsulinlike pages 1-2): P. Amutha and T. Rajkumar. Role of insulin-like growth factor, insulin-like growth factor receptors, and insulin-like growth factor-binding proteins in ovarian cancer. Indian Journal of Medical and Paediatric Oncology : Official Journal of Indian Society of Medical & Paediatric Oncology, 38:198-206, Apr 2017. URL: https://doi.org/10.4103/ijmpo.ijmpo\_3\_17, doi:10.4103/ijmpo.ijmpo\_3\_17. This article has 45 citations.
13. (baserga2000thecontradictionsof pages 1-2): R Baserga. The contradictions of the insulin-like growth factor 1 receptor. Oncogene, 19:5574-5581, Nov 2000. URL: https://doi.org/10.1038/sj.onc.1203854, doi:10.1038/sj.onc.1203854. This article has 323 citations and is from a domain leading peer-reviewed journal.
14. (baserga2000thecontradictionsof pages 4-5): R Baserga. The contradictions of the insulin-like growth factor 1 receptor. Oncogene, 19:5574-5581, Nov 2000. URL: https://doi.org/10.1038/sj.onc.1203854, doi:10.1038/sj.onc.1203854. This article has 323 citations and is from a domain leading peer-reviewed journal.
15. (baserga2000thecontradictionsof pages 5-6): R Baserga. The contradictions of the insulin-like growth factor 1 receptor. Oncogene, 19:5574-5581, Nov 2000. URL: https://doi.org/10.1038/sj.onc.1203854, doi:10.1038/sj.onc.1203854. This article has 323 citations and is from a domain leading peer-reviewed journal.
16. (belfiore2009insulinreceptorisoforms pages 25-26): A. Belfiore, F. Frasca, G. Pandini, L. Sciacca, and R. Vigneri. Insulin receptor isoforms and insulin receptor/insulin-like growth factor receptor hybrids in physiology and disease. Endocrine reviews, 30 6:586-623, Oct 2009. URL: https://doi.org/10.1210/er.2008-0047, doi:10.1210/er.2008-0047. This article has 1311 citations and is from a domain leading peer-reviewed journal.
17. (belfiore2009insulinreceptorisoforms pages 37-38): A. Belfiore, F. Frasca, G. Pandini, L. Sciacca, and R. Vigneri. Insulin receptor isoforms and insulin receptor/insulin-like growth factor receptor hybrids in physiology and disease. Endocrine reviews, 30 6:586-623, Oct 2009. URL: https://doi.org/10.1210/er.2008-0047, doi:10.1210/er.2008-0047. This article has 1311 citations and is from a domain leading peer-reviewed journal.
18. (bondy2004signalingbyinsulinlike pages 1-2): C. Bondy and Clara M. Cheng. Signaling by insulin-like growth factor 1 in brain. European journal of pharmacology, 490 1-3:25-31, Apr 2004. URL: https://doi.org/10.1016/j.ejphar.2004.02.042, doi:10.1016/j.ejphar.2004.02.042. This article has 425 citations and is from a domain leading peer-reviewed journal.
19. (dupont2004insulinandinsulinlike pages 1-2): J. Dupont and D. Leroith. Insulin and insulin-like growth factor i receptors: similarities and differences in signal transduction. Hormone Research in Paediatrics, 55:22-26, Nov 2004. URL: https://doi.org/10.1159/000063469, doi:10.1159/000063469. This article has 306 citations and is from a peer-reviewed journal.
20. (dupont2004insulinandinsulinlike pages 4-5): J. Dupont and D. Leroith. Insulin and insulin-like growth factor i receptors: similarities and differences in signal transduction. Hormone Research in Paediatrics, 55:22-26, Nov 2004. URL: https://doi.org/10.1159/000063469, doi:10.1159/000063469. This article has 306 citations and is from a peer-reviewed journal.
21. (girnita2004cyclolignansasinhibitors pages 1-1): A. Girnita, L. Girnita, F. Prete, A. Bartolazzi, O. Larsson, and M. Axelson. Cyclolignans as inhibitors of the insulin-like growth factor-1 receptor and malignant cell growth. Cancer Research, 64:236-242, Jan 2004. URL: https://doi.org/10.1158/0008-5472.can-03-2522, doi:10.1158/0008-5472.can-03-2522. This article has 440 citations and is from a highest quality peer-reviewed journal.
22. (laron2001insulinlikegrowthfactor pages 2-2): Z. Laron. Insulin-like growth factor 1 (igf-1): a growth hormone. Molecular Pathology, 54:311-316, Oct 2001. URL: https://doi.org/10.1136/mp.54.5.311, doi:10.1136/mp.54.5.311. This article has 922 citations.
23. (leroith2007mechanismsofdisease pages 1-2): Derek LeRoith and Shoshana Yakar. Mechanisms of disease: metabolic effects of growth hormone and insulin-like growth factor 1. Nature Clinical Practice Endocrinology & Metabolism, 3:302-310, Mar 2007. URL: https://doi.org/10.1038/ncpendmet0427, doi:10.1038/ncpendmet0427. This article has 407 citations.
24. (leroith2007mechanismsofdisease pages 6-8): Derek LeRoith and Shoshana Yakar. Mechanisms of disease: metabolic effects of growth hormone and insulin-like growth factor 1. Nature Clinical Practice Endocrinology & Metabolism, 3:302-310, Mar 2007. URL: https://doi.org/10.1038/ncpendmet0427, doi:10.1038/ncpendmet0427. This article has 407 citations.
25. (li2009inhibitionofthe pages 1-2): Rongshi Li, Alan Pourpak, and Stephan W. Morris. Inhibition of the insulin-like growth factor-1 receptor (igf1r) tyrosine kinase as a novel cancer therapy approach. Journal of medicinal chemistry, 52 16:4981-5004, Aug 2009. URL: https://doi.org/10.1021/jm9002395, doi:10.1021/jm9002395. This article has 177 citations and is from a highest quality peer-reviewed journal.
26. (li2009inhibitionofthe pages 2-4): Rongshi Li, Alan Pourpak, and Stephan W. Morris. Inhibition of the insulin-like growth factor-1 receptor (igf1r) tyrosine kinase as a novel cancer therapy approach. Journal of medicinal chemistry, 52 16:4981-5004, Aug 2009. URL: https://doi.org/10.1021/jm9002395, doi:10.1021/jm9002395. This article has 177 citations and is from a highest quality peer-reviewed journal.
27. (li2009inhibitionofthe pages 21-22): Rongshi Li, Alan Pourpak, and Stephan W. Morris. Inhibition of the insulin-like growth factor-1 receptor (igf1r) tyrosine kinase as a novel cancer therapy approach. Journal of medicinal chemistry, 52 16:4981-5004, Aug 2009. URL: https://doi.org/10.1021/jm9002395, doi:10.1021/jm9002395. This article has 177 citations and is from a highest quality peer-reviewed journal.
28. (li2009inhibitionofthe pages 22-24): Rongshi Li, Alan Pourpak, and Stephan W. Morris. Inhibition of the insulin-like growth factor-1 receptor (igf1r) tyrosine kinase as a novel cancer therapy approach. Journal of medicinal chemistry, 52 16:4981-5004, Aug 2009. URL: https://doi.org/10.1021/jm9002395, doi:10.1021/jm9002395. This article has 177 citations and is from a highest quality peer-reviewed journal.
29. (li2009inhibitionofthe pages 26-28): Rongshi Li, Alan Pourpak, and Stephan W. Morris. Inhibition of the insulin-like growth factor-1 receptor (igf1r) tyrosine kinase as a novel cancer therapy approach. Journal of medicinal chemistry, 52 16:4981-5004, Aug 2009. URL: https://doi.org/10.1021/jm9002395, doi:10.1021/jm9002395. This article has 177 citations and is from a highest quality peer-reviewed journal.
30. (li2009inhibitionofthe pages 28-29): Rongshi Li, Alan Pourpak, and Stephan W. Morris. Inhibition of the insulin-like growth factor-1 receptor (igf1r) tyrosine kinase as a novel cancer therapy approach. Journal of medicinal chemistry, 52 16:4981-5004, Aug 2009. URL: https://doi.org/10.1021/jm9002395, doi:10.1021/jm9002395. This article has 177 citations and is from a highest quality peer-reviewed journal.
31. (li2009inhibitionofthe pages 35-36): Rongshi Li, Alan Pourpak, and Stephan W. Morris. Inhibition of the insulin-like growth factor-1 receptor (igf1r) tyrosine kinase as a novel cancer therapy approach. Journal of medicinal chemistry, 52 16:4981-5004, Aug 2009. URL: https://doi.org/10.1021/jm9002395, doi:10.1021/jm9002395. This article has 177 citations and is from a highest quality peer-reviewed journal.
32. (li2009inhibitionofthe pages 4-5): Rongshi Li, Alan Pourpak, and Stephan W. Morris. Inhibition of the insulin-like growth factor-1 receptor (igf1r) tyrosine kinase as a novel cancer therapy approach. Journal of medicinal chemistry, 52 16:4981-5004, Aug 2009. URL: https://doi.org/10.1021/jm9002395, doi:10.1021/jm9002395. This article has 177 citations and is from a highest quality peer-reviewed journal.
33. (li2009inhibitionofthe pages 5-6): Rongshi Li, Alan Pourpak, and Stephan W. Morris. Inhibition of the insulin-like growth factor-1 receptor (igf1r) tyrosine kinase as a novel cancer therapy approach. Journal of medicinal chemistry, 52 16:4981-5004, Aug 2009. URL: https://doi.org/10.1021/jm9002395, doi:10.1021/jm9002395. This article has 177 citations and is from a highest quality peer-reviewed journal.
34. (li2009inhibitionofthe pages 6-7): Rongshi Li, Alan Pourpak, and Stephan W. Morris. Inhibition of the insulin-like growth factor-1 receptor (igf1r) tyrosine kinase as a novel cancer therapy approach. Journal of medicinal chemistry, 52 16:4981-5004, Aug 2009. URL: https://doi.org/10.1021/jm9002395, doi:10.1021/jm9002395. This article has 177 citations and is from a highest quality peer-reviewed journal.
35. (li2009inhibitionofthe pages 7-9): Rongshi Li, Alan Pourpak, and Stephan W. Morris. Inhibition of the insulin-like growth factor-1 receptor (igf1r) tyrosine kinase as a novel cancer therapy approach. Journal of medicinal chemistry, 52 16:4981-5004, Aug 2009. URL: https://doi.org/10.1021/jm9002395, doi:10.1021/jm9002395. This article has 177 citations and is from a highest quality peer-reviewed journal.
36. (li2009inhibitionofthe pages 9-10): Rongshi Li, Alan Pourpak, and Stephan W. Morris. Inhibition of the insulin-like growth factor-1 receptor (igf1r) tyrosine kinase as a novel cancer therapy approach. Journal of medicinal chemistry, 52 16:4981-5004, Aug 2009. URL: https://doi.org/10.1021/jm9002395, doi:10.1021/jm9002395. This article has 177 citations and is from a highest quality peer-reviewed journal.
37. (li2019structuralbasisof pages 1-2): Jie Li, E. Choi, Hongtao Yu, and X. Bai. Structural basis of the activation of type 1 insulin-like growth factor receptor. Nature Communications, Oct 2019. URL: https://doi.org/10.1038/s41467-019-12564-0, doi:10.1038/s41467-019-12564-0. This article has 165 citations and is from a highest quality peer-reviewed journal.
38. (li2019structuralbasisof pages 4-5): Jie Li, E. Choi, Hongtao Yu, and X. Bai. Structural basis of the activation of type 1 insulin-like growth factor receptor. Nature Communications, Oct 2019. URL: https://doi.org/10.1038/s41467-019-12564-0, doi:10.1038/s41467-019-12564-0. This article has 165 citations and is from a highest quality peer-reviewed journal.
39. (pollak2008insulinandinsulinlike pages 10-11): M. Pollak. Insulin and insulin-like growth factor signalling in neoplasia. Nature Reviews Cancer, 8:915-928, Dec 2008. URL: https://doi.org/10.1038/nrc2536, doi:10.1038/nrc2536. This article has 2538 citations and is from a domain leading peer-reviewed journal.
40. (pollak2008insulinandinsulinlike pages 2-3): M. Pollak. Insulin and insulin-like growth factor signalling in neoplasia. Nature Reviews Cancer, 8:915-928, Dec 2008. URL: https://doi.org/10.1038/nrc2536, doi:10.1038/nrc2536. This article has 2538 citations and is from a domain leading peer-reviewed journal.
41. (pollak2008insulinandinsulinlike pages 3-4): M. Pollak. Insulin and insulin-like growth factor signalling in neoplasia. Nature Reviews Cancer, 8:915-928, Dec 2008. URL: https://doi.org/10.1038/nrc2536, doi:10.1038/nrc2536. This article has 2538 citations and is from a domain leading peer-reviewed journal.
42. (pollak2008insulinandinsulinlike pages 8-9): M. Pollak. Insulin and insulin-like growth factor signalling in neoplasia. Nature Reviews Cancer, 8:915-928, Dec 2008. URL: https://doi.org/10.1038/nrc2536, doi:10.1038/nrc2536. This article has 2538 citations and is from a domain leading peer-reviewed journal.
43. (raile2006clinicalandfunctional pages 5-7): K. Raile, J. Klammt, A. Schneider, A. Keller, S. Laue, Robert J. Smith, R. Pfäffle, Juergen Kratzsch, Eberhard Keller, and Wieland Kiess. Clinical and functional characteristics of the human arg59ter insulin-like growth factor i receptor (igf1r) mutation: implications for a gene dosage effect of the human igf1r. The Journal of clinical endocrinology and metabolism, 91 6:2264-71, Jun 2006. URL: https://doi.org/10.1210/jc.2005-2146, doi:10.1210/jc.2005-2146. This article has 104 citations.
44. (raile2006clinicalandfunctional pages 7-8): K. Raile, J. Klammt, A. Schneider, A. Keller, S. Laue, Robert J. Smith, R. Pfäffle, Juergen Kratzsch, Eberhard Keller, and Wieland Kiess. Clinical and functional characteristics of the human arg59ter insulin-like growth factor i receptor (igf1r) mutation: implications for a gene dosage effect of the human igf1r. The Journal of clinical endocrinology and metabolism, 91 6:2264-71, Jun 2006. URL: https://doi.org/10.1210/jc.2005-2146, doi:10.1210/jc.2005-2146. This article has 104 citations.
45. (raile2006clinicalandfunctional pages 8-8): K. Raile, J. Klammt, A. Schneider, A. Keller, S. Laue, Robert J. Smith, R. Pfäffle, Juergen Kratzsch, Eberhard Keller, and Wieland Kiess. Clinical and functional characteristics of the human arg59ter insulin-like growth factor i receptor (igf1r) mutation: implications for a gene dosage effect of the human igf1r. The Journal of clinical endocrinology and metabolism, 91 6:2264-71, Jun 2006. URL: https://doi.org/10.1210/jc.2005-2146, doi:10.1210/jc.2005-2146. This article has 104 citations.
46. (singh2014insulinreceptor(ir) pages 1-2): Pushpendra Singh, Jimi Marin Alex, and Felix Bast. Insulin receptor (ir) and insulin-like growth factor receptor 1 (igf-1r) signaling systems: novel treatment strategies for cancer. Medical Oncology, 31:1-14, Dec 2014. URL: https://doi.org/10.1007/s12032-013-0805-3, doi:10.1007/s12032-013-0805-3. This article has 270 citations and is from a peer-reviewed journal.
47. (tian2015quantitativeanalysisof pages 10-11): Dan Tian, Isaiah Mitchell, and Pamela K. Kreeger. Quantitative analysis of insulin-like growth factor 2 receptor and insulin-like growth factor binding proteins to identify control mechanisms for insulin-like growth factor 1 receptor phosphorylation. BMC Systems Biology, Dec 2015. URL: https://doi.org/10.1186/s12918-016-0263-6, doi:10.1186/s12918-016-0263-6. This article has 24 citations and is from a peer-reviewed journal.
48. (tsui2008evidenceforan pages 8-9): S. Tsui, Vibha Naik, N. Hoa, C. Hwang, N. Afifiyan, A. S. Sinha Hikim, A. Gianoukakis, Raymond S. Douglas, and Terry J. Smith. Evidence for an association between thyroid-stimulating hormone and insulin-like growth factor 1 receptors: a tale of two antigens implicated in graves’ disease1. The Journal of Immunology, 181:4397-4405, Sep 2008. URL: https://doi.org/10.4049/jimmunol.181.6.4397, doi:10.4049/jimmunol.181.6.4397. This article has 430 citations.
49. (adams2000structureandfunction pages 10-12): T. E. Adams, V. C. Epa, T. P. J. Garrett, and C. W. Ward\*. Structure and function of the type 1 insulin-like growth factor receptor. Cellular and Molecular Life Sciences, 57:1050-1093, Jul 2000. URL: https://doi.org/10.1007/pl00000744, doi:10.1007/pl00000744. This article has 869 citations and is from a domain leading peer-reviewed journal.
50. (adams2000structureandfunction pages 38-39): T. E. Adams, V. C. Epa, T. P. J. Garrett, and C. W. Ward\*. Structure and function of the type 1 insulin-like growth factor receptor. Cellular and Molecular Life Sciences, 57:1050-1093, Jul 2000. URL: https://doi.org/10.1007/pl00000744, doi:10.1007/pl00000744. This article has 869 citations and is from a domain leading peer-reviewed journal.
51. (adams2000structureandfunction pages 7-9): T. E. Adams, V. C. Epa, T. P. J. Garrett, and C. W. Ward\*. Structure and function of the type 1 insulin-like growth factor receptor. Cellular and Molecular Life Sciences, 57:1050-1093, Jul 2000. URL: https://doi.org/10.1007/pl00000744, doi:10.1007/pl00000744. This article has 869 citations and is from a domain leading peer-reviewed journal.
52. (belfiore2009insulinreceptorisoforms pages 1-2): A. Belfiore, F. Frasca, G. Pandini, L. Sciacca, and R. Vigneri. Insulin receptor isoforms and insulin receptor/insulin-like growth factor receptor hybrids in physiology and disease. Endocrine reviews, 30 6:586-623, Oct 2009. URL: https://doi.org/10.1210/er.2008-0047, doi:10.1210/er.2008-0047. This article has 1311 citations and is from a domain leading peer-reviewed journal.
53. (belfiore2009insulinreceptorisoforms pages 27-28): A. Belfiore, F. Frasca, G. Pandini, L. Sciacca, and R. Vigneri. Insulin receptor isoforms and insulin receptor/insulin-like growth factor receptor hybrids in physiology and disease. Endocrine reviews, 30 6:586-623, Oct 2009. URL: https://doi.org/10.1210/er.2008-0047, doi:10.1210/er.2008-0047. This article has 1311 citations and is from a domain leading peer-reviewed journal.
54. (belfiore2009insulinreceptorisoforms pages 28-29): A. Belfiore, F. Frasca, G. Pandini, L. Sciacca, and R. Vigneri. Insulin receptor isoforms and insulin receptor/insulin-like growth factor receptor hybrids in physiology and disease. Endocrine reviews, 30 6:586-623, Oct 2009. URL: https://doi.org/10.1210/er.2008-0047, doi:10.1210/er.2008-0047. This article has 1311 citations and is from a domain leading peer-reviewed journal.
55. (belfiore2009insulinreceptorisoforms pages 29-31): A. Belfiore, F. Frasca, G. Pandini, L. Sciacca, and R. Vigneri. Insulin receptor isoforms and insulin receptor/insulin-like growth factor receptor hybrids in physiology and disease. Endocrine reviews, 30 6:586-623, Oct 2009. URL: https://doi.org/10.1210/er.2008-0047, doi:10.1210/er.2008-0047. This article has 1311 citations and is from a domain leading peer-reviewed journal.
56. (belfiore2009insulinreceptorisoforms pages 36-37): A. Belfiore, F. Frasca, G. Pandini, L. Sciacca, and R. Vigneri. Insulin receptor isoforms and insulin receptor/insulin-like growth factor receptor hybrids in physiology and disease. Endocrine reviews, 30 6:586-623, Oct 2009. URL: https://doi.org/10.1210/er.2008-0047, doi:10.1210/er.2008-0047. This article has 1311 citations and is from a domain leading peer-reviewed journal.