1. Phylogeny  
   Bone morphogenetic protein receptor type‐1B (BMPR1B), also known as ALK6, belongs to the type‐I receptors of the transforming growth factor‐β (TGF‐β) superfamily. Phylogenetically, BMPR1B is highly conserved among vertebrates, with orthologs identified across mammalian species, and it shares a close evolutionary relationship with other type‐I BMP receptors such as BMPR1A (ALK3) and ACVR1 (ALK2) (duffhuesUnknownyeardikep. pages 3-4, gomez‐puerto2019bonemorphogeneticprotein pages 1-2). Within the kinome, BMPR1B is assigned to the serine/threonine kinase family and, more specifically, to the subgroup of activin receptor–like kinases (ALKs) that mediate BMP signaling. The receptor’s evolutionary lineage is traced back to gene duplications that occurred early in vertebrate evolution, resulting in a set of BMP type‐I receptors that include ALK1, ALK2, ALK3, and ALK6 (duffhuesUnknownyeardikep. pages 3-4, weiss2013thetgfbetasuperfamily pages 3-5). Structural and sequence similarities between BMPR1B and its paralogs suggest that functional divergence occurred primarily through variations in extracellular ligand‐binding determinants and intracellular regulatory segments. The conservation of its kinase domain and the glycine–serine (GS) region indicates that the catalytic mechanism is preserved across species, underlining the receptor’s essential role in mediating BMP signals during embryogenesis and tissue homeostasis (duffhuesUnknownyeardikep. pages 3-4, gomez‐puerto2019bonemorphogeneticprotein pages 1-2, nickel2019specificationofbmp pages 11-13).
2. Reaction Catalyzed  
   BMPR1B catalyzes the transfer of a phosphate group from ATP to its protein substrates. On ligand binding and heterotetrameric complex formation with type II BMP receptors, BMPR1B becomes activated through phosphorylation of its intracellular GS domain by the constitutively active type II receptors. Once activated, BMPR1B functions as a serine/threonine kinase that phosphorylates receptor‐regulated SMAD proteins (R‐SMADs), specifically SMAD1, SMAD5, and SMAD8. The chemical reaction can be summarized as follows:  
     ATP + [R‐SMAD] → ADP + [R‐SMAD]-phosphate + H⁺  
   This phosphorylation event initiates downstream signaling whereby phosphorylated R‐SMADs associate with co‐SMAD (SMAD4) and translocate into the nucleus to regulate gene transcription (duffhuesUnknownyeardikep. pages 5-6, cao2005thebmpsignaling pages 1-2).
3. Cofactor Requirements  
   The catalytic activity of BMPR1B depends on the presence of ATP and necessitates divalent metal ions as cofactors, with Mg²⁺ being essential for efficient kinase function. The magnesium ion acts by coordinating with ATP in the active site, which is critical for the stabilization of the transition state during the phosphoryl transfer reaction. This cofactor requirement is a common feature among serine/threonine kinases, ensuring proper catalytic efficiency (cao2005thebmpsignaling pages 1-2, gipson2020structuralperspectiveof pages 24-27).
4. Substrate Specificity  
   BMPR1B displays substrate specificity by preferentially targeting receptor‐regulated SMAD proteins, notably SMAD1, SMAD5, and SMAD8. These substrates contain a conserved C‐terminal SSXS motif, which is the site phosphorylated by BMPR1B upon activation. Although consensus substrate motifs for many serine/threonine kinases have been characterized in detail in recent studies (e.g., Johnson et al., 2023), BMPR1B’s activity in vivo is defined by its ability to recognize and phosphorylate these R‐SMADs within the context of BMP signaling. The receptor’s intracellular kinase domain is structured to accommodate the target motif, thereby ensuring efficient signal propagation through the canonical SMAD pathway (duffhuesUnknownyeardikep. pages 5-6, gomez‐puerto2019bonemorphogeneticprotein pages 5-6, wu2024therolesand pages 17-18).
5. Structure  
   BMPR1B is a transmembrane receptor with a modular domain organization. Its extracellular region contains a ligand‐binding domain characterized by cysteine‐rich repeats that form a three‐finger toxin fold, which is essential for high‐affinity binding to specific BMP ligands such as BMP7/OP-1 and growth differentiation factor‐5 (GDF5) (duffhuesUnknownyeardikep. pages 3-4, carreira2014bonemorphogeneticproteins pages 3-4). This domain is followed by a single transmembrane helix that anchors the receptor in the cell membrane. On the cytosolic side, BMPR1B features a juxtamembrane glycine–serine (GS) region that serves as a regulatory element; phosphorylation of the GS domain by type II receptors relieves inhibitory interactions (for instance, with FKBP12 observed in related receptors) and allows subsequent autophosphorylation required for full activation (duffhuesUnknownyeardikep. pages 4-5, gipson2020structuralperspectiveof pages 11-12). The C-terminal region comprises the kinase domain, which adopts a typical serine/threonine kinase fold, consisting of an N-terminal lobe with a five-stranded β-sheet and terminal α-helices, a conserved C-helix (αC), and a catalytic loop that forms the ATP-binding pocket. The activation loop, which undergoes conformational changes upon phosphorylation, is essential for substrate access and proper catalytic function (mahlawat2012structureofthe pages 1-2, gipson2020structuralperspectiveof pages 24-27, carreira2014bonemorphogeneticproteins pages 3-4). Structural studies, including crystallographic analyses of homologous receptors, have revealed that while the overall architecture is highly conserved, subtle differences in extracellular loops and L45 loop sequences contribute to ligand specificity and signaling nuances among type I BMP receptors (gipson2020structuralperspectiveof pages 3-4, nickel2019specificationofbmp pages 11-13).
6. Regulation  
   The regulation of BMPR1B occurs at multiple levels. In its basal state, BMPR1B is maintained in an inactive conformation partly through the binding of the immunophilin FKBP12, which associates with the GS domain and prevents premature activation by blocking access to phosphorylation sites. Upon BMP ligand binding, the receptor assembles into a heterotetrameric complex with two type II receptors, which phosphorylate the GS domain of BMPR1B, leading to a conformational change that displaces FKBP12 and activates the kinase domain (duffhuesUnknownyeardikep. pages 4-5, gipson2020structuralperspectiveof pages 11-12). Following activation, BMPR1B autophosphorylates additional residues that further stabilize the active conformation and promote substrate binding. Downstream, receptor-regulated SMADs (SMAD1, SMAD5, and SMAD8) are phosphorylated at their conserved C-terminal motifs, initiating their nuclear translocation (cao2005thebmpsignaling pages 1-2, wu2024therolesand pages 17-18). Regulation is also modulated by extracellular antagonists such as Noggin, Chordin, and Gremlin, which can bind BMP ligands, thereby limiting receptor activation. Additionally, intracellular inhibitors such as SMAD6 and SMAD7 provide negative feedback by interfering with receptor-SMAD complex formation or promoting receptor ubiquitination and degradation (alarmo2008characterizationofbone pages 20-23, duffhuesUnknownyeardikep. pages 4-5). The cumulative effect of these post-translational modifications and protein–protein interactions ensures that BMPR1B activity is finely tuned during developmental processes and tissue homeostasis.
7. Function  
   BMPR1B plays a critical role in mediating BMP signaling during skeletal development and chondrogenesis. Upon binding to BMP ligands – notably BMP7/OP-1 and GDF5 – BMPR1B transduces extracellular cues into intracellular responses through the phosphorylation of SMAD1/5/8, which ultimately regulates the transcription of genes involved in cartilage formation and bone differentiation (duffhuesUnknownyeardikep. pages 5-6, gomez‐puerto2019bonemorphogeneticprotein pages 5-6). BMPR1B expression is predominantly observed in early mesenchymal cells and differentiated chondrocytes, reflecting its central role in the promotion and regulation of chondrocyte differentiation. Functional studies have demonstrated that BMPR1B signaling is essential for proper limb development and skeletal morphogenesis, as evidenced by the association of BMPR1B mutations with phenotypes such as brachydactyly and other skeletal dysplasias (cao2005thebmpsignaling pages 2-4, duffhuesUnknownyeardikep. pages 6-7). In addition, BMPR1B positively regulates chondrocyte differentiation through its interaction with GDF5, contributing to the maintenance of cartilage-specific gene expression and matrix production (wu2024therolesand pages 17-18, gomez‐puerto2019bonemorphogeneticprotein pages 1-2). The receptor also participates in non-canonical signaling pathways, including the activation of MAPKs, which further modulate cellular processes such as proliferation, apoptosis, and differentiation. This multifaceted role underscores the importance of BMPR1B not only in embryonic development but also in the maintenance and repair of skeletal tissues (cao2005thebmpsignaling pages 9-11, busch2019bonemarrowniche pages 11-15).
8. Other Comments  
   BMPR1B is a target of significant therapeutic and research interest due to its central role in BMP signaling and its implications in various skeletal disorders. Several small-molecule inhibitors have been developed to modulate BMP receptor kinase activity; for instance, inhibitors such as dorsomorphin and its derivatives (e.g., LDN-193189 and DMH1) have been used experimentally to suppress BMP-mediated signaling in disease contexts, including heterotopic ossification and potentially cancer (busch2019bonemarrowniche pages 11-15, gipson2020structuralperspectiveof pages 24-27). Mutations in BMPR1B have been linked to developmental abnormalities; loss-of-function mutations are associated with skeletal malformations such as brachydactyly, while gain-of-function mutations have been implicated in vascular disorders like pulmonary arterial hypertension (duffhuesUnknownyeardikep. pages 6-7, nickel2019specificationofbmp pages 11-13). Although the detailed catalog of BMPR1B mutations and their functional impacts is less extensive compared to other BMP receptors, available studies indicate that alterations in the kinase domain or GS region can significantly disrupt downstream SMAD signaling, leading to abnormal chondrocyte differentiation and defective bone formation (cao2005thebmpsignaling pages 2-4, duffhuesUnknownyeardikep. pages 6-7). In addition, BMPR1B is being evaluated as a potential therapeutic target in skeletal dysplasias and related disorders, and its modulation through selective inhibitors or ligand traps remains an active area of investigation. The receptor’s regulation by extracellular antagonists and intracellular inhibitory SMADs also presents opportunities to fine-tune its signaling in contexts of disease (gomez‐puerto2019bonemorphogeneticprotein pages 5-6, wu2024therolesand pages 17-18, tabosh2023…characterizationof pages 38-40).
9. References  
   duffhuesUnknownyeardikep. pages 3-4, duffhuesUnknownyeardikep. pages 4-5, duffhuesUnknownyeardikep. pages 5-6, duffhuesUnknownyeardikep. pages 6-7, duffhuesUnknownyeardikep. pages 9-10; gomez‐puerto2019bonemorphogeneticprotein pages 1-2, gomez‐puerto2019bonemorphogeneticprotein pages 5-6; nickel2019specificationofbmp pages 11-13, nickel2019specificationofbmp pages 25-26; alarmo2008characterizationofbone pages 20-23; busch2019bonemarrowniche pages 11-15; cao2005thebmpsignaling pages 1-2, cao2005thebmpsignaling pages 2-4, cao2005thebmpsignaling pages 9-11; carreira2014bonemorphogeneticproteins pages 2-3, carreira2014bonemorphogeneticproteins pages 3-4; gipson2020structuralperspectiveof pages 3-4, gipson2020structuralperspectiveof pages 4-6, gipson2020structuralperspectiveof pages 6-7, gipson2020structuralperspectiveof pages 7-9, gipson2020structuralperspectiveof pages 11-12, gipson2020structuralperspectiveof pages 12-14, gipson2020structuralperspectiveof pages 14-15, gipson2020structuralperspectiveof pages 15-17, gipson2020structuralperspectiveof pages 24-27, gipson2020structuralperspectiveof pages 27-31; kalal2025acomprehensivereview pages 1-2, kalal2025acomprehensivereview pages 2-4, kalal2025acomprehensivereview pages 4-6, kalal2025acomprehensivereview pages 12-13; khodr2021initialmolecularsteps pages 191-193, khodr2021initialmolecularsteps pages 196-198, khodr2021initialmolecularsteps pages 201-203, khodr2021initialmolecularsteps pages 35-41, khodr2021initialmolecularsteps pages 57-62, khodr2021initialmolecularsteps pages 97-101, khodr2021initialmolecularsteps pages 167-171, khodr2021initialmolecularsteps pages 203-207; mahlawat2012structureofthe pages 1-2; wang2020tgfβasa pages 11-12; weiss2013thetgfbetasuperfamily pages 3-5; wu2024therolesand pages 3-3, wu2024therolesand pages 3-4, wu2024therolesand pages 17-18; ye2007bonemorphogeneticproteins pages 2-4, ye2007bonemorphogeneticproteins pages 4-5; chaikuad2012structureofthe pages 1-2, chaikuad2012structureofthe pages 8-8; møen2024fkbp12andregulation pages 21-25, møen2024fkbp12andregulation pages 25-29; tabosh2023…characterizationof pages 38-40.

References

1. (duffhuesUnknownyeardikep. pages 5-6): G Sanchez Duffhues. Di ke, p. ten.(2020). Unknown journal, Unknown year.
2. (gomez‐puerto2019bonemorphogeneticprotein pages 5-6): Maria Catalina Gomez‐Puerto, Prasanna Vasudevan Iyengar, Amaya García de Vinuesa, Peter ten Dijke, and Gonzalo Sanchez‐Duffhues. Bone morphogenetic protein receptor signal transduction in human disease. The Journal of Pathology, 247:9-20, Nov 2019. URL: https://doi.org/10.1002/path.5170, doi:10.1002/path.5170. This article has 243 citations.
3. (nickel2019specificationofbmp pages 11-13): Joachim Nickel and Thomas D. Mueller. Specification of bmp signaling. Cells, 8:1579, Dec 2019. URL: https://doi.org/10.3390/cells8121579, doi:10.3390/cells8121579. This article has 137 citations and is from a peer-reviewed journal.
4. (alarmo2008characterizationofbone pages 20-23): EL Alarmo. Characterization of bone morphogenetic protein 7 in breast cancer. Unknown journal, 2008.
5. (busch2019bonemarrowniche pages 11-15): Caroline Busch and Helen Wheadon. Bone marrow niche crosses paths with bmps: a road to protection and persistence in cml. Biochemical Society Transactions, 47:1307-1325, Sep 2019. URL: https://doi.org/10.1042/bst20190221, doi:10.1042/bst20190221. This article has 10 citations and is from a peer-reviewed journal.
6. (cao2005thebmpsignaling pages 1-2): Xu Cao and Di Chen. The bmp signaling and in vivo bone formation. Gene, 357 1:1-8, Aug 2005. URL: https://doi.org/10.1016/j.gene.2005.06.017, doi:10.1016/j.gene.2005.06.017. This article has 456 citations and is from a peer-reviewed journal.
7. (cao2005thebmpsignaling pages 2-4): Xu Cao and Di Chen. The bmp signaling and in vivo bone formation. Gene, 357 1:1-8, Aug 2005. URL: https://doi.org/10.1016/j.gene.2005.06.017, doi:10.1016/j.gene.2005.06.017. This article has 456 citations and is from a peer-reviewed journal.
8. (cao2005thebmpsignaling pages 9-11): Xu Cao and Di Chen. The bmp signaling and in vivo bone formation. Gene, 357 1:1-8, Aug 2005. URL: https://doi.org/10.1016/j.gene.2005.06.017, doi:10.1016/j.gene.2005.06.017. This article has 456 citations and is from a peer-reviewed journal.
9. (carreira2014bonemorphogeneticproteins pages 3-4): Ana Claudia Carreira, Gutemberg Gomes Alves, William Fernando Zambuzzi, Mari Cleide Sogayar, and José Mauro Granjeiro. Bone morphogenetic proteins: structure, biological function and therapeutic applications. Archives of Biochemistry and Biophysics, 561:64-73, Nov 2014. URL: https://doi.org/10.1016/j.abb.2014.07.011, doi:10.1016/j.abb.2014.07.011. This article has 284 citations and is from a peer-reviewed journal.
10. (duffhuesUnknownyeardikep. pages 3-4): G Sanchez Duffhues. Di ke, p. ten.(2020). Unknown journal, Unknown year.
11. (duffhuesUnknownyeardikep. pages 4-5): G Sanchez Duffhues. Di ke, p. ten.(2020). Unknown journal, Unknown year.
12. (duffhuesUnknownyeardikep. pages 6-7): G Sanchez Duffhues. Di ke, p. ten.(2020). Unknown journal, Unknown year.
13. (gipson2020structuralperspectiveof pages 11-12): Gregory R. Gipson, Erich J. Goebel, Kaitlin N. Hart, Emily C. Kappes, Chandramohan Kattamuri, Jason C. McCoy, and Thomas B. Thompson. Structural perspective of bmp ligands and signaling. Bone, 140:115549, Nov 2020. URL: https://doi.org/10.1016/j.bone.2020.115549, doi:10.1016/j.bone.2020.115549. This article has 56 citations and is from a domain leading peer-reviewed journal.
14. (gipson2020structuralperspectiveof pages 12-14): Gregory R. Gipson, Erich J. Goebel, Kaitlin N. Hart, Emily C. Kappes, Chandramohan Kattamuri, Jason C. McCoy, and Thomas B. Thompson. Structural perspective of bmp ligands and signaling. Bone, 140:115549, Nov 2020. URL: https://doi.org/10.1016/j.bone.2020.115549, doi:10.1016/j.bone.2020.115549. This article has 56 citations and is from a domain leading peer-reviewed journal.
15. (gipson2020structuralperspectiveof pages 24-27): Gregory R. Gipson, Erich J. Goebel, Kaitlin N. Hart, Emily C. Kappes, Chandramohan Kattamuri, Jason C. McCoy, and Thomas B. Thompson. Structural perspective of bmp ligands and signaling. Bone, 140:115549, Nov 2020. URL: https://doi.org/10.1016/j.bone.2020.115549, doi:10.1016/j.bone.2020.115549. This article has 56 citations and is from a domain leading peer-reviewed journal.
16. (gipson2020structuralperspectiveof pages 27-31): Gregory R. Gipson, Erich J. Goebel, Kaitlin N. Hart, Emily C. Kappes, Chandramohan Kattamuri, Jason C. McCoy, and Thomas B. Thompson. Structural perspective of bmp ligands and signaling. Bone, 140:115549, Nov 2020. URL: https://doi.org/10.1016/j.bone.2020.115549, doi:10.1016/j.bone.2020.115549. This article has 56 citations and is from a domain leading peer-reviewed journal.
17. (gipson2020structuralperspectiveof pages 6-7): Gregory R. Gipson, Erich J. Goebel, Kaitlin N. Hart, Emily C. Kappes, Chandramohan Kattamuri, Jason C. McCoy, and Thomas B. Thompson. Structural perspective of bmp ligands and signaling. Bone, 140:115549, Nov 2020. URL: https://doi.org/10.1016/j.bone.2020.115549, doi:10.1016/j.bone.2020.115549. This article has 56 citations and is from a domain leading peer-reviewed journal.
18. (gomez‐puerto2019bonemorphogeneticprotein pages 1-2): Maria Catalina Gomez‐Puerto, Prasanna Vasudevan Iyengar, Amaya García de Vinuesa, Peter ten Dijke, and Gonzalo Sanchez‐Duffhues. Bone morphogenetic protein receptor signal transduction in human disease. The Journal of Pathology, 247:9-20, Nov 2019. URL: https://doi.org/10.1002/path.5170, doi:10.1002/path.5170. This article has 243 citations.
19. (kalal2025acomprehensivereview pages 1-2): A. Kalal and Satyajit Mohapatra. A comprehensive review exploring the role of bone morphogenetic proteins [bmp]: biological mechanisms. Current Issues in Molecular Biology, Feb 2025. URL: https://doi.org/10.3390/cimb47030156, doi:10.3390/cimb47030156. This article has 1 citations and is from a peer-reviewed journal.
20. (kalal2025acomprehensivereview pages 12-13): A. Kalal and Satyajit Mohapatra. A comprehensive review exploring the role of bone morphogenetic proteins [bmp]: biological mechanisms. Current Issues in Molecular Biology, Feb 2025. URL: https://doi.org/10.3390/cimb47030156, doi:10.3390/cimb47030156. This article has 1 citations and is from a peer-reviewed journal.
21. (kalal2025acomprehensivereview pages 2-4): A. Kalal and Satyajit Mohapatra. A comprehensive review exploring the role of bone morphogenetic proteins [bmp]: biological mechanisms. Current Issues in Molecular Biology, Feb 2025. URL: https://doi.org/10.3390/cimb47030156, doi:10.3390/cimb47030156. This article has 1 citations and is from a peer-reviewed journal.
22. (khodr2021initialmolecularsteps pages 191-193): V Khodr. Initial molecular steps of bone regeneration: cellular scale modulation. Unknown journal, 2021.
23. (khodr2021initialmolecularsteps pages 196-198): V Khodr. Initial molecular steps of bone regeneration: cellular scale modulation. Unknown journal, 2021.
24. (khodr2021initialmolecularsteps pages 201-203): V Khodr. Initial molecular steps of bone regeneration: cellular scale modulation. Unknown journal, 2021.
25. (khodr2021initialmolecularsteps pages 35-41): V Khodr. Initial molecular steps of bone regeneration: cellular scale modulation. Unknown journal, 2021.
26. (khodr2021initialmolecularsteps pages 57-62): V Khodr. Initial molecular steps of bone regeneration: cellular scale modulation. Unknown journal, 2021.
27. (khodr2021initialmolecularsteps pages 97-101): V Khodr. Initial molecular steps of bone regeneration: cellular scale modulation. Unknown journal, 2021.
28. (mahlawat2012structureofthe pages 1-2): Pardeep Mahlawat, Udayar Ilangovan, Tanuka Biswas, Lu-Zhe Sun, and Andrew P. Hinck. Structure of the alk1 extracellular domain and characterization of its bone morphogenetic protein (bmp) binding properties. Biochemistry, 51 32:6328-41, Aug 2012. URL: https://doi.org/10.1021/bi300942x, doi:10.1021/bi300942x. This article has 54 citations and is from a peer-reviewed journal.
29. (nickel2019specificationofbmp pages 25-26): Joachim Nickel and Thomas D. Mueller. Specification of bmp signaling. Cells, 8:1579, Dec 2019. URL: https://doi.org/10.3390/cells8121579, doi:10.3390/cells8121579. This article has 137 citations and is from a peer-reviewed journal.
30. (wang2020tgfβasa pages 11-12): Weiguang Wang, Diana Rigueur, and K. Lyons. Tgfβ as a gatekeeper of bmp action in the developing growth plate. Bone, pages 115439, May 2020. URL: https://doi.org/10.1016/j.bone.2020.115439, doi:10.1016/j.bone.2020.115439. This article has 24 citations and is from a domain leading peer-reviewed journal.
31. (weiss2013thetgfbetasuperfamily pages 3-5): Alexander Weiss and Liliana Attisano. The tgfbeta superfamily signaling pathway. WIREs Developmental Biology, 2:47-63, Oct 2013. URL: https://doi.org/10.1002/wdev.86, doi:10.1002/wdev.86. This article has 781 citations.
32. (wu2024therolesand pages 17-18): Mengrui Wu, Shali Wu, Wei Chen, and Yi-Ping Li. The roles and regulatory mechanisms of tgf-β and bmp signaling in bone and cartilage development, homeostasis and disease. Cell Research, 34:101-123, Jan 2024. URL: https://doi.org/10.1038/s41422-023-00918-9, doi:10.1038/s41422-023-00918-9. This article has 173 citations and is from a domain leading peer-reviewed journal.
33. (wu2024therolesand pages 3-3): Mengrui Wu, Shali Wu, Wei Chen, and Yi-Ping Li. The roles and regulatory mechanisms of tgf-β and bmp signaling in bone and cartilage development, homeostasis and disease. Cell Research, 34:101-123, Jan 2024. URL: https://doi.org/10.1038/s41422-023-00918-9, doi:10.1038/s41422-023-00918-9. This article has 173 citations and is from a domain leading peer-reviewed journal.
34. (wu2024therolesand pages 3-4): Mengrui Wu, Shali Wu, Wei Chen, and Yi-Ping Li. The roles and regulatory mechanisms of tgf-β and bmp signaling in bone and cartilage development, homeostasis and disease. Cell Research, 34:101-123, Jan 2024. URL: https://doi.org/10.1038/s41422-023-00918-9, doi:10.1038/s41422-023-00918-9. This article has 173 citations and is from a domain leading peer-reviewed journal.
35. (ye2007bonemorphogeneticproteins pages 2-4): L. Ye, J. Lewis-Russell, H. G. Kyanaston, and W. Jiang. Bone morphogenetic proteins and their receptor signaling in prostate cancer. Histology and histopathology, 22 10:1129-47, Oct 2007. URL: https://doi.org/10.14670/hh-22.1129, doi:10.14670/hh-22.1129. This article has 122 citations and is from a peer-reviewed journal.
36. (ye2007bonemorphogeneticproteins pages 4-5): L. Ye, J. Lewis-Russell, H. G. Kyanaston, and W. Jiang. Bone morphogenetic proteins and their receptor signaling in prostate cancer. Histology and histopathology, 22 10:1129-47, Oct 2007. URL: https://doi.org/10.14670/hh-22.1129, doi:10.14670/hh-22.1129. This article has 122 citations and is from a peer-reviewed journal.
37. (carreira2014bonemorphogeneticproteins pages 2-3): Ana Claudia Carreira, Gutemberg Gomes Alves, William Fernando Zambuzzi, Mari Cleide Sogayar, and José Mauro Granjeiro. Bone morphogenetic proteins: structure, biological function and therapeutic applications. Archives of Biochemistry and Biophysics, 561:64-73, Nov 2014. URL: https://doi.org/10.1016/j.abb.2014.07.011, doi:10.1016/j.abb.2014.07.011. This article has 284 citations and is from a peer-reviewed journal.
38. (chaikuad2012structureofthe pages 1-2): A. Chaikuad, I. Alfano, G. Kerr, C. Sanvitale, Jan H. Boergermann, J. Triffitt, F. von Delft, S. Knapp, P. Knaus, and A. Bullock. Structure of the bone morphogenetic protein receptor alk2 and implications for fibrodysplasia ossificans progressiva. The Journal of Biological Chemistry, 287:36990-36998, Sep 2012. URL: https://doi.org/10.1074/jbc.m112.365932, doi:10.1074/jbc.m112.365932. This article has 210 citations.
39. (chaikuad2012structureofthe pages 8-8): A. Chaikuad, I. Alfano, G. Kerr, C. Sanvitale, Jan H. Boergermann, J. Triffitt, F. von Delft, S. Knapp, P. Knaus, and A. Bullock. Structure of the bone morphogenetic protein receptor alk2 and implications for fibrodysplasia ossificans progressiva. The Journal of Biological Chemistry, 287:36990-36998, Sep 2012. URL: https://doi.org/10.1074/jbc.m112.365932, doi:10.1074/jbc.m112.365932. This article has 210 citations.
40. (duffhuesUnknownyeardikep. pages 9-10): G Sanchez Duffhues. Di ke, p. ten.(2020). Unknown journal, Unknown year.
41. (gipson2020structuralperspectiveof pages 14-15): Gregory R. Gipson, Erich J. Goebel, Kaitlin N. Hart, Emily C. Kappes, Chandramohan Kattamuri, Jason C. McCoy, and Thomas B. Thompson. Structural perspective of bmp ligands and signaling. Bone, 140:115549, Nov 2020. URL: https://doi.org/10.1016/j.bone.2020.115549, doi:10.1016/j.bone.2020.115549. This article has 56 citations and is from a domain leading peer-reviewed journal.
42. (gipson2020structuralperspectiveof pages 15-17): Gregory R. Gipson, Erich J. Goebel, Kaitlin N. Hart, Emily C. Kappes, Chandramohan Kattamuri, Jason C. McCoy, and Thomas B. Thompson. Structural perspective of bmp ligands and signaling. Bone, 140:115549, Nov 2020. URL: https://doi.org/10.1016/j.bone.2020.115549, doi:10.1016/j.bone.2020.115549. This article has 56 citations and is from a domain leading peer-reviewed journal.
43. (gipson2020structuralperspectiveof pages 3-4): Gregory R. Gipson, Erich J. Goebel, Kaitlin N. Hart, Emily C. Kappes, Chandramohan Kattamuri, Jason C. McCoy, and Thomas B. Thompson. Structural perspective of bmp ligands and signaling. Bone, 140:115549, Nov 2020. URL: https://doi.org/10.1016/j.bone.2020.115549, doi:10.1016/j.bone.2020.115549. This article has 56 citations and is from a domain leading peer-reviewed journal.
44. (gipson2020structuralperspectiveof pages 4-6): Gregory R. Gipson, Erich J. Goebel, Kaitlin N. Hart, Emily C. Kappes, Chandramohan Kattamuri, Jason C. McCoy, and Thomas B. Thompson. Structural perspective of bmp ligands and signaling. Bone, 140:115549, Nov 2020. URL: https://doi.org/10.1016/j.bone.2020.115549, doi:10.1016/j.bone.2020.115549. This article has 56 citations and is from a domain leading peer-reviewed journal.
45. (gipson2020structuralperspectiveof pages 7-9): Gregory R. Gipson, Erich J. Goebel, Kaitlin N. Hart, Emily C. Kappes, Chandramohan Kattamuri, Jason C. McCoy, and Thomas B. Thompson. Structural perspective of bmp ligands and signaling. Bone, 140:115549, Nov 2020. URL: https://doi.org/10.1016/j.bone.2020.115549, doi:10.1016/j.bone.2020.115549. This article has 56 citations and is from a domain leading peer-reviewed journal.
46. (kalal2025acomprehensivereview pages 4-6): A. Kalal and Satyajit Mohapatra. A comprehensive review exploring the role of bone morphogenetic proteins [bmp]: biological mechanisms. Current Issues in Molecular Biology, Feb 2025. URL: https://doi.org/10.3390/cimb47030156, doi:10.3390/cimb47030156. This article has 1 citations and is from a peer-reviewed journal.
47. (khodr2021initialmolecularsteps pages 167-171): V Khodr. Initial molecular steps of bone regeneration: cellular scale modulation. Unknown journal, 2021.
48. (khodr2021initialmolecularsteps pages 203-207): V Khodr. Initial molecular steps of bone regeneration: cellular scale modulation. Unknown journal, 2021.
49. (møen2024fkbp12andregulation pages 21-25): J Møen. Fkbp12 and regulation of alk2-and alk3-ligand activity in multiple myeloma cells. Unknown journal, 2024.
50. (møen2024fkbp12andregulation pages 25-29): J Møen. Fkbp12 and regulation of alk2-and alk3-ligand activity in multiple myeloma cells. Unknown journal, 2024.
51. (tabosh2023…characterizationof pages 38-40): T Al Tabosh. … characterization of endothelial cells from hht and pah patients carrying alk1 mutations to propose new therapeutic approaches for these two vascular diseases. Unknown journal, 2023.