1. Phylogeny  
   ACVR1, commonly referred to as ALK2, is a type I receptor serine/threonine kinase that belongs to the transforming growth factor‐β (TGF‐β) superfamily; it is evolutionarily conserved across vertebrate species including mammals, amphibians, and fish, with orthologs identified in human, mouse, and zebrafish, demonstrating high sequence conservation especially in the catalytic kinase and glycine–serine (GS) regulatory domains (katagiri2021accumulatedknowledgeof pages 1-2, chaikuad2012structureofthe pages 1-2).  
   Within the kinome, ALK2 is grouped into the activin receptor–like kinase family (ALK1–7), wherein it shares structural and functional characteristics with other BMP type I receptors such as ALK3 (BMPR1A) and ALK6 (BMPR1B), while its sequence motifs distinguish it from members that mediate largely TGF‐β/activin signals (cui2019perspectivesofsmall pages 1-2, katagiri2021accumulatedknowledgeof pages 1-2).  
   Phylogenetic analyses indicate that the core catalytic elements and regulatory motifs in ALK2 trace back to the common ancestor of eukaryotes, positioning it among a highly conserved set of TGF‐β family receptors responsible for orchestrating developmental signaling processes (chaikuad2012structureofthe pages 1-2, dinther2010alk2r206hmutation pages 1-2).  
   The evolutionary relationships among ALK family members reveal a divergence between receptors that preferentially activate the SMAD1/5/8 branch, as in the case of ALK2, and those that signal via SMAD2/3, underscoring the specialized roles that have emerged during metazoan evolution (katagiri2021accumulatedknowledgeof pages 2-4, chaikuad2012structureofthe pages 1-2).
2. Reaction Catalyzed  
   ACVR1/ALK2 catalyzes an ATP‐dependent phosphorylation reaction in which ATP is used to transfer a phosphate group to serine or threonine residues on substrate proteins, principally targeting the receptor‐regulated SMAD proteins (SMAD1, SMAD5, and SMAD8) that are essential for propagating bone morphogenetic protein (BMP) signals (chaikuad2012structureofthe pages 1-2, mohedas2013developmentofan pages 7-8).  
   The chemical reaction can be represented as follows: ATP + [protein]–(L‑serine or L‑threonine) → ADP + [protein]–(L‑serine/threonine)-phosphate + H⁺, thereby initiating a cascade of intracellular signaling events through the phosphorylation and activation of SMAD transcription factors (chaikuad2012structureofthe pages 1-2, mohedas2013developmentofan pages 7-8).  
   This phosphotransfer reaction is essential for converting an extracellular BMP ligand binding event into a specific intracellular response that alters gene transcription, ultimately affecting developmental and homeostatic processes (groppe2023polypeptidesubstrateaccessibility pages 3-5, chaikuad2012structureofthe pages 3-5).
3. Cofactor Requirements  
   The kinase activity of ACVR1/ALK2 is dependent on divalent cations, with magnesium (Mg²⁺) being the principal cofactor that facilitates the proper positioning of ATP within the catalytic site for efficient phosphotransfer (mohedas2013developmentofan pages 1-2, tsuchida2008signaltransductionpathway pages 1-2).  
   Additionally, experimental assays indicate that manganese (Mn²⁺) can support the autophosphorylation activity of ALK2, though Mg²⁺ is typically the physiological cofactor that ensures efficient catalytic turnover (groppe2023polypeptidesubstrateaccessibility pages 15-17, mohedas2013developmentofan pages 1-2).  
   The requirement for ATP as a phosphate donor further underscores the enzyme’s classification as an ATP-dependent serine/threonine kinase (chaikuad2012structureofthe pages 1-2, groppe2023polypeptidesubstrateaccessibility pages 3-5).
4. Substrate Specificity  
   ACVR1/ALK2 exhibits substrate specificity primarily for receptor-regulated SMAD proteins, particularly SMAD1, SMAD5, and SMAD8, which are phosphorylated on conserved serine residues located at the extreme C-terminal tails that typically conform to a Ser-X-Ser motif (chaikuad2012structureofthe pages 3-5, mohedas2013developmentofan pages 4-5).  
   The catalytic domain’s structural configuration facilitates recognition of these substrates, ensuring that phosphorylation occurs at precise serine/threonine sites that are critical for subsequent formation of SMAD complexes with the common mediator SMAD4 (groppe2023polypeptidesubstrateaccessibility pages 7-9, chaikuad2012structureofthe pages 8-9).  
   Moreover, although in vitro kinase assays sometimes employ surrogate substrates such as casein, the physiological substrates of ALK2 are the C-terminal peptides of BMP-specific SMADs, which contain intrinsically disordered regions that permit efficient phosphorylation (mohedas2013developmentofan pages 4-5, groppe2023polypeptidesubstrateaccessibility pages 3-5).
5. Structure  
   ACVR1/ALK2 is characterized by a modular domain organization comprising an extracellular N-terminal ligand-binding domain, a single-pass transmembrane helix, a juxtamembrane glycine–serine rich (GS) domain, and an intracellular kinase domain (chaikuad2012structureofthe pages 1-2, katagiri2021accumulatedknowledgeof pages 1-2).  
   The extracellular domain is responsible for binding BMP ligands such as BMP7 and GDF2/BMP9, thereby initiating receptor oligomerization and subsequent activation of the intracellular kinase domain through formation of heterotetrameric complexes with type II receptors like ACVR2A, ACVR2B, or AMHR2 (chaikuad2012structureofthe pages 2-3, tsuchida2008signaltransductionpathway pages 1-2).  
   The GS domain, which lies immediately adjacent to the transmembrane region, acts as a regulatory switch; in its unphosphorylated state it maintains the kinase in an inactive conformation partly by facilitating binding of the inhibitory protein FKBP12, whereas phosphorylation of the GS domain by associated type II receptors relieves this inhibition (chaikuad2012structureofthe pages 8-8, katagiri2021accumulatedknowledgeof pages 4-6).  
   The intracellular kinase domain exhibits a bilobal architecture consisting of an N-terminal lobe dominated by beta-sheets and a larger C-terminal lobe composed mainly of alpha-helices; between these lobes lies the ATP-binding pocket, which contains several key residues such as Lys235, Glu248, and His286 that stabilize ATP binding and participate in catalysis (nemec2024discoveryoftwo pages 7-8, mohedas2013developmentofan pages 4-5).  
   Structural studies, including high-resolution X-ray crystallography analyses, have revealed that the activation loop and the hydrophobic spines within the kinase domain are critical for determining the active versus inactive conformations, while a helix–loop–helix (HLH) element adjacent to the catalytic domain plays an important allosteric role in modulating substrate access to the active site (groppe2023polypeptidesubstrateaccessibility pages 7-9, chaikuad2012structureofthe pages 8-9).  
   Detailed structural characterization has provided molecular scaffolds that guide the design of selective small molecule inhibitors by revealing how ligands such as dorsomorphin bind to the ATP pocket and stabilize a specific conformation of the kinase (mohedas2013developmentofan pages 7-8, nemec2024discoveryoftwo pages 3-4).
6. Regulation  
   The activity of ACVR1/ALK2 is intricately regulated by several mechanisms that coordinate its activation and inhibition.  
   Ligand binding to the extracellular domain, primarily by BMPs such as BMP7 or BMP9, promotes receptor oligomerization with type II BMP receptors; this association triggers transphosphorylation of the GS domain, which is a necessary step for converting the receptor from an inactive to an active state (chaikuad2012structureofthe pages 2-3, katagiri2021accumulatedknowledgeof pages 4-6).  
   Under basal conditions, the inhibitory protein FKBP12 binds to the GS domain and functions as a molecular buffer by maintaining ALK2 in a low-activity conformation; this binding prevents inadvertent phosphorylation of downstream substrates in the absence of ligand stimulation (chaikuad2012structureofthe pages 1-2, groppe2023polypeptidesubstrateaccessibility pages 1-2).  
   Mutations within the GS domain, notably the recurrent R206H mutation associated with fibrodysplasia ossificans progressiva (FOP), disrupt the interaction with FKBP12 and destabilize the inactive conformation, thereby leading to constitutive or ligand-hyperresponsive kinase activity (dinther2010alk2r206hmutation pages 1-2, hatsell2015acvr1r206h pages 15-16).  
   Additional allosteric regulation is provided by the HLH regulatory element, which modulates substrate accessibility to the kinase active site; subtle conformational shifts in this element can result in an increase in basal kinase activity without markedly altering FKBP12 binding in some gain-of-function mutants (groppe2023polypeptidesubstrateaccessibility pages 15-17, groppe2023polypeptidesubstrateaccessibility pages 19-20).  
   Beyond these immediate regulatory interactions, ACVR1/ALK2 is subject to further modulation by additional post-translational modifications and by the formation of heteromeric receptor complexes that fine-tune its signal transduction output (katagiri2021accumulatedknowledgeof pages 8-10, shi2019targetingheterotopicossification pages 2-4).
7. Function  
   ACVR1/ALK2 functions primarily as a bone morphogenetic protein (BMP) type I receptor that mediates signaling essential for a variety of developmental processes in vertebrates.  
   Upon activation by its cognate BMP ligands, ALK2 phosphorylates receptor‐regulated SMAD proteins (SMAD1, SMAD5, and SMAD8), which then form complexes with the common mediator SMAD4; these complexes translocate to the nucleus where they regulate the transcription of target genes involved in osteogenesis, chondrogenesis, cardiogenesis, neurogenesis, and reproductive system development (chaikuad2012structureofthe pages 1-2, valer2019acvr1functionin pages 1-4).  
   In addition to its canonical SMAD-dependent signaling, ACVR1/ALK2 is capable of engaging non-canonical pathways such as those involving p38 mitogen-activated protein kinases (MAPKs), which contribute to its roles in tissue remodeling and cellular stress responses (cui2019perspectivesofsmall pages 5-6, katagiri2021accumulatedknowledgeof pages 6-8).  
   Furthermore, ACVR1/ALK2 has an inhibitory role in the TGF‑β/activin signaling axis by competing for binding to type II receptors, thus suppressing activin-induced SMAD2/3 signaling, which highlights its dual regulatory capacity in modulating distinct branches of the TGF‑β superfamily signaling network (chaikuad2012structureofthe pages 2-3, katagiri2021accumulatedknowledgeof pages 8-10).  
   The expression of ACVR1/ALK2 occurs in a wide range of tissues, including skeletal muscle, cartilage, bone, heart, and nervous tissue, reflecting its broad functional contributions to organogenesis and tissue homeostasis (valer2019acvr1functionin pages 6-8, cui2019perspectivesofsmall pages 5-6).  
   Moreover, ACVR1/ALK2 plays a central role in regulating bone formation and skeletal development, as evidenced by its involvement in osteogenic differentiation and its dysregulation in pathologies such as fibrodysplasia ossificans progressiva (FOP), where gain-of-function mutations result in heterotopic ossification (dinther2010alk2r206hmutation pages 1-2, hatsell2015acvr1r206h pages 15-16).
8. Other Comments  
   Selective modulation of ACVR1/ALK2 is of great therapeutic interest, particularly in the context of fibrodysplasia ossificans progressiva (FOP) and other disorders associated with aberrant BMP signaling.  
   Inhibitors such as dorsomorphin and its derivatives (e.g., LDN-193189 and LDN-212854) have been developed to target the ATP-binding site of ALK2, effectively suppressing its kinase activity and downstream SMAD phosphorylation, although their selectivity profiles are continually being refined to minimize off-target effects (mohedas2013developmentofan pages 2-4, mohedas2013developmentofan pages 7-8).  
   In addition to these chemical inhibitors, recent studies have introduced highly selective chemical probes such as MU1700 and M4K2234 that distinguish between ALK1 and ALK2, thereby providing research tools that can dissect the individual contributions of these receptors in complex biological processes (nemec2024discoveryoftwo pages 7-8, nemec2024discoveryoftwo pages 28-28).  
   Furthermore, the kinase inhibitor saracatinib, originally developed as a Src-family kinase inhibitor, has demonstrated potent inhibitory activity against ALK2 and is being evaluated as a clinical candidate for FOP due to its ability to reduce heterotopic ossification in preclinical models (williams2021saracatinibisan pages 1-4, williams2021saracatinibisan pages 14-18).  
   Mutations in ACVR1, particularly the recurrent R206H mutation, have been extensively characterized and are known to confer a gain-of-function phenotype that leads to inappropriate activation of BMP signaling pathways, thereby precipitating the pathological formation of bone in soft tissues; this mutation has been the subject of numerous biochemical and structural studies aimed at understanding its precise impact on receptor regulation and substrate accessibility (dinther2010alk2r206hmutation pages 1-2, hatsell2015acvr1r206h pages 15-16, groppe2023polypeptidesubstrateaccessibility pages 1-2).  
   Additionally, ACVR1/ALK2’s involvement in disease is not limited to FOP; aberrant receptor signaling has also been implicated in diffuse intrinsic pontine glioma (DIPG) and other conditions where dysregulated BMP signaling contributes to abnormal tissue differentiation and growth (katagiri2021accumulatedknowledgeof pages 6-8, valer2019acvr1functionin pages 19-21).  
   Ongoing clinical studies and chemical biology research continue to refine our understanding of ALK2’s structural features and regulatory networks, thereby advancing the development of highly selective inhibitors that offer promise for therapeutic intervention in these severe disorders (williams2021saracatinibisan pages 25-27, nemec2024discoveryoftwo pages 25-26).
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