1. Phylogeny  
   Activin receptor type‑2A (ACVR2A) is a highly conserved transmembrane serine/threonine kinase that belongs to the TGF‑β superfamily and is present in all vertebrate lineages, including mammals, birds, amphibians, and fish (OpenTargets Search: -ACVR2A). ACVR2A shares a close evolutionary relationship with other activin type‑II receptors, such as ACVR2B and with BMPR2, which together represent core components of the TGF‑β receptor system that evolved early in metazoan history (attisano1996activationofsignalling pages 7-8, lotinun2012activinreceptorsignaling pages 1-2). Phylogenetic analyses indicate that the gene encoding ACVR2A can be traced back to a common ancestral kinase present in early vertebrates, and its structure and function have been maintained across species owing to its critical role in ligand recognition and signal transduction (oh2002activintypeiia pages 6-7, kosaki1999leftrightaxismalformations pages 7-7). Moreover, structural similarities in the extracellular ligand‐binding domains and kinase domains place ACVR2A within a well‐defined subgroup of serine/threonine kinases that regulate fundamental developmental and physiological processes (chu2022typeiibmp pages 1-2).
2. Reaction Catalyzed  
   ACVR2A catalyzes the transfer of a phosphate group from ATP to target proteins, specifically mediating the phosphorylation of serine/threonine residues on type‑I receptor substrates that contain conserved activation domains (attisano1996activationofsignalling pages 6-7). The overall reaction can be described by the chemical equation: ATP + [protein]‑OH → ADP + [protein]‑O‑phosphate + H⁺ (attisano1996activationofsignalling pages 1-2).
3. Cofactor Requirements  
   The catalytic activity of ACVR2A requires divalent metal ion cofactors, with magnesium (Mg²⁺) serving as the primary cofactor that facilitates ATP binding and phosphoryl transfer within its kinase domain (lotinun2012activinreceptorsignaling pages 1-2, attisano1996activationofsignalling pages 1-2).
4. Substrate Specificity  
   ACVR2A displays substrate specificity characteristic of TGF‑β family type‑II receptors by phosphorylating serine/threonine residues on its interacting partner, namely the type‑I receptors. In the receptor complex, ACVR2A targets residues within the glycine‑serine (GS) domain of type‑I receptors—a region that is critical for their subsequent autophosphorylation and activation (attisano1996activationofsignalling pages 7-8, lotinun2012activinreceptorsignaling pages 4-6). Although an explicit consensus peptide motif has not been fully defined for ACVR2A, the substrate recognition appears to depend on regions surrounding the phosphorylatable residue that ensure proper conformation for downstream SMAD activation (attisano1996activationofsignalling pages 3-5, chu2022typeiibmp pages 11-12).
5. Structure  
   ACVR2A is a single‐pass transmembrane protein comprising three main structural regions. The N‑terminal extracellular domain adopts a three‑finger toxin fold that is optimized for binding activin ligands, such as activin A and activin B, through a central hydrophobic interface and distinct loop regions that contribute to ligand specificity (chu2022typeiibmp pages 1-2, chu2022typeiibmp pages 12-13). The extracellular domain is connected to a single transmembrane helix, which anchors the receptor in the plasma membrane and facilitates its spatial orientation for complex formation (attisano1996activationofsignalling pages 8-8). The cytoplasmic portion contains a serine/threonine kinase domain that features key catalytic motifs, including the activation loop and the conserved DFG motif, which are critical for ATP binding and phosphate transfer (attisano1996activationofsignalling pages 1-2, goh2017activinreceptortype pages 1-2). Structural studies, including crystallographic analyses and computational models from AlphaFold, reveal that the kinase domain is organized into a bilobal structure with a small N‑lobe and a larger C‑lobe, and contains elements such as the C‑helix that are essential for stabilizing the active conformation (chu2022typeiibmp pages 10-11, attisano1996activationofsignalling pages 8-8, weber2007asilenthbond pages 12-14).
6. Regulation  
   The regulation of ACVR2A occurs primarily through ligand‑dependent heteromeric receptor complex formation and subsequent phosphorylation events. Upon binding activin ligands, ACVR2A recruits type‑I receptors to form a heterotetrameric complex composed of two type‑II and two type‑I receptors, wherein ACVR2A constitutively possesses kinase activity that phosphorylates the GS domain of the type‑I receptors (attisano1996activationofsignalling pages 6-7, chu2022typeiibmp pages 11-12). This phosphorylation event is a critical regulatory step that leads to type‑I receptor autophosphorylation and downstream SMAD activation. In addition, ACVR2A signaling is modulated by extracellular antagonists such as follistatin and inhibin A, which bind activin ligands and reduce their availability for receptor engagement (sasaki2015geneticvariantsin pages 1-2, agapova2016ligandtrapfor pages 12-13). Post‑translational modifications, particularly phosphorylation of residues within the kinase domain, further fine‑tune receptor activity, while receptor internalization and degradation mechanisms such as ubiquitination can control receptor turnover and signal duration (attisano1996activationofsignalling pages 3-5, lotinun2012activinreceptorsignaling pages 7-8, olsen2015activinainhibits pages 1-2).
7. Function  
   ACVR2A is a central mediator in activin signaling that influences a broad range of biological processes. Upon binding its cognate ligands—activin A, activin B, and inhibin A—ACVR2A forms a receptor complex with type‑I receptors, leading to phosphorylation of receptor‑regulated SMAD proteins (primarily SMAD2 and SMAD3) that translocate to the nucleus and modulate gene transcription (attisano1996activationofsignalling pages 7-8, chu2022typeiibmp pages 11-12, lotinun2012activinreceptorsignaling pages 1-2). Through this canonical SMAD pathway, ACVR2A plays a key role in regulating adipogenesis, as evidenced by its involvement in the induction of adipocyte differentiation by growth and differentiation factor‑6 (GDF6) based on similarity (OpenTargets Search: -ACVR2A, agapova2016ligandtrapfor pages 12-13). Moreover, ACVR2A activity is implicated in the control of cellular proliferation and differentiation in multiple tissues, including roles in bone formation and homeostasis, as well as in reproductive biology where genetic variants in its promoter region have been linked to fertility traits (goh2017activinreceptortype pages 1-2, sasaki2015geneticvariantsin pages 2-5, sasaki2015geneticvariantsin pages 5-7). Its downstream signaling via SMAD proteins also contributes to the regulation of tissue repair, fibrosis, and cellular metabolism, placing ACVR2A as an important node within the TGF‑β superfamily signaling network (chu2022typeiibmp pages 10-11, olsen2015activinainhibits pages 5-6).
8. Other Comments  
   ACVR2A has garnered considerable attention as a therapeutic target due to its involvement in diverse disease processes. Ligand trap strategies employing soluble ACVR2A‑Fc fusion proteins have been developed to sequester activin ligands, thereby modulating signaling in conditions such as osteoporosis, bone diseases, and tibia fractures (agapova2016ligandtrapfor pages 12-13, lotinun2012activinreceptorsignaling pages 7-8). In addition, alterations in ACVR2A expression and genetic variants in its regulatory regions have been associated with various malignancies, including colorectal adenocarcinoma and hepatocellular carcinoma, and with traits linked to neuroimaging outcomes and educational attainment as identified through large‑scale genetic association studies (OpenTargets Search: -ACVR2A). In musculoskeletal biology, experimental disruption of ACVR2A signaling in osteoblasts has been shown to increase bone mass, underscoring its role as a negative regulator of bone formation (goh2017activinreceptortype pages 1-2, goh2017activinreceptortype pages 8-9). Although specific disease‑causing mutations in ACVR2A have not been as extensively characterized as those in some other TGF‑β receptors, its central role in activin signal transduction makes it a focus for ongoing drug discovery efforts aimed at modulating aberrant TGF‑β superfamily signaling (attisano1996activationofsignalling pages 8-8, hatsell2015acvr1r206h pages 15-16, katagiri2021accumulatedknowledgeof pages 10-11).
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