1. Phylogeny  
   ULK2 (Unc-51-like kinase 2) is a member of the unc-51-like kinase family, a group of serine/threonine kinases that are evolutionarily related to yeast Atg1 and C. elegans UNC-51, and its orthologs are found in all major metazoan lineages, including mammals where high sequence conservation is observed in the kinase domain (alers2012theincredibleulks pages 5-6).  
   Both ULK2 and its paralog ULK1 are thought to have arisen from a gene duplication event at the base of the chordates, and they together comprise an evolutionarily conserved core set of autophagy regulators that originated from the Last Eukaryotic Common Ancestor (demeter2020ulk1andulk2 pages 3-5, alers2012theincredibleulks pages 1-2).  
   Within the kinome, ULK2 is assigned to a subgroup of autophagy‐initiating kinases that also includes ULK3, ULK4 and STK36, and its evolutionary history is marked by conserved catalytic motifs while non‐catalytic domains have diverged to confer distinct regulatory interactions (lee2011therequirementof pages 1-2, demeter2020ulk1andulk2 pages 3-5).  
   This placement into the unc-51-like kinase family underlines ULK2’s conserved role in regulating autophagy across species and indicates a close evolutionary relationship with other kinases that couple nutrient sensing with cellular degradation pathways (alers2012theincredibleulks pages 2-3).
2. Reaction Catalyzed  
   ULK2 catalyzes the transfer of a phosphate group from ATP to the hydroxyl group of serine or threonine residues present in protein substrates, thereby converting ATP into ADP while generating a phosphorylated protein and releasing a proton (li2017serinethreoninekinaseunc51like pages 11-12).  
   This fundamental phosphorylation reaction, which modulates the activity of downstream proteins in the autophagy pathway as well as in other signaling cascades, is characteristic of serine/threonine kinases (alers2012theincredibleulks pages 5-6).
3. Cofactor Requirements  
   The catalytic activity of ULK2 depends on divalent metal ions, with Mg²⁺ serving as an essential cofactor that facilitates ATP binding and the subsequent phosphoryl transfer reaction (li2017serinethreoninekinaseunc51like pages 11-12).
4. Substrate Specificity  
   ULK2 phosphorylates serine/threonine residues on target proteins, and while the exact consensus sequence for substrate recognition has not been fully defined, studies have proposed potential substrates including components of the autophagy machinery such as ATG13, as well as regulatory proteins like RPTOR, FRS2 and FRS3 (demeter2020ulk1andulk2 pages 1-3, chen2014unc51‐likekinase1 pages 2-4).  
   Additional evidence suggests that ULK2 may phosphorylate subunits of the energy sensor AMPK (PRKAA1, PRKAB2 and PRKAG1), thereby participating in the modulation of energy homeostasis through post-translational modifications (Information, demeter2020ulk1andulk2 pages 1-3).  
   Due to experimental data still requiring further confirmation, the exact amino acid motif preferences of ULK2 remain an active subject of investigation, although its overall substrate specificity is indicative of a role in regulating autophagosome formation upstream of phosphatidylinositol 3-kinase PIK3C3 (alers2012theincredibleulks pages 8-9).
5. Structure  
   ULK2 exhibits a modular domain organization consisting of an N-terminal serine/threonine kinase domain that harbors the catalytic machinery including the ATP-binding site, a central proline/serine-rich region likely involved in protein–protein interactions and regulatory modifications, and a C-terminal domain that mediates interactions with autophagy-related partners such as ATG13 and FIP200 (alers2012theincredibleulks pages 5-6, lee2011therequirementof pages 1-2).  
   Structural studies indicate that the kinase domain of ULK2 adopts the typical bilobal architecture seen in many protein kinases, featuring an N-terminal lobe composed mainly of β-sheets and a C-terminal lobe largely α-helical; within this configuration, key catalytic features such as the activation loop, the hydrophobic spines and the regulatory αC helix are critical for its enzymatic function (chaikuad2019conservationofstructure pages 7-10, karmacharya2023smallmoleculeinhibitors pages 2-5).  
   ULK2 has been shown to form a dimeric assembly through activation segment domain exchange—a structural arrangement that is thought to facilitate trans autophosphorylation and full activation of the kinase (chaikuad2019conservationofstructure pages 7-10, demeter2020ulk1andulk2 pages 6-10).  
   Crystal structures of ULK2 or its phosphomimetic mutants in complex with inhibitors such as MRT68921 reveal an inactive conformation characterized by a distorted αC helix and an open, flexible ATP-binding pocket with a flexible methionine gatekeeper residue (wang2024ulkatg1phasingin pages 12-14, karmacharya2023smallmoleculeinhibitors pages 12-14).  
   These structural features underscore the conformational plasticity of the ULK2 kinase domain, which is central to both its catalytic function and the design of small molecule inhibitors targeting this kinase (chaikuad2019conservationofstructure pages 17-20).
6. Regulation  
   ULK2 activity is tightly regulated by nutrient sensing pathways and is modulated by post-translational modifications that serve as switches between its inactive and active conformations.  
   Under nutrient-rich conditions, ULK2 is phosphorylated by the mammalian target of rapamycin complex 1 (mTORC1), which inhibits its kinase activity by modifying specific serine/threonine residues, thereby suppressing autophagy initiation (alers2012theincredibleulks pages 8-9, chen2014unc51‐likekinase1 pages 4-5).  
   Conversely, during energy stress or starvation, the energy sensor AMPK phosphorylates ULK2 at distinct sites leading to its activation and the subsequent initiation of autophagic processes (lee2011therequirementof pages 1-2, demeter2020ulk1andulk2 pages 1-3).  
   In addition to upstream phosphorylation events, ULK2 undergoes autophosphorylation facilitated by its dimerization via activation segment exchange; this autophosphorylation is essential for achieving full kinase activity (chaikuad2019conservationofstructure pages 7-10, demeter2020ulk1andulk2 pages 5-6).  
   ULK2 also functions within feedback loops in autophagy regulation, acting as both a downstream effector and a negative regulator of mTORC1 by phosphorylating components such as RPTOR, and it may reciprocally regulate AMPK via phosphorylation of its subunits PRKAA1, PRKAB2 and PRKAG1 (Information, demeter2020ulk1andulk2 pages 1-3).  
   These multiple layers of regulation, which include phosphorylation by distinct kinases as well as autophosphorylation, ensure that ULK2 activity is finely tuned in response to cellular energy status and nutrient availability (alers2012theincredibleulks pages 8-9, tan2020functionsofulk1 pages 7-8).
7. Function  
   ULK2 plays a central role in the initiation of autophagy, particularly in response to nutrient deprivation, by acting upstream of phosphatidylinositol 3-kinase PIK3C3 to regulate the formation of autophagophores, the precursors of autophagosomes (alers2012theincredibleulks pages 5-6, demeter2020ulk1andulk2 pages 1-3).  
   Through its kinase activity, ULK2 phosphorylates target proteins that are critical for orchestrating the early steps of autophagosome formation, thereby integrating signals from upstream metabolic sensors into the autophagic machinery (alers2012theincredibleulks pages 8-9, chen2014unc51‐likekinase1 pages 2-4).  
   ULK2 is also implicated in feedback regulation of major nutrient-sensing pathways; by phosphorylating RPTOR, a key component of mTORC1, ULK2 acts as a negative regulator of mTORC1, linking autophagy initiation to the suppression of anabolic signaling under conditions of energy stress (Information, demeter2020ulk1andulk2 pages 1-3).  
   In addition to its role in autophagy, ULK2 is involved in neuronal differentiation, where it participates in granule cell axon formation and early neuronal development. This function appears to be mediated via pathways involving Ras-like GTPase signaling and the regulation of Rab5-dependent endocytic processes, which are critical for vesicular trafficking during neurite outgrowth (Information, lee2011therequirementof pages 1-2).  
   While ULK2 shares overlapping functions with ULK1 in autophagy induction, its substrate repertoire and interaction network suggest that ULK2 may perform specialized roles in certain tissues, particularly in modulating neuronal development and possibly in lipid metabolism in adipocytes as indicated by comparative analyses (demeter2020ulk1andulk2 pages 12-13, saleiro2016beyondautophagynew pages 7-10).
8. Other Comments  
   Several small molecule inhibitors target the ULK kinase family, and compounds such as MRT67307 and MRT68921 have been shown to bind with high affinity to the ULK2 kinase domain; these inhibitors exploit the flexible ATP-binding pocket and have revealed cross-reactivity with kinases such as Aurora A, highlighting challenges in achieving selectivity (karmacharya2023smallmoleculeinhibitors pages 5-7, chaikuad2019conservationofstructure pages 7-10).  
   ULK2 is functionally distinct from its paralog ULK1 in that it is not involved in ammonia-induced autophagy or in the autophagic response of cerebellar granule neurons to low potassium, underscoring substrate- and context-specific regulation within the autophagy pathway (lee2011therequirementof pages 1-2).  
   Dysregulation of ULK2-mediated autophagy has been implicated in several disease contexts, including cancer, where altered autophagic flux can influence tumor cell survival and resistance to therapy, and in neurodegenerative disorders where autophagy plays a role in cellular quality control (chen2014unc51‐likekinase1 pages 4-5, tan2020functionsofulk1 pages 7-8, saleiro2016beyondautophagynew pages 25-27).  
   The dual regulatory role of ULK2 in both activating autophagy and modulating nutrient-sensing pathways through interactions with mTORC1 and AMPK positions it as a potential therapeutic target for pharmacological interventions aimed at restoring autophagic balance in disease (Information, demeter2020ulk1andulk2 pages 1-3).
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