1. Phylogeny  
   NUAK1 is a member of the SNF1/AMPK‐related kinase family, a subgroup of serine/threonine protein kinases that play key roles in energy sensing and cellular homeostasis. Orthologs of NUAK1 have been identified across all mammalian species and in other eukaryotes, reflecting its conserved catalytic core and regulatory function over evolutionary time (molina2021nuakkinasesbrain–ovary pages 1-2, bambang2019decipheringtherole pages 1-2). NUAK1 is closely related to NUAK2, and both form a distinct branch within the AMPK‐related kinase family; this branch is characterized by a conserved catalytic domain and shared regulatory features inherited from the ancestral SNF1 kinase in yeast (vis2021nuak1andnuak2 pages 2-3, bambang2019decipheringtherole pages 1-2). In the context of the human kinome, NUAK1 clusters with other kinases that arose early in eukaryotic evolution and are integral components of the cellular metabolic network.
2. Reaction Catalyzed  
   NUAK1 catalyzes the transfer of a phosphate group from ATP to the hydroxyl group of serine and threonine residues on substrate proteins. In its canonical reaction, ATP and a target serine/threonine-containing protein are converted into ADP and the corresponding phosphorylated protein, with the concomitant release of a proton, as expected for serine/threonine kinases (faisal2020developmentandtherapeutic pages 18-19, vis2021nuak1andnuak2 pages 2-3).
3. Cofactor Requirements  
   The catalytic activity of NUAK1 is dependent on divalent metal ions, most notably Mg²⁺, which is required for optimal ATP binding and subsequent phosphoryl transfer. This dependence on Mg²⁺ is characteristic of serine/threonine kinases in the AMPK family and is essential for stabilizing the negative charges that develop during the reaction (faisal2020developmentandtherapeutic pages 18-19, molina2021nuakkinasesbrain–ovary pages 1-2).
4. Substrate Specificity  
   NUAK1 phosphorylates a range of substrates that are central to its diverse cellular roles. Its target proteins include components regulating cell adhesion, cell ploidy, senescence, and the DNA damage response. For example, NUAK1 phosphorylates the PPP1R12A subunit of the myosin phosphatase 1 complex. Upon phosphorylation, PPP1R12A becomes competent to bind 14-3-3 proteins, which leads to inhibition of myosin light chain (MLC2) dephosphorylation and subsequent changes in actomyosin contractility (faisal2020developmentandtherapeutic pages 2-3, vis2021nuak1andnuak2 pages 15-15). In addition, NUAK1 phosphorylates LATS1 on serine residues (notably Ser-464), thereby controlling LATS1 stability and influencing processes related to cellular senescence and ploidy regulation (faisal2020developmentandtherapeutic pages 19-20, bambang2019decipheringtherole pages 3-4). It also targets p53 by phosphorylating it at Ser-15 and Ser-392, a modification that facilitates p53’s recruitment to the CDKN1A promoter and participates in the DNA damage response, as well as substrates such as ATM and CASP6 that are involved in stress response and apoptosis (faisal2020developmentandtherapeutic pages 20-21, port2018colorectaltumorsrequire pages 1-2). Although specific consensus motifs for NUAK1 have not been fully characterized, its substrate specificity appears to be tailored to the regulation of cytoskeletal dynamics and cell cycle–related proteins.
5. Structure  
   NUAK1 is a 661–amino acid protein with an approximate molecular weight of 74–76 kDa. It is organized around a central catalytic kinase domain that is characteristic of the AMPK‐related kinases. This domain includes the classical bilobal kinase structure with an N-terminal lobe composed predominantly of β‐sheets and a larger C-terminal lobe that is mainly α‐helical (molina2021nuakkinasesbrain–ovary pages 5-6, palma2023nuak1coordinatesgrowth pages 1-2). Key structural features of the catalytic domain include the ATP‐binding site, the activation (T) loop, a conserved DFG motif, and a C‐helix that is critical for proper positioning of catalytic residues. NUAK1 also contains regulatory regions outside of the catalytic domain, including an Akt phosphorylation site near the C terminus (Ser600) that is important for its regulation by survival signaling pathways (molina2021nuakkinasesbrain–ovary pages 5-6, palma2023nuak1coordinatesgrowth pages 1-2). Although high‐resolution X‐ray crystallographic structures are not currently available, computational models based on AlphaFold predictions support the existence of a typical kinase fold with additional regulatory surfaces that could mediate interactions with substrates and adaptor proteins, such as 14-3-3 proteins. Furthermore, structural elements that facilitate interactions with the myosin phosphatase complex have been proposed, reflecting its role in the regulation of cell adhesion, although these regions remain to be precisely mapped.
6. Regulation  
   NUAK1 activity is intricately regulated by a combination of phosphorylation events and protein–protein interactions. Its activation involves phosphorylation by upstream kinases such as LKB1 and Akt. LKB1, typically in complex with STRAD and MO25, phosphorylates NUAK1 on a conserved threonine residue within the activation loop; this modification is essential for catalytic activity and marks one of the primary regulatory inputs (vis2021nuak1andnuak2 pages 2-3, molina2021nuakkinasesbrain–ovary pages 1-2). In parallel, Akt phosphorylates NUAK1 at Ser600, modulating its function in cell survival and tumor progression (mo2021roleofark5 pages 1-2, faisal2020developmentandtherapeutic pages 1-2). In addition to these activating events, NUAK1 is subjected to post‐translational regulation via phosphorylation by cell cycle–regulated kinases such as cyclin-dependent kinases and Polo-like kinase 1 (PLK1). Specifically, PLK1 phosphorylates NUAK1 at serine residues (e.g., at Ser476 and Ser480), which facilitates the binding of the SCFβTrCP E3 ubiquitin ligase complex. This binding promotes polyubiquitination and subsequent proteasomal degradation of NUAK1 during the G2/M phase of the cell cycle, thereby ensuring temporal control over its cellular levels and activity (banerjee2014interplaybetweenpolo pages 11-12, banerjee2014interplaybetweenpolo pages 12-12). Also, interactions with 14-3-3 proteins following phosphorylation of its substrates contribute to the regulatory network that modulates NUAK1’s downstream signaling. Collectively, these modifications and interactions enable tight spatial and temporal control of NUAK1 signaling in response to diverse cellular cues.
7. Function  
   NUAK1 fulfills a multiplicity of roles in cellular physiology. One of its primary functions is the regulation of cell adhesion. By phosphorylating the PPP1R12A subunit of the myosin phosphatase 1 complex, NUAK1 enhances the binding of 14-3-3 proteins, which in turn reduces the dephosphorylation of myosin light chain 2 (MLC2). This modulation of myosin phosphatase activity alters actomyosin contractility, thereby influencing cell adhesion, motility, and cytoskeletal reorganization (faisal2020developmentandtherapeutic pages 3-4, vis2021nuak1andnuak2 pages 15-15). In parallel, NUAK1 is pivotal in maintaining proper cellular ploidy and regulating senescence. It phosphorylates LATS1 at Ser464, a modification that controls LATS1 stability and thereby influences cell cycle progression and the onset of cellular senescence (faisal2020developmentandtherapeutic pages 19-20, bambang2019decipheringtherole pages 3-4). NUAK1 also plays a role in the DNA damage response by phosphorylating the tumor suppressor p53 at Ser15 and Ser392. These modifications promote p53 recruitment to target promoters such as CDKN1A/WAF1, facilitating transcriptional activation of genes involved in cell cycle arrest and repair mechanisms (faisal2020developmentandtherapeutic pages 20-21, port2018colorectaltumorsrequire pages 1-2). Beyond these roles, NUAK1 is implicated in tumor progression. Its activity is augmented by phosphorylation via Akt1, which links NUAK1 function to pathways that foster cancer cell proliferation, invasion, and metastasis. For instance, NUAK1 has been shown to coordinate growth factor‐dependent activation of mTORC2 and AKT signaling, thereby contributing to enhanced survival and metabolic adaptation in tumor cells (palma2023nuak1coordinatesgrowth pages 1-2, vis2021nuak1andnuak2 pages 12-14). The cumulative impact of these pathways positions NUAK1 as a multifunctional regulator with critical roles in cell cycle control, cytoskeletal dynamics, adhesion, senescence, DNA damage response, and oncogenic transformation (faisal2020developmentandtherapeutic pages 21-22, bambang2019decipheringtherole pages 7-8).
8. Other Comments  
   The therapeutic potential of targeting NUAK1 has attracted significant interest in recent years, particularly in the context of cancer. Several small molecule inhibitors have been developed that selectively target NUAK1 activity by binding to its ATP‐binding pocket. Notable among these are the 7-membered ring compounds such as HTH-01-015 and the 2,4,5-trisubstituted pyrimidine WZ4003. These inhibitors have demonstrated high potency and selectivity, and they have provided critical insights into NUAK1 function in preclinical models (banerjee2014interplaybetweenpolo pages 11-12, faisal2020developmentandtherapeutic pages 15-17). NUAK1 overexpression has been correlated with aggressive tumor behavior and poor prognosis in cancers such as colorectal cancer and non-small cell lung cancer, making it a potential biomarker for disease progression (port2018colorectaltumorsrequire pages 1-2, vis2021nuak1andnuak2 pages 15-15). In addition, NUAK1 has been implicated in neurodegenerative processes and in the regulation of metabolic homeostasis, although these associations are still under investigation (molina2021nuakkinasesbrain–ovary pages 6-8, faisal2020developmentandtherapeutic pages 21-22). Mutations or alterations that affect its phosphorylation sites and degradation signals could have significant impacts on its cellular functions. The high degree of conservation of its catalytic domain and regulatory motifs underscores the potential for developing more refined and potent inhibitors, which may serve not only as tools to dissect NUAK1’s biological role but also as leads for therapeutic intervention in cancer and metabolic disorders (palma2023nuak1coordinatesgrowth pages 22-22, vis2021nuak1andnuak2 pages 5-7).
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