1. Phylogeny – STK17A, also known as DRAK1, is a member of the death‐associated protein kinase (DAPK) family, which comprises several serine/threonine kinases including DAPK1, DAPK2, DAPK3, DRAK1, and DRAK2. STK17A is evolutionarily conserved among vertebrates and its core catalytic domain shares approximately 47–50% sequence homology with DAPK1, placing it within the serine/threonine protein kinase superfamily. Its closest paralog within this family is DRAK2, and orthologs of STK17A are found across mammalian species. This evolutionary conservation is in line with the essential roles played by the DAPK family in apoptosis and stress responses (kogel2001thedapkinase pages 2-4, farag2019death‐associatedproteinkinase pages 2-4, mao2013serinethreoninekinase17a pages 1-2).
2. Reaction Catalyzed – STK17A catalyzes a phosphorylation reaction in which a phosphate group is transferred from ATP to specific hydroxyl groups on serine or threonine residues of protein substrates. The generalized chemical reaction is represented as:  
     ATP + [protein] – (L-serine or L-threonine) → ADP + [protein] – (phospho-L-serine/threonine) + H⁺.  
   This reaction is characteristic of serine/threonine kinases, whereby the binding and hydrolysis of ATP lead to the post-translational modification of target proteins (kogel2001thedapkinase pages 2-4, tur2017restorationofdap pages 1-3).
3. Cofactor Requirements – The catalytic activity of STK17A requires divalent magnesium ions (Mg²⁺) as a cofactor. Magnesium ions facilitate ATP binding by coordinating the phosphate groups and are essential for proper catalytic efficiency, as observed for other kinases in the serine/threonine group (kogel2001thedapkinase pages 1-2, farag2019death‐associatedproteinkinase pages 4-6).
4. Substrate Specificity – Although the detailed consensus phosphorylation motif for STK17A has not been definitively established, studies indicate that STK17A phosphorylates serine/threonine residues on target proteins. It is implicated in modifying substrates involved in apoptotic signaling pathways and cytoskeletal regulation. Data from related DAPK family members suggest that substrates such as components of the actin–myosin system may be involved, although the precise amino acid specificity remains less well defined when compared to other serine/threonine kinases (kogel2001thedapkinase pages 2-4, farag2019death‐associatedproteinkinase pages 10-12, manivannan2019rnaselinduces pages 6-8).
5. Structure – STK17A is organized with a conserved N-terminal catalytic kinase domain and a C-terminal regulatory region that contains nuclear localization signals (NLSs). The N-terminal domain adopts the canonical bilobed structure seen in protein kinases, featuring a β-sheet-rich N-lobe and an α-helix–rich C-lobe, and includes a highly conserved lysine residue (commonly designated K90) essential for coordinating ATP within the active site. The activation segment (T-loop) is present and, although its precise regulatory phosphorylation status in STK17A has not been fully characterized, it is presumed to play a role in modulating activity. In contrast to DAPK1 and DAPK2, STK17A lacks a calcium/calmodulin-binding autoinhibitory domain; instead, its C-terminal region contains one or more nuclear localization signals that govern subcellular trafficking and are critical for its pro-apoptotic function. Structural models derived from crystallographic studies of related kinases and from predictive methods such as AlphaFold support the view that STK17A shares the typical serine/threonine kinase fold with additional unique regulatory sequences that distinguish it from other family members (reddy2017roleofdsrnainduced pages 41-47, kogel2001thedapkinase pages 2-4, farag2019death‐associatedproteinkinase pages 4-6, zheng2022newinsightsinto pages 2-4).
6. Regulation – The regulatory mechanisms for STK17A involve both transcriptional and post-translational processes. Transcriptionally, STK17A is induced in response to cellular stress stimuli; it has been identified as a direct target of the p53 pathway following DNA damage, and its expression is also upregulated via interferon signaling triggered by double-stranded RNA and subsequent OAS/RNase L activation. Post-translational regulation includes phosphorylation events that modulate the activity of its catalytic domain, and the proper execution of its pro-apoptotic functions depends on nuclear localization mediated by conserved NLS sequences in the C-terminal region. Notably, unlike other members of the DAPK family, STK17A does not contain a calcium/calmodulin regulatory domain, which distinguishes its mode of regulation. These regulatory controls ensure that STK17A activity is tightly coupled to cellular stress responses and the antiviral defense system (reddy2017roleofdsrnainduced pages 17-23, reddy2017roleofdsrnainduced pages 47-51, mao2013serinethreoninekinase17a pages 1-2, farag2019death‐associatedproteinkinase pages 4-6, tur2017restorationofdap pages 1-3).
7. Function – STK17A functions as a positive regulator of apoptosis. Its activation is associated with the engagement of the c-Jun N-terminal kinase (JNK) signaling pathway and the subsequent cleavage of apoptotic effectors such as caspase-3 and poly(ADP-ribose) polymerase (PARP), thereby facilitating mitochondrial-mediated cell death. In addition, STK17A modulates the levels of cellular reactive oxygen species (ROS), linking its activity to the regulation of oxidative stress. Induction of STK17A occurs in response to interferon signaling and the activation of the RNase L pathway following exposure to double-stranded RNA, which ties its function to innate antiviral responses. Functionally, STK17A exhibits context-dependent roles in cancer; for example, studies in glioblastoma models have demonstrated that its overexpression correlates with enhanced cell proliferation and survival, whereas in colorectal cancer, reduced STK17A expression is observed along with alterations in epithelial characteristics. Genetic association studies have further linked STK17A to hematological traits and neuroimaging measurements, underscoring its involvement in blood cell biology and neural function (reddy2017roleofdsrnainduced pages 17-23, mao2013serinethreoninekinase17a pages 1-2, short2019serinethreoninekinase pages 5-5, OpenTargets Search: -STK17A, zheng2022newinsightsinto pages 4-5).
8. Other Comments – Several selective small molecule inhibitors targeting STK17A have been developed. Notably, quinazoline-based dual inhibitors have been reported to exhibit high potency against both STK17A and its close homolog STK17B, with compound inhibitory concentrations (IC₅₀) as low as 23 nM reported in biochemical assays. Additional studies have described indirubin-3′-monoxime derivatives with nanomolar inhibitory activity against DRAK1, and FDA-approved kinase inhibitors such as baricitinib have also been shown to inhibit STK17A with high potency. Disease association data from integrated genetic studies have linked STK17A to variability in hematological traits as well as neuroimaging biomarkers; moreover, experimental models in cancer have reported dual roles for STK17A in cell survival and apoptosis. Ongoing research continues to define its substrate specificity and interaction partners, as well as to refine chemical probes for future functional studies (chaudhry2024potentselectiveand pages 1-2, farag2019death‐associatedproteinkinase pages 23-26, tur2017restorationofdap pages 1-3, OpenTargets Search: -STK17A).
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