1. Phylogeny  
   Serine/threonine‐protein kinase 24 (STK24), also known as MST3, is a member of the mammalian STE20‐like kinase family and is classified within the germinal center kinase III (GCKIII) subgroup. STK24 is evolutionarily conserved across mammalian species, with high sequence identity observed among human, mouse, and rat orthologs, which reflects its preservation from early eukaryotic ancestors as mapped in comparative kinome studies (qiu2023molecularmechanismsinvolved pages 1-3, jiang2018proteinkinaseserinethreonine pages 11-14). Phylogenetic analyses have placed STK24 alongside other GCKIII members, including MST4 and STK25, with these kinases sharing a conserved catalytic core and similar regulatory architectures that are characteristic of the STE20‐like kinases (alzahrani2013ste20likekinaseslk pages 1-2, thiriet2013cytoplasmicproteinserinethreonine pages 1-4).
2. Reaction Catalyzed  
   STK24 catalyzes the transfer of a phosphate group from ATP to specific serine or threonine residues on substrate proteins. The chemical reaction can be summarized as follows:  
     ATP + [protein]‐(L‐serine or L‐threonine) → ADP + [protein]‐(L‐serine/threonine)‐phosphate + H⁺  
   This reaction is mediated via the catalytic activity of the kinase domain and is in line with the general phosphotransferase mechanism characteristic of serine/threonine kinases (jiang2018proteinkinaseserinethreonine pages 1-2, mu2022anallostericregulation pages 3-4).
3. Cofactor Requirements  
   The enzymatic activity of STK24 is dependent on divalent metal ions. In particular, Mg²⁺ is required as a cofactor to facilitate the binding of ATP to the kinase domain, a requirement that is consistent with the biochemical mechanisms of other serine/threonine kinases (mu2022anallostericregulation pages 3-4, thiriet2013cytoplasmicproteinserinethreonine pages 1-4).
4. Substrate Specificity  
   STK24 phosphorylates target proteins on serine and threonine residues. Although a single consensus motif has not been unequivocally defined, experimental studies have demonstrated that STK24 phosphorylates substrates involved in diverse signaling processes. For instance, STK24 is known to phosphorylate STK38L on its Thr-442 residue, thereby stimulating its kinase activity, and it phosphorylates PTPN12, leading to inhibition of its phosphatase activity (jiang2018proteinkinaseserinethreonine pages 11-14, qiu2023molecularmechanismsinvolved pages 8-9). In addition, STK24 contributes to the modulation of NF-κB signaling by promoting phosphorylation events within the IKK complex, although the precise substrate motif remains to be fully delineated (qiu2023molecularmechanismsinvolved pages 9-10).
5. Structure  
   STK24 is organized into two principal regions: an N-terminal kinase domain and a C-terminal regulatory domain. The N-terminal domain, which spans approximately amino acids 36 to 286, adopts the typical bilobal kinase fold with a smaller N-lobe responsible for ATP binding and a larger C-lobe that harbors the substrate-binding site (qiu2023molecularmechanismsinvolved pages 1-3). Key catalytic features within this domain include a conserved lysine residue that facilitates ATP anchoring and a strategically positioned activation loop that undergoes phosphorylation to modulate kinase activity. In many kinases of this family, the Gly-rich loop and the DFG motif are critical for correctly orienting ATP and substrate; these features are presumed to be conserved in STK24 based on structural homology with related STE20-like kinases (thiriet2013cytoplasmicproteinserinethreonine pages 11-14, mu2022anallostericregulation pages 3-4). The C-terminal regulatory domain, which extends from residue 287 to 443, contains nuclear localization and export signals that are thought to be important for subcellular trafficking and potentially contribute to the autoinhibition of the kinase under basal conditions. Isoform diversity is also noted within the MST3 family; for example, brain-specific isoforms such as MST3b arise from alternative splicing events that alter the N-terminal region while preserving the conserved catalytic domain (zhou2000identificationofa pages 1-2, zhou2000identificationofa pages 7-8).
6. Regulation  
   The activity of STK24 is subject to complex regulation through multiple post-translational modifications and protein–protein interactions. Autophosphorylation within the activation loop is a key regulatory mechanism that facilitates full catalytic activity, and phosphorylation events by upstream kinases further modulate STK24 activity. Proteolytic cleavage by caspase-3 has been reported to remove inhibitory regulatory regions, resulting in a truncated form that translocates to the nucleus and promotes pro-apoptotic signaling (qiu2023molecularmechanismsinvolved pages 3-7, li2023cellularimpactsof pages 23-25). In addition, STK24 is regulated by interactions with scaffolding proteins and phosphatases such as PP2A, which can dephosphorylate and thereby inactivate the kinase. These regulatory inputs are critical for controlling the balance between its roles in cell survival, apoptosis, and cytoskeletal dynamics (thiriet2013cytoplasmicproteinserinethreonine pages 11-14, li2023cellularimpactsof pages 4-6).
7. Function  
   STK24 functions as a serine/threonine kinase with multifaceted roles in cellular signaling. It promotes apoptosis in response to stress stimuli, including oxidative stress, by modulating the phosphorylation status of key MAPK pathway components such as JNK1, JNK2, and p38 isoforms (MAPK11, MAPK12, MAPK13, and MAPK14) during oxidative stress conditions (jiang2018proteinkinaseserinethreonine pages 11-14, li2023cellularimpactsof pages 23-25). In a staurosporine-induced apoptotic pathway that operates independently of caspase activation, STK24 regulates the nuclear translocation of AIFM1 and ENDOG while modulating associated DNase activity, thereby contributing to cell death (li2023cellularimpactsof pages 23-25, qiu2023molecularmechanismsinvolved pages 7-8). Furthermore, STK24 phosphorylates STK38L on Thr-442, leading to the stimulation of its kinase activity, and in association with STK26, it plays a role in negatively regulating Golgi reorientation during polarized cell migration triggered by RHO activation (jiang2018proteinkinaseserinethreonine pages 11-14, lai2023serinethreonineproteinkinasestk24 pages 14-14). STK24 also influences cellular migration through the regulation of PTPN12 activity, with subsequent effects on PXN phosphorylation, processes that are critical for cell motility and tissue remodeling (qiu2023molecularmechanismsinvolved pages 9-10, li2023cellularimpactsof pages 6-7). In the nervous system, STK24 has been implicated as a key regulator of axon regeneration in both the optic and radial nerves, suggesting roles in neuroregeneration (qiu2023molecularmechanismsinvolved pages 8-9).
8. Other Comments  
   Inhibitor development for STK24 has been pursued as part of efforts to modulate the activity of the STE20-like kinase family in disease contexts. For example, compounds based on a pyrrolopyrimidine scaffold have been designed to target the ATP-binding pocket of kinases in this family, with PF-06447475 being one such inhibitor that displays nanomolar potency in biochemical assays (bata2021inhibitorsofthe pages 3-5). Dysregulation of STK24 has been associated with a variety of pathological states, including inflammatory conditions and certain cancers. In experimental autoimmune encephalomyelitis, a murine model for multiple sclerosis, deficiency of STK24 leads to reduced inflammatory cytokine production and attenuated disease severity, underscoring its role in inflammatory signaling (jiang2018proteinkinaseserinethreonine pages 11-14). In addition, its involvement in regulating cell migration and Golgi dynamics links STK24 to processes that are critical in tumor progression and metastasis. STK24’s modulation of apoptotic pathways, both caspase-dependent and caspase-independent, further highlights its potential as a therapeutic target in diseases where aberrant cell death or cell survival signals are central to pathogenesis (jiang2018proteinkinaseserinethreonine pages 11-14, bata2021inhibitorsofthe pages 3-5).
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The references above provide the primary literature support for the nomenclature and functional profile of STK24 (MST3) as described in this report.

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