1. Phylogeny  
   MSK1 (RPS6KA5) is a serine/threonine protein kinase that is conserved across vertebrate species; it is present in all examined mammalian systems and is notably absent in lampreys, which indicates its origin is linked to an early gene duplication event in vertebrate evolution (cuenda2016emergingrolesof pages 1-2). This enzyme belongs to the ribosomal protein S6 kinase family and is phylogenetically related to the RSK and MSK2 paralogs within the AGC kinase group, a lineage that emerged in early eukaryotes and has been maintained in mammals due to its central role in signal transduction (cuenda2016emergingrolesof pages 4-5, janknecht2003regulationofthe pages 1-2). Its conserved domain architecture featuring dual kinase domains is a characteristic element that underscores its evolutionary relationship with other MAPK‐activated kinases (mccoy2007identificationofnovel pages 1-2).
2. Reaction Catalyzed  
   MSK1 catalyzes the phosphorylation of target proteins by transferring a phosphate group from ATP to the hydroxyl group of serine or threonine residues within its substrates, following the general reaction:  
     ATP + [protein]–(L‑serine or L‑threonine) → ADP + [protein]–(L‑serine/threonine‑phosphate) + H⁺ (mccoy2005msk1activityis pages 1-2, simon2004mitogenandstressactivated pages 1-2).
3. Cofactor Requirements  
   Consistent with the catalytic requirements of serine/threonine kinases, the activity of MSK1 is dependent on the presence of divalent metal ions, with Mg²⁺ serving as the essential cofactor to properly coordinate ATP binding and stabilize the phosphoryl transfer transition state (mccoy2005msk1activityis pages 1-2, simon2004mitogenandstressactivated pages 1-2).
4. Substrate Specificity  
   MSK1 exhibits a refined substrate specificity that is primarily focused on nuclear proteins implicated in transcriptional regulation and chromatin remodeling. Its best‐characterized substrates include the transcription factors CREB1 and ATF1, which are phosphorylated at key regulatory serine residues (for example, Ser133 in CREB1), thereby facilitating CRE‐mediated gene transcription (bramicherrier2005parsingmolecularand pages 1-2, simon2004mitogenandstressactivated pages 1-2). In addition, MSK1 phosphorylates histone proteins—most notably histone H3—whose modification is essential for chromatin relaxation and subsequent gene activation (thomson1999thenucleosomalresponse pages 9-10). Beyond these substrates, MSK1 also regulates the activity of transcription factors such as RELA (NF-κB p65), STAT3, and ETV1/ER81 by catalyzing phosphorylation events that modulate their ability to activate gene expression (janknecht2003regulationofthe pages 1-2, mccoy2007identificationofnovel pages 1-2). Its substrate recognition appears to depend not only on the presence of serine/threonine residues but also on local sequence context that allows for selective phosphorylation within nuclear signaling networks (simon2004mitogenandstressactivated pages 3-4, mackenzie2013msk1andmsk2 pages 9-11).
5. Structure  
   MSK1 is organized into a distinct dual-kinase architecture that includes an N-terminal kinase domain (NTKD) and a C-terminal kinase domain (CTKD) within a single polypeptide. The NTKD, belonging to the AGC kinase family, is primarily responsible for phosphorylating downstream substrates such as transcription factors and histone proteins, whereas the CTKD—related to calmodulin-dependent kinases—facilitates autophosphorylation events critical for full activation of the enzyme (cuenda2016emergingrolesof pages 1-2, mccoy2007identificationofnovel pages 1-2). A MAPK docking motif, localized toward the C-terminal region, mediates the interaction of MSK1 with upstream MAP kinases (ERK1/2 and p38 MAPK), ensuring that phosphorylation by these kinases occurs in a timely and spatially regulated manner (mccoy2005msk1activityis pages 8-10, cuenda2016emergingrolesof pages 7-8). In addition, MSK1 possesses nuclear localization signals that target the enzyme to the nucleus, where it executes its function in chromatin regulation (janknecht2003regulationofthe pages 1-2). Structural studies using site-directed mutagenesis and mass spectrometry have delineated several key phosphorylation sites that are distributed between the two domains; among these, residues such as Ser212 and Ser376 in the NTKD and Thr581 in the CTKD have been identified as critical residues whose phosphorylation is necessary for relieving autoinhibition and stabilizing the active kinase conformation (mccoy2005msk1activityis pages 4-6, mccoy2005msk1activityis pages 7-8). These features, including the activation loop and the arrangement of regulatory phosphorylation sites, are essential for the concerted regulation and catalytic efficacy of MSK1.
6. Regulation  
   The regulatory mechanism of MSK1 is orchestrated by its integration into MAP kinase signaling cascades. It is activated upon phosphorylation by upstream kinases in the ERK1/2 and p38 MAPK pathways, which are stimulated by extracellular mitogens and stress signals such as UV-C irradiation, epidermal growth factor (EGF), and anisomycin (mccoy2005msk1activityis pages 1-2, choi2017mitogenandstressactivated pages 1-2). Activation of MSK1 is dependent on a series of multisite phosphorylation events; key residues such as Thr581, Ser360, Ser212, Ser376, and Ser381 have been implicated in the full activation of the kinase (mccoy2005msk1activityis pages 4-6, mccoy2005msk1activityis pages 7-8). In some contexts, MSK1 is capable of autophosphorylation, which reinforces its activation state and modulates its substrate specificity (mccoy2007identificationofnovel pages 5-6). The MAPK docking motif present in its structure is critical for recruiting active ERK or p38, ensuring that MSK1 is phosphorylated in response to appropriate stimuli (cuenda2016emergingrolesof pages 6-7, mccoy2007identificationofnovel pages 2-3). Additionally, the kinase activity of MSK1 can be inhibited pharmacologically by compounds such as H89, which have been shown to reduce its catalytic activity in vitro without necessarily abolishing its phosphorylation state, thus highlighting a layer of regulatory complexity (aggeli2006involvementofjnks pages 5-6, thomson1999thenucleosomalresponse pages 6-9). MSK1 also plays a regulatory role in controlling inflammatory gene expression; it phosphorylates and thereby influences the activity of transcription factors like RELA, with evidence showing that MSK1-mediated phosphorylation is required for proper repression of inflammatory genes upon glucocorticoid stimulation, a process involving its association with the glucocorticoid receptor NR3C1 (bramicherrier2005parsingmolecularand pages 1-2, janknecht2003regulationofthe pages 1-2).
7. Function  
   MSK1 functions as a pivotal mediator that translates extracellular mitogenic and stress signals into specific nuclear responses through the phosphorylation of transcription factors and chromatin-modifying proteins. It phosphorylates CREB1 and ATF1 at serine residues such as Ser133, which is a critical event for the initiation of immediate early gene transcription in various cell types including neurons and fibroblasts (simon2004mitogenandstressactivated pages 1-2, bramicherrier2005parsingmolecularand pages 1-2). In addition to these transcription factors, MSK1 phosphorylates histone H3, contributing to chromatin remodeling processes that underpin gene activation (thomson1999thenucleosomalresponse pages 9-10). MSK1 also modulates signaling cascades that involve the NF-κB pathway; for instance, it regulates RELA transcriptional activity in response to tumor necrosis factor (TNF) and glucocorticoids via phosphorylation events, and in skeletal myoblasts, it is required for the phosphorylation of RELA at Ser276 (bramicherrier2005parsingmolecularand pages 1-2). Moreover, MSK1 is implicated in the modulation of other signaling molecules such as STAT3 and ETV1/ER81, thereby influencing a broad spectrum of gene regulatory networks that control inflammation, cellular stress responses, and neuronal plasticity (janknecht2003regulationofthe pages 1-2, simon2004mitogenandstressactivated pages 8-9). In neurons, MSK1 contributes to activity-dependent genomic responses and synaptic plasticity, as evidenced by its recruitment following environmental stimulation (privitera2020experiencerecruitsmsk1 pages 15-16). Collectively, MSK1 acts as a central node in the coordination of cellular responses to external stimuli by integrating signals from MAP kinase cascades and effectuating changes in gene expression via phosphorylation of key nuclear substrates.
8. Other Comments  
   Experimental studies have employed pharmacological inhibitors such as H89 to dissect MSK1 function; H89 has been shown to inhibit MSK1’s kinase activity in vitro, although its effects on phosphorylation patterns are complex and do not entirely abrogate the enzyme’s activation (thomson1999thenucleosomalresponse pages 6-9, aggeli2006involvementofjnks pages 5-6). MSK1 has been implicated in a range of pathological conditions through its role in regulating inflammatory gene expression, where its activity can lead to both activation and repression of target genes depending on the cellular context (bramicherrier2005parsingmolecularand pages 1-2, choi2017mitogenandstressactivated pages 1-2). In the realm of neurodegeneration, for example, studies have demonstrated that the Ras–MAPK–MSK1 pathway modulates ataxin-1 protein levels and toxicity in models of spinocerebellar ataxia type 1, underscoring the relevance of MSK1 in disease processes and its potential as a therapeutic target (park2013ras–mapk–msk1pathwaymodulates pages 3-4, park2013ras–mapk–msk1pathwaymodulates pages 10-12). These findings establish MSK1 as an enzyme of interest for further investigation in both inflammatory and neurodegenerative disorders, with ongoing research aimed at identifying more selective inhibitors and elucidating the full spectrum of its interacting partners and downstream signaling effects (sundar2012mitogenandstressactivated pages 16-16).
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