1. Phylogeny  
   MAP kinase‐activated protein kinase 3 (MAPKAPK3), also known as Chromosome 3p kinase, is a member of the MAPK‐activated protein kinase family that falls within the MK2/3 subgroup. It shares approximately 75% amino acid identity with MAPKAPK2 and is believed to have evolved from a common ancestral gene, with homologs identified in diverse species such as Drosophila and C. elegans, indicating a deep evolutionary conservation among metazoans (cargnello2011activationandfunction pages 24-25, ludwig19963pkanovel pages 1-2). This kinase is classified within the larger kinome framework originally reported by Manning et al. and forms part of the signaling apparatus downstream of stress‐activated MAP kinases. Like many other serine/threonine kinases, MAPKAPK3 occupies a central node in the eukaryotic signaling network that evolved early in animal evolution and is co‐expressed and functionally intertwined with other stress‐responsive kinases (cargnello2011activationandfunction pages 1-1, cargnello2011activationandfunction pages 1-2). Its genomic localization on chromosome 3p is notable given that this region is frequently deleted in certain cancers, suggesting that the evolutionary pressures that have maintained MAPKAPK3 in vertebrates may also be linked to its roles in stress and inflammatory response pathways (ludwig19963pkanovel pages 1-2).
2. Reaction Catalyzed  
   MAPKAPK3 catalyzes the ATP‐dependent phosphorylation of protein substrates on serine and, in some contexts, threonine residues. The general chemical reaction involves the transfer of the γ‐phosphate group from ATP to an –OH group on the substrate protein, yielding ADP and a phosphorylated protein product along with the release of a proton: ATP + [protein] –OH → ADP + [protein] –O–PO3^2– + H⁺ (cargnello2011activationandfunction pages 24-25, meloche2010inhibitionofcdk1cyclin pages 270-272).
3. Cofactor Requirements  
   Like most serine/threonine kinases, the catalytic activity of MAPKAPK3 is dependent on the presence of divalent metal ions that coordinate the ATP molecule during the phosphoryl transfer reaction. In this context, magnesium ions (Mg²⁺) are required as an essential cofactor to facilitate the proper binding and orientation of ATP within the kinase active site, ensuring efficient phosphorylation of protein substrates (cargnello2011activationandfunction pages 4-5, meloche2010inhibitionofcdk1cyclin pages 270-272).
4. Substrate Specificity  
   MAPKAPK3 exhibits a substrate specificity that largely overlaps with that of MAPKAPK2. It preferentially phosphorylates serine residues within a consensus peptide motif characterized as Hyd–X–R–X₂–S, where “Hyd” denotes a large hydrophobic residue, “X” represents any amino acid, and “S” is the target serine residue undergoing phosphorylation (cargnello2011activationandfunction pages 24-25, ludwig19963pkanovel pages 1-2). This motif defines a binding and catalytic preference that directs MAPKAPK3 to a subset of substrates involved in stress responses. Notably, substrates identified include small heat shock proteins such as HSP27/HSPB1, where phosphorylation by MAPKAPK3 leads to dissociation of large small heat‐shock protein oligomers and a diminution of their chaperone activities (cargnello2011activationandfunction pages 24-25, williams2017emergingrolesof pages 31-35). In addition, MAPKAPK3 mediates the phosphorylation of several structural and regulatory proteins including KRT18, KRT20, RCSD1, RPS6KA3, TAB3, and the RNA‐binding protein TTP/ZFP36, thereby influencing processes such as cytoskeletal dynamics and inflammatory cytokine production (cargnello2011activationandfunction pages 25-26, ludwig19963pkanovel pages 1-2).
5. Structure  
   The structural organization of MAPKAPK3 is reminiscent of its close paralog MAPKAPK2. It primarily consists of a centralized kinase domain that shows significant homology to calcium/calmodulin‐dependent protein kinases (CAMKs) (cargnello2011activationandfunction pages 12-13, ludwig19963pkanovel pages 2-2). Within this domain, several conserved subdomains can be identified, including a glycine-rich ATP-binding loop, a conserved catalytic loop containing a critical aspartate involved in proton transfer, and an activation loop that undergoes phosphorylation-dependent conformational changes (cargnello2011activationandfunction pages 23-24). In addition to the catalytic core, MAPKAPK3 possesses a proline‐rich N-terminal region that is capable of interacting with Src homology 3 (SH3) domains of proteins such as c-Abl, and a C-terminal region that harbors sequences functioning as a bipartite nuclear localization signal (NLS) as well as a nuclear export signal (NES) (cargnello2011activationandfunction pages 23-24, ludwig19963pkanovel pages 7-8). These regulatory motifs facilitate nucleocytoplasmic shuttling in response to stress stimuli. The overall three-dimensional configuration, as inferred from crystallographic studies of related kinases and supported by AlphaFold predictions, reveals a bilobal organization with a smaller N-terminal lobe predominantly involved in ATP binding and a larger C-terminal lobe responsible for substrate engagement, with the activation loop bridging these functional regions (cargnello2011activationandfunction pages 12-13, ludwig19963pkanovel pages 7-8).
6. Regulation  
   MAPKAPK3 is principally regulated by phosphorylation events that are mediated by upstream MAP kinases, most notably p38‐alpha (MAPK14). Upon exposure to stress stimuli such as oxidative stress, UV irradiation, or inflammatory cytokines, p38 MAPK becomes activated through dual phosphorylation within its conserved activation loop and, in turn, phosphorylates MAPKAPK3 on key threonine residues within its activation loop (cargnello2011activationandfunction pages 24-25, meloche2010inhibitionofcdk1cyclin pages 267-270). This phosphorylation relieves autoinhibitory conformations present in the kinase and promotes its catalytic activity. In addition, MAPKAPK3 may be subject to regulation by other MAPK pathways, including the ERK cascade and SAPK/JNK, albeit its physiological activity is primarily coupled with the p38 pathway (ludwig19963pkanovel pages 8-10, meloche2010inhibitionofcdk1cyclin pages 267-270). Post‐translational modifications that dictate subcellular localization are also significant; MAPKAPK3 harbors a functional bipartite NLS and an NES that together modulate its nucleocytoplasmic trafficking. Under resting conditions, MAPKAPK3 predominantly localizes to the nucleus but can be rapidly exported to the cytoplasm in a CRM1‐dependent manner following stress‐induced phosphorylation (cargnello2011activationandfunction pages 23-24). These regulatory modifications ensure that MAPKAPK3 is present in the appropriate cellular compartment to engage its substrates during stress responses (cargnello2011activationandfunction pages 21-23).
7. Function  
   MAPKAPK3 functions as a stress‐activated serine/threonine kinase with diverse roles in cellular homeostasis. It is involved in mediating responses to environmental and inflammatory stress by modulating the phosphorylation state of substrates that regulate cytokine production, endocytosis, cell migration, chromatin remodeling, and transcriptional regulation (cargnello2011activationandfunction pages 24-25, ludwig19963pkanovel pages 1-2). Upon activation by p38 MAP kinase, MAPKAPK3 phosphorylates substrates such as HSP27/HSPB1, leading to their dissociation from large oligomeric complexes, a process that diminishes the chaperone activity of these heat‐shock proteins and diminishes cellular protection against oxidative stress (cargnello2011activationandfunction pages 24-25, ludwig19963pkanovel pages 1-2). In addition, MAPKAPK3 has been shown to phosphorylate proteins including KRT18, KRT20, RCSD1, RPS6KA3, TAB3, and TTP/ZFP36, which are involved in maintaining cytoskeletal integrity, modulating inflammatory signaling cascades, and influencing post‐transcriptional regulation of gene expression (cargnello2011activationandfunction pages 25-26, williams2017emergingrolesof pages 31-35). Its role in the inflammatory response is highlighted by its involvement in regulating the production of key cytokines such as tumor necrosis factor (TNF) and interleukin-6 (IL6) through the phosphorylation of RNA-binding proteins that control mRNA stability (cargnello2011activationandfunction pages 29-30, ludwig19963pkanovel pages 1-2). Although MAPKAPK3 typically exhibits lower intrinsic kinase activity and lower protein expression compared to MAPKAPK2, its functional contributions are critical in fine-tuning stress and inflammatory responses within the cell (cargnello2011activationandfunction pages 21-23, ludwig19963pkanovel pages 1-2).
8. Other Comments  
   MAPKAPK3 is of considerable interest due to its potential roles in various stress-related and inflammatory diseases. The kinase’s localization on chromosome 3p and its reduced expression compared to MAPKAPK2 have drawn attention to its possible involvement in tumor suppression pathways, with deletions in this chromosomal region being observed in certain malignancies (ludwig19963pkanovel pages 1-2, waldman2010effectsofoncogenic pages 90-93). Although there are currently fewer highly selective inhibitors specifically targeting MAPKAPK3 compared to other kinases in the MAPKAPK family, the development of ATP-competitive compounds that affect both MK2 and MK3 has provided useful experimental tools to dissect its biological function (meloche2010inhibitionofcdk1cyclin pages 267-270, cargnello2011activationandfunction pages 30-31). Moreover, ongoing research continues to elucidate the spectrum of physiological substrates for MAPKAPK3, its impact on cytoskeletal rearrangements, and its modulation of cytokine production. In addition, the interplay between MAPKAPK3 and other MAPKAP kinases in regulating inflammatory and stress response pathways underlines the complexity of kinase signaling networks in human health and disease (ludwig19963pkanovel pages 1-2, cargnello2011activationandfunction pages 24-25). The dual role of MAPKAPK3 in modulating both protective stress responses and pro-inflammatory signaling makes it a potential candidate for therapeutic intervention in diseases where these pathways are dysregulated (cargnello2011activationandfunction pages 24-25, meloche2010inhibitionofcdk1cyclin pages 270-272).
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