1. Phylogeny  
   Mitogen‐activated protein kinase 15 (MAPK15), also known as ERK7 or ERK8, is classified as an atypical member of the MAP kinase family within the human kinome. Phylogenetic analysis based on the protein kinase complement described by Manning et al. (as reflected in johnson2023anatlasof pages 1-2 and li2011evolutionaryhistoryof pages 11-12) shows that MAPK15 diverges significantly from the conventional MAPKs such as ERK1/2 and p38. Its amino acid sequence places it in the broader CMGC group of serine/threonine kinases. In contrast with the well‐conserved catalytic domains of classical MAPKs, MAPK15 has evolved unique regulatory elements that are not found in its canonical counterparts. Orthologs of MAPK15 are present across metazoan species, a pattern that implicates it in an ancient and conserved eukaryotic signaling pathway. Such conservation, despite its divergence in regulatory features, implies that the fundamental roles of MAPK15—especially in controlling autophagy, ciliogenesis, and genome integrity—have been under evolutionary constraint (johnson2023anatlasof pages 1-2, li2011evolutionaryhistoryof pages 11-12, maheshwari2012identificationofconserved pages 66-69).
2. Reaction Catalyzed  
   MAPK15 catalyzes the transfer of a phosphate group from adenosine triphosphate (ATP) to protein substrates at serine or threonine residues. The chemical reaction can be summarized as follows:  
   ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺  
   This reaction, typical for serine/threonine kinases, is responsible for the post-translational modification that regulates the activity, stability, and function of numerous substrate proteins (arfelli2023uhmk1isa pages 1-4, o’boyle2025anatlasof pages 27-31).
3. Cofactor Requirements  
   The catalytic activity of MAPK15 is dependent on the presence of divalent metal ions. In particular, Mg²⁺ is required as a cofactor since it facilitates the coordination of ATP within the catalytic cleft and promotes the transfer of the γ-phosphate group to the substrate protein (arfelli2023uhmk1isa pages 1-4, o’boyle2025anatlasof pages 27-31).
4. Substrate Specificity  
   MAPK15 functions as a serine/threonine kinase and its substrate specificity has been assessed through large-scale phosphoproteomic studies. Although a definitive consensus sequence for MAPK15 has not been fully elucidated, data from Johnson et al. (2023) place it among those kinases that phosphorylate autophagy‐, ciliogenesis‐, and genome integrity‐associated proteins (johnson2023anatlasof pages 1-2). In vitro experiments have demonstrated that MAPK15 phosphorylates well-known substrates, including the transcription factor FOS and myelin basic protein (MBP) (arfelli2023uhmk1isa pages 4-7, yaronbarir2024theintrinsicsubstrate pages 7-8). Moreover, MAPK15 interacts with autophagy-related proteins such as GABARAP, MAP1LC3B, and GABARAPL1. These interactions are critical for stimulating autophagosome formation and for the efficient degradation of the autophagy receptor SQSTM1, with a concomitant reduction in the inhibitory phosphorylation state of MAP1LC3B. This functional association implies that substrate sites phosphorylated by MAPK15 contain sequence elements that are amenable to binding by these autophagy adaptors (arfelli2023uhmk1isa pages 1-4, johnson2023anatlasof pages 7-7).
5. Structure  
   The three-dimensional structure of MAPK15 comprises a central catalytic kinase domain that exhibits the canonical bilobal arrangement found in most MAP kinases. The smaller N-terminal lobe is mainly composed of antiparallel β-sheets, while the larger C-terminal lobe is dominated by α-helices. Key catalytic features include an activation loop, whose phosphorylation status is critical for triggering conformational changes that convert the kinase into an active state; a DFG motif that coordinates Mg²⁺ in the ATP-binding site; a conserved C-helix essential for catalysis; and a hydrophobic spine that stabilizes the active conformation of the enzyme (strambi2013structurepredictionand pages 13-14, johnson2023anatlasof pages 6-7). In addition to the canonical domain, MAPK15 harbors unique regulatory regions outside the kinase core. For example, its extended C-terminal portion contains sequences such as a nuclear localization signal (NLS) that facilitate binding to chromatin and contribute to subcellular localization. AlphaFold structural predictions further support the presence of these atypical surface features that may be central to interactions with substrates such as PCNA and autophagy adaptors (oleaflores2019extracellularsignalregulatedkinase pages 6-7, o’boyle2025anatlasof pages 1-5, pei2023computationalanalysisof pages 15-16).
6. Regulation  
   MAPK15 is subject to multiple layers of regulation that ensure its activity is tightly controlled in response to cellular conditions. Autophosphorylation within the activation loop is a key event that activates MAPK15. Although no classical upstream MAPK kinase (MAP2K) has been firmly established for MAPK15, evidence indicates that noncanonical kinases, for example RET/PTC3 in certain contexts, may contribute to its activation (shrestha2022theregulationof pages 34-38, strambi2013structurepredictionand pages 14-14). In addition, MAPK15 activity is modulated by its binding to chromatin. This chromatin association enhances its interaction with proliferating cell nuclear antigen (PCNA) and plays a critical role in safeguarding genome integrity by counteracting MDM2-mediated degradation of PCNA (arfelli2023uhmk1isa pages 1-4, southekal2021integrativeanalysisof pages 114-120). Oxidative stress, as induced by hydrogen peroxide (H₂O₂) treatment, also regulates MAPK15 by promoting its phosphorylation of ELAVL1. This modification prevents ELAVL1 from binding to the PDCD4 mRNA 3′ untranslated region, thereby permitting miR-21-mediated degradation of PDCD4 mRNA (arfelli2023uhmk1isa pages 7-10, shrestha2022theregulationof pages 87-89). Furthermore, MAPK15 interacts with components of the autophagy machinery—namely GABARAP, MAP1LC3B, and GABARAPL1—to regulate both basal and starvation-induced autophagy. In addition to these kinase activity-dependent mechanisms, MAPK15 exhibits kinase activity-independent roles such as functioning as a negative regulator of cellular growth and modulating the trafficking of proteins, exemplified by its ability to inhibit the relocation of sugar-adding enzymes from the Golgi to the endoplasmic reticulum (arfelli2023uhmk1isa pages 1-4, shrestha2022theregulationof pages 28-31).
7. Function  
   MAPK15 plays versatile roles in cellular physiology, carried out predominantly through its kinase activity. A principal function is the regulation of autophagy. MAPK15 interacts with key autophagy-related proteins—GABARAP, MAP1LC3B, and GABARAPL1—to stimulate autophagosome assembly and to promote the degradation of SQSTM1. This process also involves a reduction in the inhibitory phosphorylation of MAP1LC3B, a modification that normally restrains autophagy (arfelli2023uhmk1isa pages 1-4, gomez2024illuminatingfunctionof pages 1-3). Additionally, MAPK15 has a central role in ciliogenesis. It regulates the formation of the primary cilium and ensures the proper localization of ciliary proteins that are essential for maintaining cilium structure, transport functions, and signaling cascades (arfelli2023uhmk1isa pages 12-15, o’boyle2025anatlasof pages 21-24).  
   MAPK15 also participates in protein trafficking and secretion control. It restricts the retrograde movement of sugar-adding enzymes from the Golgi apparatus to the endoplasmic reticulum, which in turn limits the synthesis of sugar-coated proteins. Under conditions of amino acid starvation, MAPK15 mediates the disassembly of transitional endoplasmic reticulum sites, which leads to the inhibition of secretion (arfelli2023uhmk1isa pages 35-37, southekal2021integrativeanalysisof pages 19-25).  
   Another critical function of MAPK15 is the maintenance of genomic integrity. Through its chromatin-binding capacity and interaction with PCNA, MAPK15 protects PCNA from MDM2-mediated degradation, a safeguarding mechanism essential for proper DNA replication and repair (arfelli2023uhmk1isa pages 1-4, southekal2021integrativeanalysisof pages 114-120). Moreover, MAPK15 has been implicated in regulating dopamine transporter (DAT) activity and expression via activation of RhoA, a pathway that influences neuronal signaling (arfelli2023uhmk1isa pages 7-10, higgins2023sarscov2hijacksp38βmapk11 pages 21-23). In response to oxidative stress, MAPK15 phosphorylates the RNA-binding protein ELAVL1; this event disrupts ELAVL1’s interaction with the 3′ UTR of PDCD4 mRNA, facilitating its degradation through miR-21 action, which adds a layer of post-transcriptional gene regulation (arfelli2023uhmk1isa pages 7-10, shrestha2022theregulationof pages 87-89).  
   Further in vitro studies have established that MAPK15 is capable of phosphorylating substrates such as FOS and MBP, which implicates it in transcriptional regulation and cytoskeletal organization, respectively (arfelli2023uhmk1isa pages 4-7, johnson2023anatlasof pages 12-18). During oocyte maturation and early embryogenesis, MAPK15 is essential for coordinating microtubule organization and progression through the meiotic cell cycle. Additionally, its interaction with the nuclear receptor ESRRA, resulting in the relocalization of ESRRA from the nucleus to the cytoplasm, culminates in the inhibition of ESRRA-dependent transcription, thereby influencing differentiation and growth control (arfelli2023uhmk1isa pages 1-4, shrestha2022theregulationof pages 34-38).
8. Other Comments  
   Despite the extensive analysis of MAPK15’s roles in autophagy, ciliogenesis, secretion, and genomic stability, there are presently no highly selective inhibitors specifically targeting MAPK15 reported in the literature (o’boyle2025anatlasof pages 21-24, shah2023limk2amultifaceted pages 1-3). Although dysregulation of MAPK15 has potential implications in cancer biology as well as in neurodegenerative conditions attributable to its roles in maintaining genome integrity and regulating autophagy, specific disease-associated mutations and detailed clinical inhibitor data have not been fully characterized (shrestha2022theregulationof pages 87-89, southekal2021integrativeanalysisof pages 114-120). Recent computational approaches that integrate deep learning and proximity proteomics have begun to offer insights into the kinase-substrate landscape of the human kinome, yet these studies focus on broad substrate prediction rather than the development of selective inhibitors for MAPK15 (jha2025deeplearningcoupledproximity pages 20-22). In addition, MAPK15 has been shown to exert kinase activity-independent functions, such as negative regulation of cellular growth, which further complicate the development of therapeutic agents. To date, the complexity of MAPK15’s dual modes of action—both as an active kinase and through scaffold-like mechanisms—remains a subject for continued research.
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