1. Phylogeny  
   Tyrosine‐protein kinase Mer (MERTK) is a member of the TAM family of receptor tyrosine kinases, which also comprises Tyro3 and Axl. MERTK orthologs have been characterized in multiple vertebrate species, including human and rodent, indicating that it is highly conserved across these taxa (strick2010focusonmolecules pages 1-2, seitz2007macrophagesanddendritic pages 28-33, ma2011polymorphismsinthe pages 1-2). Phylogenetic analyses place MERTK in a distinct subfamily within the kinome that evolved from a common ancestral gene with its TAM counterparts, thereby sharing a conserved overall domain organization and a similar role in the regulation of phagocytosis and immune homeostasis.
2. Reaction Catalyzed  
   MERTK catalyzes the transfer of the γ‐phosphate from ATP to specific tyrosine residues in substrate proteins. In this phosphotransfer reaction, ATP and the substrate protein combine to yield ADP and a phosphorylated substrate protein, which is essential for propagating intracellular signaling cascades (zhang2013discoveryofmer pages 1-2, huang2009structuralinsightsinto pages 1-2).
3. Cofactor Requirements  
   The catalytic activity of MERTK depends on the binding of ATP and requires divalent metal ions as cofactors, with Mg²⁺ being the predominant ion that facilitates efficient catalytic turnover. The presence of Mg²⁺ is necessary for proper coordination and stabilization of ATP within the kinase’s active site (zhang2013discoveryofmer pages 1-2).
4. Substrate Specificity  
   Investigations employing positional scanning peptide libraries have been used to examine the substrate specificity of MERTK. These studies indicate that MERTK phosphorylates tyrosine residues located within substrate proteins that are involved in cellular processes such as migration, survival, and efferocytosis. Although no definitive consensus motif has been uniformly established, the intrinsic substrate specificity is shaped by the particular amino acid context surrounding the target tyrosine residue (huang2009structuralinsightsinto pages 2-3).
5. Structure  
   MERTK is organized as a single‐pass transmembrane receptor with a modular domain architecture. It contains an N‐terminal signal peptide that directs the protein to the secretory pathway, followed by an extracellular region composed of two immunoglobulin-like (Ig-like) C2 domains and two fibronectin type III (FN-III) domains that mediate ligand binding. A short transmembrane helix anchors the receptor to the plasma membrane, and a large cytoplasmic region houses the tyrosine kinase domain. This intracellular kinase domain contains the conserved catalytic core with an activation loop harboring key autophosphorylation sites (notably Tyr749, Tyr753, and Tyr754) and the invariant KWAIAES motif that is characteristic of TAM kinases (audo2018mertkmutationupdate pages 1-6, huang2009structuralinsightsinto pages 4-5). In addition, structural studies have detailed conformational changes upon inhibitor binding, such as a rotation of the N‐lobe relative to the C‐lobe, and have shown that the receptor can adopt both active and autoinhibited states. Proteolytic cleavage events mediated by metalloproteinases also generate soluble forms of MERTK, which are structurally analogous to the extracellular domain and serve regulatory purposes by sequestering ligands (law2015cleavageofmer pages 11-11).
6. Regulation  
   MERTK regulation is initiated by ligand binding to its extracellular domains; known ligands include GAS6, Protein S, LGALS3, TUB, and TULP1. Ligand engagement promotes receptor dimerization and subsequent autophosphorylation on key tyrosine residues within the intracellular kinase domain, resulting in the formation of docking sites for downstream signaling adaptors such as GRB2 and PLCG2. These post-translational modifications are critical for full kinase activation and the propagation of signaling cascades including those mediated by MAPK1, MAPK2, FAK/PTK2, and RAC1 (audo2018mertkmutationupdate pages 1-6). Moreover, MERTK activity is further regulated by proteolytic cleavage mediated by ADAM17, which releases a soluble extracellular fragment that acts as a decoy receptor to limit ligand availability and modulate signal intensity (law2015cleavageofmer pages 11-12). In addition, the receptor’s interaction with co-receptors such as αvβ5 integrin facilitates cytoskeletal rearrangements necessary for efficient phagocytosis of apoptotic cells.
7. Function  
   MERTK plays multiple roles in physiological processes by transducing signals from the extracellular environment into the cell. It is expressed in macrophages, monocytes, epithelial cells—including retinal pigment epithelium—and other cell types where it regulates diverse biological functions. In macrophages and other phagocytic cells, MERTK is essential for efferocytosis, the clearance of apoptotic cells, thereby contributing to immune homeostasis and the resolution of inflammation. In the retinal pigment epithelium, MERTK-mediated phagocytosis of rod outer segment fragments is critical for retinal maintenance and vision. Beyond its role in phagocytosis, MERTK influences cell survival, migration, differentiation, and cytoskeletal reorganization through downstream signaling pathways such as those mediated by MAP kinases, FAK, and RAC1. Dysregulation of MERTK signaling is associated with various pathologies; loss-of-function mutations in MERTK are causatively linked to inherited retinal dystrophies (for example, retinitis pigmentosa), while overexpression and aberrant activation have been implicated in tumor progression, metastasis, and therapy resistance in a range of cancers (audo2018mertkmutationupdate pages 1-6, audo2018mertkmutationupdate pages 6-10, seitz2007macrophagesanddendritic pages 28-33, nguyen2014overexpressionofmertk pages 1-2).
8. Other Comments  
   Several small molecule inhibitors targeting MERTK have been developed based on structure‐based drug design. Inhibitors that bind to the ATP pocket, including substituted pyrimidine derivatives, have demonstrated high potency and selectivity, and some agents have advanced into preclinical evaluations. In addition to kinase inhibitors, targeted protein degraders have been engineered to induce ubiquitin–proteasome-mediated degradation of MERTK, reflecting alternative therapeutic strategies (cook2013mertkinhibitionin pages 12-12, zhang2013discoveryofmer pages 1-2, gadiyar2023targeteddegradationof pages 1-3). Disease associations for MERTK are diverse; mutations that result in loss of MERTK function are known to cause severe autosomal recessive inherited retinal dystrophies, while hyperactivation or overexpression is linked to cancer progression, metastasis, resistance to therapy, autoimmune disorders, and thrombotic events. Its role in inhibiting Toll-like receptor (TLR)–mediated innate immune responses further underscores its importance in maintaining immune tolerance and preventing excessive inflammation (audo2018mertkmutationupdate pages 17-19, mcdaniel2018mertkmediatesintrinsic pages 9-13).
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