1. Phylogeny  
   TYRO3 is a member of the TAM subfamily of receptor tyrosine kinases, which comprises three closely related kinases: TYRO3, AXL, and MER. This subgroup is conserved across vertebrates, with orthologs identified in human, mouse, rat, and other mammals, indicating a high degree of sequence conservation and preserved functional roles between species (hsu2019tyro3apotential pages 1-2, prieto2024thetamsubfamily pages 1-2). Phylogenetic analyses place TYRO3 within the receptor tyrosine kinase superfamily, and its close evolutionary relationship with AXL and MER implies that these kinases share a common ancestral gene that diverged early in metazoan evolution (prieto2024thetamsubfamily pages 1-2, lemke2013biologyofthe pages 17-18). The TAM receptors as a group are recognized for their specialized roles in immune regulation and apoptotic cell clearance, functions that appear to have been conserved during evolution.
2. Reaction Catalyzed  
   TYRO3 catalyzes the transfer of a phosphate group from ATP to tyrosine residues on target proteins. Specifically, the chemical reaction it mediates can be represented as:  
     ATP + [Protein]-L-tyrosine → ADP + [Protein]-L-tyrosine-phosphate + H⁺  
   This phosphorylation event is central to its role in transmitting extracellular signals into intracellular responses (brown2012crossphosphorylationsignalingand pages 10-10, hunter2015theeukaryoticprotein pages 1-3).
3. Cofactor Requirements  
   The catalytic activity of TYRO3, like that of most protein kinases, depends on the binding of ATP and the presence of divalent metal ions. In particular, Mg²⁺ is typically required as a cofactor to facilitate the proper binding and orientation of ATP within the catalytic cleft, thereby enabling the transfer of the phosphate group to substrate tyrosine residues (boubeva2011understandingtyrosinekinase pages 33-37, hunter2015theeukaryoticprotein pages 1-3).
4. Substrate Specificity  
   TYRO3 exhibits substrate specificity toward tyrosine residues on effector proteins that participate in downstream signaling pathways. Although a precise consensus sequence is not fully defined in the literature provided, the kinase is known to phosphorylate specific tyrosine motifs that allow for the recruitment of SH2 domain–containing proteins, such as the p85 regulatory subunit of phosphatidylinositol 3-kinase (PI3K) (hsu2019tyro3apotential pages 1-2, lan2000transformingactivityof pages 1-2). Recent structure-based modeling and phosphoproteomics approaches have begun to delineate the details of its substrate interactions, indicating that active site conformation and the spatial arrangement of catalytic residues influence the selection of suitable substrates (widstrom2023novelsubstrateprediction pages 9-16).
5. Structure  
   TYRO3 is organized as a transmembrane receptor tyrosine kinase with distinct structural domains that mediate its function. Its extracellular region comprises two immunoglobulin-like (Ig-like) domains followed by two fibronectin type III (FNIII) domains, which are responsible for ligand binding and recognition of its vitamin K–dependent ligands GAS6 and Protein S (hsu2019tyro3apotential pages 1-2, prieto2024thetamsubfamily pages 8-9). A single transmembrane helix anchors the receptor in the plasma membrane, while the intracellular region contains a catalytic tyrosine kinase domain. This domain features the canonical bilobal architecture with an N‐terminal lobe largely composed of β‐sheets, a C‐terminal lobe predominantly helical, and a cleft between them that binds ATP (hunter2015theeukaryoticprotein pages 6-8, loris2007exploringstructureand pages 46-49). Key catalytic features include an activation loop (A-loop), which undergoes conformational changes upon autophosphorylation to enable substrate access; a conserved C-helix that plays a role in aligning catalytic residues; and a hydrophobic spine that stabilizes the active conformation of the kinase (hsu2019tyro3apotential pages 16-17, lan2000transformingactivityof pages 6-6). These structural elements are critical for the receptor’s enzymatic function and for creating docking sites upon autophosphorylation.
6. Regulation  
   Regulation of TYRO3 activity is primarily mediated through ligand binding, receptor dimerization, and subsequent autophosphorylation of tyrosine residues within its intracellular domain. Binding of its ligands—namely GAS6, Protein S, and possibly TULP1—induces conformational changes that promote receptor dimerization and trans-autophosphorylation, thereby activating the kinase (hsu2019tyro3apotential pages 1-2, prieto2024thetamsubfamily pages 12-13). These phosphorylation events generate docking sites for downstream signaling proteins such as the p85 subunit of PI3K, which further propagates survival signals (hsu2019tyro3apotential pages 12-13, lan2000transformingactivityof pages 2-2). In certain cellular contexts, TYRO3 can also be activated by cross-phosphorylation from other TAM receptors, such as AXL, adding an additional dimension to its regulation (brown2012crossphosphorylationsignalingand pages 9-10, smart2018theemergingrole pages 3-4). Post-translational modifications beyond autophosphorylation, including potential ubiquitination and interactions with specific phosphatases, contribute to the fine-tuning of its signaling, although the provided context does not offer detailed mapping of such modifications (hsu2019tyro3apotential pages 11-12).
7. Function  
   TYRO3 plays a central role in transducing extracellular signals into intracellular responses that regulate a variety of physiological processes. Upon ligand-induced activation, TYRO3 enhances PI3K activity through its interaction with PIK3R1, which in turn activates the AKT survival pathway. This cascade leads to nuclear translocation of NF-κB and the up-regulation of genes under its control, thereby promoting cell survival (hsu2019tyro3apotential pages 1-2, brown2012crossphosphorylationsignalingand pages 10-10). In neuronal tissues, TYRO3 has been implicated in protecting neurons from excitotoxic injury, supporting survival pathways that prevent cell death (hsu2019tyro3apotential pages 1-2). Moreover, TYRO3 contributes to cellular processes such as migration and differentiation, and it plays roles in platelet aggregation and cytoskeletal reorganization which are important for hemostasis and tissue remodeling (hsu2019tyro3apotential pages 16-17, morimoto2020oncogenicroleof pages 1-6). In the immune system, TYRO3 has a regulatory role as its activation can lead to the inhibition of Toll-like receptor–mediated innate immune responses through STAT1 activation, thus modulating the production of proinflammatory cytokines (hsu2019tyro3apotential pages 12-13, rothlin2014tyro3axland pages 1-2). In oncogenic settings, overexpression or constitutive activation of TYRO3 has been associated with enhanced tumor cell survival, proliferation, migration, and resistance to apoptosis, making it a potential therapeutic target in several cancers (smart2018theemergingrole pages 17-18, morimoto2020oncogenicroleof pages 14-18).
8. Other Comments  
   Various small molecule inhibitors and antibody-based strategies have been developed targeting the TAM receptor family, including TYRO3, to interfere with its signaling and thereby counteract its roles in oncogenesis and immune suppression. In preclinical studies, compounds such as MGCD516 (Sitravatinib), RXDX-106, and spiroindoline-based inhibitors have demonstrated potent activity against TAM receptors with low nanomolar IC50 values (hsu2019tyro3apotential pages 16-17, smart2018theemergingrole pages 15-17). Additionally, indirect inhibition strategies—such as using Warfarin to block vitamin K–dependent gamma-carboxylation of GAS6—can reduce ligand-mediated activation of TYRO3 (brown2012crossphosphorylationsignalingand pages 10-10). Clinically, TYRO3 overexpression has been correlated with poor prognosis in several malignancies, and its involvement in both tumor cell intrinsic survival pathways and modulation of the tumor microenvironment underlines its potential as a therapeutic target. These disease associations extend to cancer types where TYRO3 contributes to chemoresistance and metastatic spread (morimoto2020oncogenicroleof pages 1-6, smart2018theemergingrole pages 17-18). Further, the receptor’s role in the regulation of immune responses suggests that dysregulation may also contribute to autoimmune conditions, although the primary focus of the current data is on its oncogenic and survival functions.
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