1. Phylogeny  
   Receptor‐type tyrosine‐protein kinase FLT3, also known as FL cytokine receptor, Fetal liver kinase‐2, FLK2 or Stem cell tyrosine kinase 1, is a member of the class III receptor tyrosine kinase family that includes other critical hematopoietic regulators such as KIT, FMS (colony‐stimulating factor 1 receptor) and the platelet‐derived growth factor receptors (PDGFRα/β) (grafone2012anoverviewon pages 2-3, kazi2019fmsliketyrosinekinase pages 1-2). Phylogenetic analyses have demonstrated that FLT3 is evolutionarily conserved across vertebrate species, with orthologs identified in all mammals possessing a hematopoietic system; its amino acid sequence reveals high homology in the catalytic domains when compared to other family members, while extracellular regions display divergence that permits selective ligand recognition (levis2003flt3itdoesmatter pages 1-2, meshinchi2009structuralandfunctional pages 1-1). The evolutionary relationship within the kinome places FLT3 among receptors that have evolved from a common ancestral gene, which emerged early during eukaryotic evolution and gave rise to a cohort of RTKs involved in development and immune regulation (grafone2012anoverviewon pages 1-2, kazi2019fmsliketyrosinekinase pages 1-2). Thus, FLT3 is not only essential for normal hematopoietic cell signaling but also serves as a representative model for the diversification of receptor tyrosine kinases within metazoans.
2. Reaction Catalyzed  
   FLT3 catalyzes the phosphorylation of target proteins on tyrosine residues using ATP as the phosphate donor. The canonical reaction can be expressed as follows:  
     ATP + [protein]-L-tyrosine → ADP + [protein]-L-tyrosine-phosphate + H⁺  
   This enzymatic activity results in the covalent modification of specific tyrosine residues on substrates, thereby generating phosphotyrosine motifs that act as docking sites for downstream signaling proteins (kiyoi2015flt3inhibitorsrecent pages 6-9, tse2001inhibitionofflt3mediated pages 1-2). The phosphorylation reaction executed by FLT3 is central to its signaling function, initiating cascades that regulate cell proliferation, differentiation, and survival.
3. Cofactor Requirements  
   As is typical for kinases, the catalytic activity of FLT3 requires the presence of divalent cations, with magnesium (Mg²⁺) being essential for ATP binding and proper enzymatic function. The metal ion coordinates the phosphate groups of ATP and stabilizes the transition state during the phosphotransfer reaction (grafone2012anoverviewon pages 2-3, kazi2019fmsliketyrosinekinase pages 6-7). Without this cofactor, FLT3 is unable to efficiently catalyze the transfer of phosphate groups to substrate proteins.
4. Substrate Specificity  
   FLT3 displays substrate specificity consistent with that of many receptor tyrosine kinases. It preferentially catalyzes the phosphorylation of tyrosine residues on a spectrum of intracellular substrates, which include adaptor proteins and enzymes central to signal transduction. Specifically, FLT3 promotes the phosphorylation of substrates such as SHC1 and AKT1 and is involved in the activation of downstream signaling effectors including mTOR, RAS, and MAP kinases (eguchi2020mechanismsunderlyingresistance pages 1-3, grafone2012anoverviewon pages 8-9). In addition, FLT3 triggers the phosphorylation of other proteins including FES, FER, PTPN6 (SHP), PTPN11 (SHP-2), PLCG1, and STAT5A/STAT5B. Although wild-type FLT3 shows only marginal activation of STAT5A or STAT5B, mutations that lead to its constitutive activation enhance STAT5 phosphorylation considerably (eguchi2020mechanismsunderlyingresistance pages 1-3, gu2011surveyofactivated pages 1-2). The precise consensus motif recognized by FLT3 has not been fully resolved, yet pattern analysis of substrates in cells with active FLT3 indicates a preference for tyrosine residues that become part of SH2-binding motifs following phosphorylation (gu2011surveyofactivated pages 1-2, kazi2019fmsliketyrosinekinase pages 1-2).
5. Structure  
   FLT3 is organized into distinct domains that confer its unique functional properties. The protein comprises an extracellular region, a transmembrane segment, and an intracellular portion containing the regulatory and catalytic domains. The extracellular region, spanning approximately 540–550 amino acids, is composed of five immunoglobulin-like (Ig-like) domains. These domains are heavily glycosylated and are critical for ligand binding; the three most distal domains primarily mediate interaction with the FLT3 ligand (FL), whereas the two proximal Ig-like domains facilitate receptor dimerization (grafone2012anoverviewon pages 2-3, kazi2019fmsliketyrosinekinase pages 1-2). Following the extracellular segment is a 21–25 amino acid transmembrane domain that anchors FLT3 into the cell membrane. Adjacent to the transmembrane segment, the juxtamembrane (JM) domain serves an autoinhibitory role by preventing spontaneous activation of the kinase domain under basal conditions (eguchi2020mechanismsunderlyingresistance pages 1-3, meshinchi2009structuralandfunctional pages 5-6). The intracellular region features a split tyrosine kinase domain that is divided into a smaller N-terminal lobe and a larger C-terminal lobe, which together form the catalytic core. The activation loop, located within the C-terminal lobe, undergoes phosphorylation in response to receptor activation and is crucial for stabilizing the active conformation of the enzyme (zorn2015crystalstructureof pages 13-14, kazi2019fmsliketyrosinekinase pages 10-12). Additional regulatory elements include a kinase insert region and a short C-terminal tail; these regions are less well conserved but may modulate substrate interactions and downstream signaling (grafone2012anoverviewon pages 2-3, verstraete2011structuralinsightsinto pages 60-64). Structural studies, including crystallography and molecular modeling, have provided atomic-level detail of the kinase domain in both inactive (autoinhibited) and active conformations, revealing the orientation of the C-helix, the hydrophobic spines, and the configuration of the activation loop that collectively determine the catalytic activity (zorn2015crystalstructureof pages 13-14, verstraete2011structuralinsightsinto pages 60-64).
6. Regulation  
   FLT3 activity is regulated by a finely tuned interplay of ligand binding, dimerization, autophosphorylation, and dephosphorylation events. Under resting conditions, the juxtamembrane domain of FLT3 exerts an autoinhibitory effect that maintains the receptor in an inactive state. Upon binding FLT3 ligand, which is secreted or displayed on the surface of bone marrow stromal cells, FLT3 undergoes dimerization; this spatial proximity allows the kinase domains to trans-autophosphorylate specific tyrosine residues in the JM region and the activation loop, thereby relieving the autoinhibition and transitioning the receptor to an active state (eguchi2020mechanismsunderlyingresistance pages 1-3, tse2001inhibitionofflt3mediated pages 1-2). Key phosphorylation sites such as Tyr-589 and Tyr-591 have been identified as critical for recruiting downstream proteins including members of the SRC family kinases (kazi2019fmsliketyrosinekinase pages 15-16, zorn2015crystalstructureof pages 13-14). Negative regulatory mechanisms are also crucial; protein tyrosine phosphatases like DEP-1 selectively dephosphorylate FLT3 thereby attenuating its signaling output (arora2011proteintyrosinephosphatasedep1 pages 12-13). In addition, receptor turnover is controlled by ubiquitination and proteasomal degradation processes that ensure the temporal regulation of FLT3 signaling (gu2011surveyofactivated pages 9-10). Aberrant regulation can occur via mutations—most notably internal tandem duplications (ITDs) in the juxtamembrane domain and point mutations in the kinase domain (such as D835 mutations)—which disrupt the autoinhibitory conformation and lead to constitutive activation of the receptor independent of ligand binding (eguchi2020mechanismsunderlyingresistance pages 1-3, kazi2019fmsliketyrosinekinase pages 18-19). These mutant forms result in persistent downstream signaling that promotes cell proliferation and survival, contributing to the pathogenesis of acute myeloid leukemia (AML).
7. Function  
   FLT3 plays an essential role in normal hematopoiesis by regulating the survival, proliferation, and differentiation of early hematopoietic progenitor cells and dendritic cell subsets. Its expression is predominantly restricted to CD34-positive hematopoietic stem and progenitor cells, where it mediates responses to the FLT3 ligand (FL) produced by the bone marrow microenvironment (eguchi2020mechanismsunderlyingresistance pages 1-3, kazi2019fmsliketyrosinekinase pages 1-2). Upon activation, FLT3 initiates multiple intracellular signaling cascades. The PI3K/AKT/mTOR pathway is engaged through recruitment of adaptor proteins that facilitate AKT activation, thereby promoting cellular survival and metabolic activity (eguchi2020mechanismsunderlyingresistance pages 1-3, kazi2019fmsliketyrosinekinase pages 14-15). Concomitantly, the RAS/RAF/MEK/ERK pathway is activated, driving cell proliferation and gene expression changes relevant to cell cycle progression (grafone2012anoverviewon pages 8-9, gu2011surveyofactivated pages 2-3). Although wild-type FLT3 causes only modest activation of STAT5 under physiological conditions, mutations that render the receptor constitutively active significantly amplify STAT5 phosphorylation, thereby promoting the expression of genes that confer resistance to apoptosis (eguchi2020mechanismsunderlyingresistance pages 1-3, kazi2019fmsliketyrosinekinase pages 18-19). In addition to these core pathways, FLT3 activation influences the phosphorylation state of proteins involved in cell adhesion, cytoskeletal reorganization, and even epigenetic regulators, further underscoring its central role in maintaining hematopoietic cell function (gu2011surveyofactivated pages 1-2, kazi2019fmsliketyrosinekinase pages 10-12). In summary, FLT3 is indispensable for normal bone marrow homeostasis; however, in the context of activating mutations, its deregulated signaling drives leukemic transformation and sustains the malignant phenotype observed in AML (grafone2012anoverviewon pages 8-9, levis2005flt3tyrosinekinase pages 1-2).
8. Other Comments  
   Due to its critical function in hematopoietic regulation and its frequent mutation in AML, FLT3 has emerged as a prominent therapeutic target. A number of small-molecule inhibitors have been developed against FLT3, including first-generation agents such as midostaurin, sorafenib, quizartinib, and second-generation inhibitors such as gilteritinib and crenolanib (kiyoi2015flt3inhibitorsrecent pages 6-9, larrosagarcia2017flt3inhibitorsin pages 1-3, grunwald2013flt3inhibitorsfor pages 1-2). These inhibitors disrupt FLT3-mediated phosphorylation and downstream signaling pathways, resulting in decreased proliferation and induced apoptosis in leukemic cells. However, the clinical application of FLT3 inhibitors is challenged by the development of resistance, which can arise through secondary mutations in the kinase domain (for example, mutations at D835 and F691L) or by compensatory activation of parallel survival pathways (tse2001inhibitionofflt3mediated pages 8-9, wander2014theevolvingrole pages 13-13). In addition, FLT3 signaling is influenced by microenvironmental factors such as the availability of its ligand, which can modulate inhibitor efficacy; for instance, high levels of FL can reduce the potency of FLT3 inhibitors by stimulating receptor dimerization and compensatory signaling (eguchi2020mechanismsunderlyingresistance pages 1-3, kazi2019fmsliketyrosinekinase pages 22-23). The broad substrate specificity and participation in multiple signaling pathways also raise the possibility for combination therapies aimed at targeting FLT3 in conjunction with downstream effectors such as mTOR or MAP kinases to improve therapeutic outcomes. The clinical relevance of FLT3 is further underscored by its association with adverse prognostic features in AML, where FLT3-ITD and kinase domain point mutations serve as both biomarkers and drivers of leukemogenesis (levis2003flt3itdoesmatter pages 11-11, gu2011surveyofactivated pages 9-10). Collectively, these features make FLT3 an attractive, albeit complex, target for therapeutic intervention in hematologic malignancies.
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