1. Phylogeny  
   The hepatocyte growth factor receptor, commonly known as MET (Uniprot ID: P08581), is a member of the receptor tyrosine kinase (RTK) superfamily that is evolutionarily conserved across vertebrates (woude2000metreceptortyrosine pages 1-2). MET orthologs have been identified in mammals, birds, amphibians, and fish, and its sequence conservation underscores its critical role in developmental and physiological processes (zhang2018functionofthe pages 1-3). Within the kinome, MET clusters with other RTKs that mediate growth and differentiation signals, and it shares a close evolutionary relationship with receptors such as RON, which together form a distinct subfamily implicated in epithelial‐mesenchymal interactions (woude2000metreceptortyrosine pages 2-3, benvenuti2007themetreceptor pages 9-9). Phylogenetic analyses based on kinase domain sequences and domain organization have placed MET within the group of transmembrane tyrosine kinases that originated in early vertebrates, indicating that the receptor has been part of an evolutionarily ancient signaling system (woude2000metreceptortyrosine pages 1-2). Furthermore, the modular organization of MET—with its extracellular ligand–binding domains and intracellular catalytic region—parallels the structure of many other RTKs that evolved by gene duplication and diversification from a common ancestral kinase (gherardi2003functionalmapand pages 1-2).
2. Reaction Catalyzed  
   MET functions as a protein tyrosine kinase, catalyzing the transfer of a phosphate group from ATP to specific tyrosine residues on substrate proteins (benvenuti2007themetreceptor pages 1-2). The reaction can be represented in a general form as follows: ATP + [protein] – L-tyrosine → ADP + [protein] – L-tyrosine-phosphate + H⁺ (template, see standard kinase reaction). This phosphorylation event is central to signal propagation and creates docking sites for adaptor proteins that contain SH2 domains, thereby linking MET activation to downstream signaling cascades (chen2006directinteractionof pages 1-2). Autophosphorylation of MET itself occurs on critical tyrosine residues within the activation loop and C-terminal tail, events that are prerequisite for full catalytic activation (cui2011structurebaseddrug pages 19-20).
3. Cofactor Requirements  
   The catalytic activity of MET, like that of most protein kinases, is dependent on the presence of divalent metal ions. In particular, Mg²⁺ is essential for stabilizing the binding of ATP in the active site and facilitating the phosphotransfer reaction (cui2011structurebaseddrug pages 1-2).
4. Substrate Specificity  
   MET phosphorylates tyrosine residues on selected substrate proteins that are key to mediating its diverse biological functions. Although an exact consensus motif is not fully delineated in the provided reports, MET’s intrinsic substrate specificity is oriented toward generating phosphotyrosine sites that serve as recruitment platforms for signaling adapters such as GAB1, GRB2, SRC, PLCG1, and STAT3 (benvenuti2007themetreceptor pages 2-3, danilkovitchmiagkova2002dysregulationofmet pages 3-4). In this context, MET-dependent phosphorylation events create docking sites that are recognized by SH2 and phosphotyrosine-binding domains, thereby linking receptor activation to downstream effectors in the RAS-ERK and PI3K-AKT cascades (benvenuti2007themetreceptor pages 9-9, zhang2018functionofthe pages 1-3).
5. Structure  
   MET is synthesized as a single-chain precursor that is proteolytically processed into a disulfide-linked heterodimer composed of an extracellular α-chain (approximately 50 kDa) and a transmembrane β-chain (approximately 145 kDa) (benvenuti2007themetreceptor pages 1-2). The extracellular region of MET is composed of several distinct domains: a Sema domain that mediates initial ligand binding, a cysteine-rich Met-related sequence (MRS) domain, and four immunoglobulin-like IPT (immunoglobulin-plexin-transcription factor) domains, each contributing to high-affinity binding of the ligand hepatocyte growth factor (HGF) (benvenuti2007themetreceptor pages 1-2, gherardi2003functionalmapand pages 3-4). Following the extracellular segment is a single transmembrane helix that anchors MET in the cell membrane, and the intracellular portion consists of a juxtamembrane region that contains regulatory phosphorylation sites, a catalytic tyrosine kinase domain responsible for enzymatic activity, and a C-terminal tail with multiple tyrosine residues that form the basis of a multifunctional docking site for downstream effectors (benvenuti2007themetreceptor pages 1-2, chen2006directinteractionof pages 1-2). Within the kinase domain, key structural features such as the activation loop, the C-helix, and the hydrophobic spine are critical for catalytic function and proper regulation; for example, phosphorylation of tyrosine residues in the activation loop is necessary for conformational changes that permit full kinase activity (cui2011structurebaseddrug pages 19-20, chen2006directinteractionof pages 1-2). Structural models and experimental evidence, including crystallographic studies and homology modeling, have confirmed that MET’s overall fold is typical of receptor tyrosine kinases, further supporting its classification within this enzyme family (gherardi2003functionalmapand pages 4-5, holmes2007insightsintothe pages 8-10).
6. Regulation  
   Regulation of MET activity is achieved by multiple, interrelated mechanisms that ensure appropriate signal intensity and duration. Ligand binding is the primary mode of regulation; HGF, the activating ligand for MET, is secreted as an inactive single-chain precursor and requires proteolytic cleavage by enzymes such as urokinase-type plasminogen activator, coagulation factor XII, and thrombin to generate the biologically active two-chain form (benvenuti2007themetreceptor pages 1-2, eder2009noveltherapeuticinhibitors pages 1-2). Upon ligand engagement, MET undergoes receptor dimerization and autophosphorylation on critical tyrosine residues within both the activation loop and the C-terminal tail (chen2006directinteractionof pages 1-2, cui2011structurebaseddrug pages 19-20). These phosphorylation events serve as molecular switches that promote the docking of a variety of adaptor proteins and substrates, thereby linking MET activation to downstream signaling pathways (benvenuti2007themetreceptor pages 2-3). In addition to autophosphorylation, MET is regulated by its juxtamembrane domain, which contains sites that, when phosphorylated, modulate receptor internalization and degradation via recruitment of ubiquitin ligases such as CBL (benvenuti2007themetreceptor pages 9-9, chen2006directinteractionof pages 12-13). Crosstalk with other cell surface receptors, including RON, EGFR, and various integrins, provides an additional layer of regulation by influencing MET phosphorylation status and cellular localization (benvenuti2007themetreceptor pages 8-9, danilkovitchmiagkova2002dysregulationofmet pages 4-5). These regulatory mechanisms are critical for maintaining the balance between physiological signal transduction and the pathological activation associated with cancer (eder2009noveltherapeuticinhibitors pages 6-7).
7. Function  
   MET functions as a key transducer of extracellular signals into intracellular responses that regulate a multitude of biological processes. Activation of MET by HGF initiates a complex cascade of signaling events that promote cellular processes such as proliferation, scattering, morphogenesis, and survival (benvenuti2007themetreceptor pages 1-2, eder2009noveltherapeuticinhibitors pages 1-2). Upon ligand binding, autophosphorylation of MET creates specific docking sites that recruit a range of intracellular effectors—including the PI3-kinase regulatory subunit PIK3R1, PLCG1, SRC, GRB2, STAT3, and the multifunctional adapter protein GAB1—which further propagate signals through key pathways such as the RAS-ERK cascade and the PI3K-AKT pathway (benvenuti2007themetreceptor pages 2-3, zhang2018functionofthe pages 1-3). The RAS-ERK pathway is principally associated with the morphogenetic effects of MET, including cellular reorganization and epithelial-to-mesenchymal transition, whereas the PI3K-AKT pathway mainly confers prosurvival signals that protect cells from apoptosis (benvenuti2007themetreceptor pages 8-8, zhang2018functionofthe pages 14-14). During embryonic development, MET signaling plays an essential role in gastrulation, neuronal precursor migration, angiogenesis, and kidney formation, while in adult tissues, MET contributes to wound healing and tissue regeneration (benvenuti2007themetreceptor pages 1-2, zhang2018functionofthe pages 1-3). In the context of cancer, aberrant activation of MET—whether by overexpression, gene amplification, or activating point mutations such as M1268T—leads to enhanced oncogenic processes including increased cellular motility, invasive growth, and metastasis (danilkovitchmiagkova2002dysregulationofmet pages 3-4, benvenuti2007themetreceptor pages 6-7). Moreover, MET-dependent signaling can confer resistance to conventional therapies, an effect that underscores its clinical relevance in tumors such as non-small cell lung cancer, gastric carcinoma, and papillary renal carcinoma (zhang2018functionofthe pages 13-14, eder2009noveltherapeuticinhibitors pages 3-5). Interactions with cell adhesion molecules, such as integrins and CD44, further modulate MET’s function by linking extracellular matrix adhesion to intracellular kinase signaling (benvenuti2007themetreceptor pages 4-5, chen2006directinteractionof pages 12-12). Collectively, MET’s ability to orchestrate both developmental and oncogenic signaling pathways makes it a central regulator of cellular behavior in both normal and pathological settings (eder2009noveltherapeuticinhibitors pages 6-6, fan2001themultisubstrateadapter pages 1-2).
8. Other Comments  
   A broad spectrum of therapeutic strategies targeting MET has been developed in response to its pivotal role in cancer progression. These include ligand antagonists such as anti-HGF monoclonal antibodies and engineered decoy receptors that block HGF binding without eliciting receptor activation (benvenuti2007themetreceptor pages 5-6, eder2009noveltherapeuticinhibitors pages 1-2). In addition, receptor competitors—including antibodies that promote receptor shedding, such as the DN30 antibody—have been employed to downregulate MET surface expression (benvenuti2007themetreceptor pages 6-7). Small molecule tyrosine kinase inhibitors, such as PHA-665752, SU11274, crizotinib, tivantinib, and cabozantinib, have demonstrated efficacy in preclinical and clinical studies by selectively targeting MET’s ATP-binding site and inhibiting its kinase activity (eder2009noveltherapeuticinhibitors pages 3-5, cui2011structurebaseddrug pages 20-21, zhang2018functionofthe pages 13-14). Moreover, MET is often a component of oncogene addiction in tumors with MET overexpression or gene amplification, making it a rational target for personalized cancer therapy (benvenuti2007themetreceptor pages 7-8, danilkovitchmiagkova2002dysregulationofmet pages 3-4). MET signaling also cooperates with other receptor tyrosine kinases, notably EGFR and RON, which contributes to drug resistance and necessitates combination therapeutic strategies in certain tumor types (zhang2018functionofthe pages 5-6, eder2009noveltherapeuticinhibitors pages 6-6). In experimental settings, RNA interference approaches have been utilized to silence MET expression and attenuate its downstream oncogenic signaling, further validating its role as a driver of tumor invasiveness and metastasis (benvenuti2007themetreceptor pages 8-9). The clinical relevance of MET is underscored by the identification of multiple activating mutations—such as the M1268T mutation—that enhance kinase activity and alter substrate specificity, thereby contributing to aggressive cancer phenotypes (danilkovitchmiagkova2002dysregulationofmet pages 3-4). Finally, structure‐based drug design efforts have refined our understanding of MET’s catalytic pocket and facilitated the development of novel inhibitors with improved selectivity and potency against MET-driven cancers (cui2011structurebaseddrug pages 20-21, zhang2018functionofthe pages 13-14).
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