1. Phylogeny  
   BRAF is a member of the RAF kinase family, which also includes ARAF and CRAF, and is classified among the serine/threonine protein kinases that are evolutionarily conserved across eukaryotic species. Orthologs of BRAF are found in vertebrates—including mammals, birds, and fish—reflecting its essential role in transmitting mitogenic signals that date back to early eukaryotes (gunderwala2022mechanismandinhibition pages 1-3, agianian2018currentinsightsof pages 1-1). Its domain structure, particularly the highly conserved kinase domain, situates BRAF within the MAP kinase kinase kinase (MAP3K) subgroup, which is critical in evolutionary adaptations that control cellular proliferation and differentiation (agianian2018currentinsightsof pages 1-1).
2. Reaction Catalyzed  
   BRAF catalyzes the transfer of a phosphate group from ATP to specific serine/threonine residues on substrate proteins, following the general kinase reaction: ATP + [protein]-(L-serine or L-threonine) → ADP + [protein]-(L-serine/threonine)-phosphate + H⁺ (agianian2018currentinsightsof pages 1-3, xie2009thecrystalstructure pages 1-2). In the canonical MAP kinase pathway, phosphorylation of MAP2K isoforms (MEK1/MEK2) by BRAF is a key step in activating downstream ERK signaling (agianian2018currentinsightsof pages 1-3).
3. Cofactor Requirements  
   The kinase activity of BRAF depends on the presence of divalent metal ions, with Mg²⁺ being the critical cofactor required to coordinate ATP binding and facilitate the phosphoryl transfer reaction (xie2009thecrystalstructure pages 1-2).
4. Substrate Specificity  
   BRAF exhibits substrate specificity primarily toward members of the MAP2K family, such as MEK1 and MEK2, which are phosphorylated on serine/threonine residues located in their activation loops (agianian2018currentinsightsof pages 1-3, xing2007brafmutationin pages 1-2). Although a unique consensus motif has not been universally defined for BRAF substrates, the substrates commonly feature docking sites that enable efficient phosphoryl transfer. In addition to MEK proteins, BRAF has been reported to phosphorylate PFKFB2, indicating an expansion of its substrate repertoire into metabolic regulation (gunderwala2022mechanismandinhibition pages 1-3).
5. Structure  
   BRAF is organized into a modular architecture consisting of an N-terminal regulatory region and a C-terminal kinase domain. The N-terminal portion includes the RAS-binding domain (RBD) and a cysteine-rich domain (CRD); the RBD facilitates interaction with active, GTP-bound RAS, while the CRD assists in membrane association and contributes to autoinhibition (fiesco2022structuralinsightsinto pages 1-2, park2019architectureofautoinhibited pages 1-2). The C-terminal kinase domain is arranged in a bilobal structure, with a smaller N-lobe predominantly formed by β-sheets and a larger C-lobe rich in α-helices. The interlobe cleft contains the ATP-binding pocket, where key residues such as Lys483 and Glu501 mediate ATP coordination and catalysis (xie2009thecrystalstructure pages 8-10, park2019architectureofautoinhibited pages 32-34). A central regulatory feature is the activation loop (A-loop), whose conformation—ranging from a disordered, inactive state with an “αC-out” helix to an ordered, active state with the “αC-in” alignment—is critical for substrate access and catalysis (agianian2018currentinsightsof pages 5-6, park2019architectureofautoinhibited pages 34-37). The V600 residue, located within the activation loop, is a determinant of the kinase’s conformation; its mutation to glutamate (V600E) disrupts the autoinhibited state by mimicking constitutive phosphorylation, thereby promoting persistent kinase activation (agianian2018currentinsightsof pages 16-16, xing2007brafmutationin pages 12-13, xie2009thecrystalstructure pages 8-10). Structural studies employing techniques such as X-ray crystallography and cryo-electron microscopy have revealed that BRAF can form dimers, with the dimeric interface mediated by precisely aligned catalytic spines and interactions that are further stabilized by binding of regulatory proteins such as 14-3-3 to phosphorylated sites (park2019architectureofautoinhibited pages 7-9, fiesco2022structuralinsightsinto pages 12-13, park2019architectureofautoinhibited pages 25-30).
6. Regulation  
   BRAF activity is modulated by multiple regulatory mechanisms that include post-translational modifications and protein–protein interactions. Phosphorylation is a crucial regulatory event; residues within the activation loop, such as T599 and S602, are phosphorylated under physiological conditions to enable conformational transitions that permit efficient substrate binding and catalysis (agianian2018currentinsightsof pages 16-16, xing2007brafmutationin pages 12-13). In oncogenic contexts, the V600E mutation substitutes valine with glutamate, thereby mimicking the phosphorylated state and inducing constitutive activation (xie2009thecrystalstructure pages 8-10, xing2007brafmutationin pages 12-13). In addition, BRAF is regulated by its interaction with 14-3-3 proteins, which bind to phosphorylated serine residues (notably pS365 and pS729) and either maintain the kinase in an autoinhibited monomeric state or facilitate its dimerization and subsequent activation, depending on the cellular context (park2019architectureofautoinhibited pages 25-30, fiesco2022structuralinsightsinto pages 2-4). Binding of GTP-bound RAS to the RBD disrupts autoinhibitory interactions and promotes translocation of BRAF to the plasma membrane, where it can form active dimers (fiesco2022structuralinsightsinto pages 1-2, park2019architectureofautoinhibited pages 1-2). Moreover, feedback mechanisms mediated by downstream kinases such as ERK can provide negative regulation, while ubiquitination pathways—although BRAF is known to interact with components such as FBXW7—do not solely determine its stability (hernandez2016regulationofbraf pages 15-19, hernandez2016regulationofbraf pages 30-33). Allosteric regulation via homodimer and heterodimer formation is critical for modulating catalytic efficiency and substrate specificity (gunderwala2022mechanismandinhibition pages 7-10, park2019architectureofautoinhibited pages 7-9).
7. Function  
   BRAF functions as a central signal transducer in the MAP kinase pathway. Upon activation by GTP-bound RAS, BRAF phosphorylates its primary substrates MEK1/MEK2, which in turn phosphorylate ERK1/ERK2, thereby propagating mitogenic and survival signals that ultimately influence gene expression in the nucleus (agianian2018currentinsightsof pages 1-3, park2019architectureofautoinhibited pages 1-2). In addition to its canonical role in cell proliferation and differentiation, BRAF has been implicated in the regulation of cellular metabolism through the phosphorylation of proteins such as PFKFB2, linking extracellular signals to metabolic reprogramming (gunderwala2022mechanismandinhibition pages 1-3). BRAF is expressed in a variety of tissues and is particularly prominent in cells where tight control of growth and survival is essential. Its dysregulation, most notably through mutations like V600E, results in constitutive kinase activity that drives oncogenic transformation and is associated with an array of cancers, including melanoma, thyroid cancer, and colorectal cancer (xing2007brafmutationin pages 12-13, rock2019brafinhibitorspromote pages 16-16). Through these roles, BRAF acts as an essential molecular switch that integrates upstream mitogenic signals with downstream transcriptional programs critical for cell fate determination (agianian2018currentinsightsof pages 1-3).
8. Other Comments  
   Multiple small-molecule inhibitors have been developed to target BRAF, particularly in its mutant forms such as BRAFV600E. ATP-competitive inhibitors like vemurafenib, dabrafenib, and encorafenib have demonstrated clinical efficacy in treating cancers harboring these mutations, although challenges such as inhibitor-induced paradoxical activation via dimer formation have emerged (xie2009thecrystalstructure pages 8-10, rock2019brafinhibitorspromote pages 2-2). Structural studies employing organoruthenium inhibitors have further elucidated the active conformation of BRAF and informed the design of next-generation inhibitors aimed at overcoming resistance mechanisms (xie2009thecrystalstructure pages 8-10, xie2009thecrystalstructure pages 10-11). BRAF mutations, particularly V600E, serve as important biomarkers for diagnostic and prognostic purposes and are a key focus of precision oncology. The integration of BRAF mutational status with comprehensive molecular profiling is critical for tailoring therapeutic strategies in a tumor-agnostic setting (xing2007brafmutationin pages 12-13, OpenTargets Search: -BRAF). Overall, the extensive research on BRAF has provided deep insights into its regulation, structural dynamics, functional roles in signal transduction, and implications in oncogenesis, forming the basis for ongoing therapeutic development efforts (gunderwala2022mechanismandinhibition pages 7-10, park2019architectureofautoinhibited pages 7-9).
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