1. Phylogeny:  
   IRAK3 (also known as IRAK‐M; UniProt ID Q9Y616) is a member of the interleukin‐1 receptor-associated kinase (IRAK) family, which belongs to the serine/threonine protein kinase superfamily. Within the human kinome, IRAK3 is grouped with other IRAK family members such as IRAK-1, IRAK-2, and IRAK-4, and it can be traced back to a common ancestral kinase found in early eukaryotes (Manning2002, Manning2002). Orthologs of IRAK3 are conserved across mammalian species, and its expression patterns are predominantly restricted to cells of the myeloid lineage, including monocytes and macrophages (flannery2010theinterleukin1receptorassociated pages 5-9, gan2006regulationsandroles pages 7-8). Phylogenetic analyses indicate that while the IRAK family shares a conserved death domain and central kinase domain, IRAK3 has diverged functionally from its catalytically active homologs, adopting a regulatory rather than an activating role in innate immune receptor signaling (janssens2003functionaldiversityand pages 1-2, janssens2003functionaldiversityand pages 2-3).
2. Reaction Catalyzed:  
   As a member of the serine/threonine kinase family, the archetypal reaction catalyzed by active kinases involves the transfer of the γ-phosphate from ATP to a serine or threonine residue on a protein substrate, thereby converting ATP into ADP alongside the phosphorylated protein and a proton. However, IRAK3 is characterized as a putative inactive kinase that does not exhibit measurable catalytic activity due to the absence of key catalytic residues; consequently, no productive phosphorylation reaction has been observed for IRAK3 (li2005il1receptor–associatedkinase pages 1-2, flannery2010theinterleukin1receptorassociated pages 16-20).
3. Cofactor Requirements:  
   Active protein kinases in the serine/threonine class typically require divalent metal ions such as Mg²⁺ as cofactors to coordinate ATP binding and catalysis. Although IRAK3 is structurally related to these kinases, its lack of catalytic function obviates the requirement for such cofactors in any measurable enzymatic reaction (li2005il1receptor–associatedkinase pages 1-2, ringwood2008theinvolvementof pages 6-8).
4. Substrate Specificity:  
   In catalytically active serine/threonine kinases, substrate recognition is typically governed by consensus motifs that direct phosphorylation to specific serine or threonine residues, with one atlas of substrate specificities reporting a preference for motifs resembling RxRxxp[ST] (Johnson2023 pages 759-766). For tyrosine kinases, substrate selectivity is determined by different amino acid preferences as outlined by studies of the tyrosine kinome (Yaron-Barir2024 pages 1174-1181). However, given that IRAK3 is classified as a pseudokinase and lacks measurable catalytic activity, it does not phosphorylate any substrates. Instead, its functional role in innate immune signaling is mediated through modulation of protein–protein interactions within receptor complexes rather than through enzymatic transfer of a phosphate group (li2005il1receptor–associatedkinase pages 1-2, flannery2010theinterleukin1receptorassociated pages 16-20).
5. Structure:  
   IRAK3 is composed of 596 amino acids corresponding to an approximate molecular weight of 68 kDa. The protein’s domain architecture includes an N-terminal death domain (DD) that is critical for mediating interactions with other members of the receptor complex such as the adaptor protein MyD88, and a central kinase-like domain that, despite retaining the overall fold characteristic of serine/threonine kinases, lacks the conserved catalytic residues necessary for phosphotransfer activity (flannery2010theinterleukin1receptorassociated pages 16-20, li2005il1receptor–associatedkinase pages 1-2). In addition, IRAK3 contains a C-terminal region that includes a TRAF6-binding motif, which is important for its role in modulating downstream signaling events (flannery2010theinterleukin1receptorassociated pages 43-47). Structural studies based on crystallographic data and AlphaFold models indicate that while IRAK3 shares the bilobal structure seen in active kinases—with an N-terminal lobe comprised predominantly of β-sheets and a C-terminal lobe largely α-helical—the activation loop, hydrophobic spine, and C-helix are not configured in a conformation that supports catalytic activity (ringwood2008theinvolvementof pages 6-8, janssens2003functionaldiversityand pages 2-3).
6. Regulation:  
   Regulation of IRAK3 occurs primarily at the level of protein expression and protein–protein interactions rather than through conventional post-translational modifications that alter enzymatic activity. Expression of IRAK3 is induced in monocytes and macrophages upon exposure to pathogen-associated molecular patterns such as lipopolysaccharide (LPS), contributing to the development of endotoxin tolerance (gan2006regulationsandroles pages 3-5, nguyen2020analysisofinterleukin1 pages 1-2). Although IRAK3 does not catalyze phosphorylation reactions, its regulatory function is thought to involve either the inhibition of phosphorylation of IRAK1 and IRAK4 or the stabilization of the receptor complex, thereby preventing the dissociation of these kinases (flannery2010theinterleukin1receptorassociated pages 16-20, li2005il1receptor–associatedkinase pages 1-2). In this capacity, IRAK3 modulates the amplitude and duration of downstream NF-κB and MAP kinase activation without itself undergoing extensive phosphorylation or ubiquitination events that are typical of active kinases (gan2006regulationsandroles pages 7-8, su2009theinterleukin1receptorassociated pages 9-10).
7. Function:  
   IRAK3 functions as a negative regulator within the innate immune signaling cascade that is activated by the interleukin-1 receptor (IL1R) and Toll-like receptors (TLRs). It inhibits excessive inflammatory responses by preventing the dissociation of IRAK1 and IRAK4 from the receptor complex, thereby attenuating downstream activation of NF-κB and mitigating the production of pro-inflammatory cytokines (flannery2010theinterleukin1receptorassociated pages 16-20, ringwood2008theinvolvementof pages 5-6). In addition to its established inhibitory role, under conditions of IL33-induced lung inflammation, IRAK3 has been reported to positively regulate the expression of mRNAs encoding IL6, CSF3, CXCL2, and CCL5 in dendritic cells, highlighting a context-dependent function that can influence cytokine profiles (information section, nguyen2020analysisofinterleukin1 pages 24-25). Expression of IRAK3 is largely restricted to cells of the myeloid lineage, particularly monocytes and macrophages, where its inducible expression contributes to immune homeostasis and the prevention of hyperinflammatory states, such as those observed during endotoxin tolerance in sepsis (flannery2010theinterleukin1receptorassociated pages 5-9, gan2006regulationsandroles pages 3-5).
8. Other Comments:  
   IRAK3 represents a potential therapeutic target for modulating inflammatory and immune-related diseases given its role as a checkpoint in TLR/IL-1R signaling. Although specific small-molecule inhibitors targeting IRAK3 have not been developed to date, its regulatory function in stabilizing receptor complexes and inhibiting the dissociation of IRAK1 and IRAK4 suggests that strategies aimed at modulating its protein–protein interactions may have therapeutic merit (flannery2010theinterleukin1receptorassociated pages 16-20, song2009thekinaseactivities pages 9-9). Dysregulation of IRAK3 has been implicated in conditions characterized by excessive or uncontrolled inflammation, including sepsis and chronic inflammatory disorders, emphasizing the clinical significance of maintaining proper IRAK3 function (gan2006regulationsandroles pages 3-5, ringwood2008theinvolvementof pages 5-6). Additionally, studies of other IRAK family members, such as IRAK1 and IRAK4, provide a framework for understanding how IRAK3 may be leveraged in therapeutic contexts despite its pseudokinase status (singer2018inhibitionofinterleukin1 pages 1-2, su2009theinterleukin1receptorassociated pages 1-2).
9. References:  
   flannery2010theinterleukin1receptorassociated pages 16-20; flannery2010theinterleukin1receptorassociated pages 5-9; gan2006regulationsandroles pages 3-5; gan2006regulationsandroles pages 7-8; janssens2003functionaldiversityand pages 1-2; janssens2003functionaldiversityand pages 2-3; li2005il1receptor–associatedkinase pages 1-2; martin2001interleukin1receptorassociatedkinase1 pages 2-4; nguyen2020analysisofinterleukin1 pages 1-2; nguyen2020analysisofinterleukin1 pages 23-24; nguyen2020analysisofinterleukin1 pages 24-25; patra2016recentprogressin pages 1-3; patra2016recentprogressin pages 12-13; ringwood2008theinvolvementof pages 1-2; ringwood2008theinvolvementof pages 10-13; ringwood2008theinvolvementof pages 4-5; ringwood2008theinvolvementof pages 5-6; ringwood2008theinvolvementof pages 6-8; singer2018inhibitionofinterleukin1 pages 1-2; singer2018inhibitionofinterleukin1 pages 18-19; song2009thekinaseactivities pages 9-9; su2009theinterleukin1receptorassociated pages 1-2; su2009theinterleukin1receptorassociated pages 4-5; su2009theinterleukin1receptorassociated pages 9-10; takaesu2001interleukin1(il1)receptorassociated pages 10-10; zhou2013irak‐mmediatestoll‐like pages 1-2; zhou2013irak‐mmediatestoll‐like pages 2-3; bahia2015interleukin1receptorassociated pages 3-4; bahia2015interleukin1receptorassociated pages 1-2; bahia2015interleukin1receptorassociated pages 11-11; bahia2015interleukin1receptorassociated pages 2-3; bahia2015interleukin1receptorassociated pages 8-11; hu2002regulationofil1 pages 1-2; kollewe2004sequentialautophosphorylationsteps pages 1-1; su2007differentialregulationof pages 6-6; Johnson2023 (An atlas of substrate specificities for the human serine/threonine kinome, Nature 613(7945), 759-766); Yaron-Barir2024 (The intrinsic substrate specificity of the human tyrosine kinome, Nature 629(8014), 1174-1181); Manning, G. et al. (2002, Science 298(5600), 1912-1934); Manning, G. et al. (2002, Trends in Biochemical Sciences 27(10), 514-520).

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