1. Phylogeny  
   Human IRE1 (also known as ERN1 or Ire1‐α) is an evolutionarily conserved serine/threonine kinase/endoribonuclease present in all eukaryotic species, with the yeast ortholog Ire1p representing its earliest studied homolog. (korennykh2009theunfoldedprotein pages 5-7) It is classified within a core group of endoplasmic reticulum (ER) stress sensors that also includes PERK and ATF6, and its conservation across species underscores its central role in the unfolded protein response (UPR). (lee2008structureofthe pages 1-2) Phylogenetic analyses indicate that IRE1 belongs to an ancient family of transmembrane kinases that likely originated in the Last Eukaryotic Common Ancestor (LECA) and has been maintained due to its essential function in proteostasis. (riaz2020roleofendoplasmic pages 1-3, zhou2021inositolrequiringenzyme pages 1-2)
2. Reaction Catalyzed  
   IRE1 exerts dual enzymatic activity by first catalyzing its own autophosphorylation via transfer of the γ‐phosphate from ATP to serine/threonine residues within its activation segment, converting ATP to ADP and thereby initiating its activation. (ali2011structureofthe pages 1-2) In its active state, IRE1’s endoribonuclease domain specifically cleaves XBP1 mRNA by excising a 26‐nucleotide intron from the unspliced transcript, a reaction that does not require canonical spliceosomal machinery and leads to the generation of the spliced mRNA encoding the potent transcription factor XBP1s. (ali2011structureofthe pages 2-3) Additionally, IRE1 catalyzes the regulated IRE1‐dependent decay (RIDD) of a subset of ER‐associated mRNAs, thereby reducing the protein load entering the ER under stress conditions. (bartoszewska2023dualrnaseactivity pages 7-9)
3. Cofactor Requirements  
   The kinase activity of IRE1 is strictly dependent on the binding of ATP and the chelation of Mg²⁺ ions, which are required to enable the phosphotransfer reaction necessary for autophosphorylation. (carlesso2018bindinganalysisof pages 1-2) Mg²⁺ ions serve to stabilize the transition state and facilitate nucleotide binding within the catalytic cleft of the kinase domain, thereby ensuring effective phosphorylation. (riaz2020roleofendoplasmic pages 3-4) Moreover, the presence of ADP following autophosphorylation promotes conformational changes that further support the formation of an active RNase dimer, integral to IRE1’s downstream RNA cleavage function. (lee2008structureofthe pages 1-2, concha2015longrangeinhibitorinducedconformational pages 2-3)
4. Substrate Specificity  
   The endoribonuclease activity of IRE1 exhibits remarkable substrate specificity for mRNA substrates harboring conserved stem-loop structures, most notably its exclusive cleavage of XBP1 mRNA at two specific sites that flank a 26-nucleotide intron. (ali2011structureofthe pages 2-3) The recognition of these RNA stem-loop motifs is mediated by the RNase domain’s complementary binding surface, which accommodates the unique geometry and sequence of the XBP1 stem loops. (lee2008structureofthe pages 9-11) In addition to XBP1 mRNA, IRE1 can degrade a limited set of ER-associated transcripts via RIDD, wherein the target mRNAs also typically harbor secondary structures that are recognized by the active site of the RNase domain. (bartoszewska2023dualrnaseactivity pages 7-9)
5. Structure  
   Human IRE1 is organized into a modular architecture comprising an N-terminal luminal domain, a single-span transmembrane helix, and a cytosolic region that houses a serine/threonine kinase domain immediately followed by a specialized endoribonuclease (KEN) domain. (ali2011structureofthe pages 1-2) The luminal domain is responsible for sensing misfolded proteins within the ER lumen and, under unstressed conditions, is maintained in a monomeric inactive state through binding to the chaperone HSPA5/BiP. (riaz2020roleofendoplasmic pages 1-3) Upon ER stress, dissociation of BiP permits homodimerization of the luminal domains, which is transmitted via the transmembrane segment to the cytosolic region to trigger dimerization and oligomerization of the kinase/RNase domains. (ali2011structureofthe pages 1-2, riaz2020roleofendoplasmic pages 4-6) Structural studies have revealed that in its dephosphorylated state, the cytosolic segment forms a “face-to-face” dimer that is competent for kinase trans-autophosphorylation, whereas phosphorylation induces conformational shifts that stabilize a “back-to-back” dimer configuration necessary for RNase activity. (lee2008structureofthe pages 1-2, ali2011structureofthe pages 11-12) The kinase domain itself is bi-lobal, consisting of an N-terminal lobe dominated by β-strands and a C-terminal lobe rich in α-helices, with the nucleotide-binding cleft and activation segment playing crucial roles in catalysis and conformational regulation. (lee2008structureofthe pages 7-8, concha2015longrangeinhibitorinducedconformational pages 2-2) Adjacent to the kinase domain, the KEN domain forms a distinct globular unit that bears catalytic residues essential for RNA cleavage, and its dimerization creates a composite active site that accommodates the RNA substrate’s stem-loop structure. (lee2008structureofthe pages 9-11, korennykh2009theunfoldedprotein pages 5-7)
6. Regulation  
   In unstressed cells, IRE1 is maintained in an inactive state largely due to its interaction with the ER chaperone BiP, which binds to the luminal domain and prevents dimerization; this inhibitory interaction is relieved under conditions of ER stress when accumulating misfolded proteins sequester BiP away from IRE1. (ali2011structureofthe pages 1-2, riaz2020roleofendoplasmic pages 3-4) Subsequent homodimerization and oligomerization of IRE1’s luminal domains promote trans-autophosphorylation of its kinase domain, which in turn triggers a conformational transition that activates the RNase domain through a shift from a face-to-face to a back-to-back dimer configuration. (riaz2020roleofendoplasmic pages 4-6, lee2008structureofthe pages 9-11) Phosphorylation at key serine and threonine residues within the activation segment is essential not only for catalytic activity but also for stabilizing the active conformation that permits high-affinity ADP binding and effective RNase dimerization. (itzhak2014multipleautophosphorylationssignificantly pages 6-7) Allosteric modulation of IRE1’s activity is also achieved through the binding of ATP-competitive inhibitors; certain compounds, including kinase inhibiting RNase attenuators (KIRAs) and other small molecules such as 4µ8C, can either inhibit or paradoxically stimulate the RNase function by altering the kinase domain conformation and thus the overall dimerization state. (feldman2019pharmacologicalmodulationand pages 12-18, ghosh2014allostericinhibitionof pages 1-3) In addition, regulatory proteins and chaperones (for example, HSP47, HSP72, and others) interact with IRE1 to fine-tune its stability, phosphorylation status, and oligomer formation, thereby integrating multiple cellular signals in the decision between adaptive and terminal UPR responses. (riaz2020roleofendoplasmic pages 8-9, son2021roleofthe pages 23-29)
7. Function  
   Human IRE1 functions as a pivotal sensor and effector within the unfolded protein response, a signaling pathway that is activated in response to the accumulation of misfolded proteins in the ER. (hetz2011theunfoldedprotein pages 1-2) Upon activation, IRE1’s endoribonuclease activity catalyzes the unconventional splicing of XBP1 mRNA, resulting in the production of spliced XBP1 (XBP1s), a transcription factor that drives the expression of genes involved in protein folding, ER-associated degradation (ERAD), and lipid biosynthesis to restore ER homeostasis. (ali2011structureofthe pages 2-3) In parallel, IRE1 also mediates regulated IRE1-dependent decay (RIDD) by cleaving select ER-localized mRNAs, a process that contributes to reducing the load of newly synthesized proteins entering the ER during stress conditions. (bartoszewska2023dualrnaseactivity pages 1-3) The dynamic balance between the adaptive splicing of XBP1 mRNA and the pro-apoptotic RIDD activity of IRE1 is critical for determining cell fate, influencing whether a cell adapts to or succumbs under prolonged or severe ER stress. (son2021roleofthe pages 29-34) IRE1 is ubiquitously expressed in mammalian tissues, reflecting its central role in managing ER proteostasis and its integration with cellular processes such as metabolism, calcium signaling, and inflammatory responses. (riaz2020roleofendoplasmic pages 1-3, yildirim2022investigatingthetargets pages 118-120) In pathophysiological contexts, dysregulated IRE1 signaling has been implicated in a variety of diseases including cancer, metabolic disorders, neurodegeneration, and inflammatory conditions, underscoring its importance as a therapeutic target. (ghosh2014allostericinhibitionof pages 3-4, fieldman2019pharmacologicalmodulationand pages 12-18)
8. Other Comments  
   Several small-molecule inhibitors targeting IRE1’s kinase and/or RNase domains have been developed to modulate its activity in disease contexts; for example, compounds such as 4µ8C selectively inhibit the RNase function by binding covalently within the active site, while others categorized as KIRAs stabilize inactive kinase conformations, thereby indirectly attenuating RNase activity. (cross2012themolecularbasis pages 1-2, tavernier2018regulationofire1 pages 13-14) These inhibitors are under investigation for their potential to mitigate pathological ER stress responses in diseases such as cancer, diabetes, and inflammatory disorders, where aberrant IRE1 signaling contributes to disease progression. (feldman2019pharmacologicalmodulationand pages 12-18, yildirim2022investigatingthetargets pages 118-120) In addition, numerous post-translational modifications, including phosphorylation, modulate IRE1’s activity and have been mapped to key residues within the activation segment, providing targets for future therapeutic intervention. (itzhak2014multipleautophosphorylationssignificantly pages 7-7, chawla2008regulationofire1 pages 28-31) Furthermore, recent studies have highlighted the involvement of IRE1’s interactions with various regulatory proteins and chaperones, which together form a dynamic UPRosome that fine-tunes the cell’s response to ER stress and influences decisions regarding adaptation versus cell death. (riaz2020roleofendoplasmic pages 9-11, thomas2021decodingnoncanonicalmrna pages 4-4) Taken together, the multifaceted regulation of IRE1 and its dual catalytic activities underscore its potential as a central node in pharmacological strategies aimed at restoring ER homeostasis in a range of diseases linked to chronic ER stress. (ghosh2014allostericinhibitionof pages 1-3, son2021roleofthe pages 29-34)

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