Biosynthetic Disulfide Bond Formation and Redox Homeostasis in the Endoplasmic Reticulum

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Abstract

We have combined genetic and biochemical methods to delineate the enzymatic pathway for disulfide bond formation in the endoplasmic reticulum (ER). The two primary components of this pathway are an ER localized oxidase known as Ero1; and Protein Disulfide Isomerase (PDI) a thiol oxidoreductase that carries disulfide bonds generated by Ero1 to substrate proteins. One of the most surprising aspects of this system is its exquisite regulation. Both PDI and Ero1 cooperate to sense the abundance of disulfide bonds in the ER lumen. Ero1 is normally in an inactive resting state and is activated on demand only when there is a deficit of disulfide bonds as sensed by PDI. The apparent purpose of this regulation is to minimize the formation of reactive oxygen species that inevitably accompanies disulfide bond generation.

Short Biography

Chris Kaiser is currently a Professor of Biology at Massachusetts Institute of Technology (MIT). He is a former Head of the Department of Biology and former Provost of MIT. He received his A.B. magna cum laude in biochemical sciences from Harvard College and his Ph.D. in biology from MIT with David Botstein. He carried out his postdoctoral training at the University of California, Berkeley with Randy Schekman. He has been on the MIT faculty since 1991 where he runs a laboratory studying the cell biology of protein folding and membrane protein sorting in the secretory pathway. In addition to his research activities, Kaiser is also a dedicated educator. He taught Genetics at MIT for twenty-one years and is the recipient of a MacVicar Faculty Fellowship in recognition of his teaching service. In addition to serving on a number of review panels and editorial boards, Kaiser is a coauthor of a major textbook Molecular Cell Biology (W.H. Freeman and Co.)