Biochimica et Biophysica Acta 1817 (2012) S32–S39

Contents lists available at SciVerse ScienceDirect

Biochimica et Biophysica Acta

journal homepage: www.elsevier.com/locate/bbabio

S4 Membrane transporter

4L1

Biogenesis of proteins targeted to the intermembrane space of mitochondria

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Mitochondria have developed the specific translocation machineries to accommodate recognition, import and correct sorting of various mitochondrial proteins to the mitochondrial compartments. The MIA (Mitochondrial Intermembrane Space Assembly) pathway is dedicated to the biogenesis of intermembrane space proteins. A hallmark of this pathway is regulated transfer of disulfide bonds. The MIA pathway represents a novel disulfide-transferring system to control vectorial translocation of proteins into the mitochondria. Mia40, one of the essential components of this pathway, acts in a receptor-like manner and dictates substrate entry to the MIA pathway, while embedded in the inner mitochondrial membrane. Based on our estimation of minimal length of the ribosome-arrested precursors required for the Mia40 interaction, Mia40 was found to be localized in the vicinity of the outer mitochondrial membrane translocase, TOM. A novel factor responsible for the Mia40 recruitment into the TOM machinery called Fcj1/mitofilin was identified. Mitofilin is an important component of the novel multi-subunit complex responsible for maintaining of the inner membrane architecture. Erv1 is another player of the MIA pathway. In addition to its function in reoxidation of Mia40, Erv1 directly participates in Mia40–substrate complex dynamics by forming a ternary complex.

doi:10.1016/j.bbabio.2012.06.096

4L2

New perspectives on assembling *c*-type cytochromes, particularly from sulphate reducing bacteria and mitochondria

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A *c*-type cytochrome is formed via a deceptively simple post translational modification reaction in which the two thiol groups of the cysteines in a CXXCH motif (there are rare variations when the motif is CXXCK, ZXXCH or the number of X residues is more than two) add to the two vinyl groups of heme to give thioether bonds. These vinyl groups are not especially activated (in the sense that the carbon–carbon double bond of the thiol reagent N-ethyl maleimide is activated by adjacent carbonyl groups) to an addition reaction of this kind but the thioether bonds so formed are very stable and can only be broken in vitro by reaction with a heavy metal such as mercury or silver. The reasons for making these bonds are not entirely understood but ideas have been discussed [1,2].

Although all *c*-type cytochromes characterised to date have the same stereospecificity of heme attachment [1], it has turned out over the last twenty years or so that there are several different systems that catalyse this process. The subject has recently been comprehensively reviewed [3–8]. Here we focus on some recent new developments emerging from studies of the Systems I and III for *c*-type cytochrome biogenesis and discussed, particularly in regard to developments in studying System I in sulphate reducing bacteria. A significant uncertainty in this field is how heme is transported across membranes.

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doi:10.1016/j.bbabio.2012.06.097

4L3

Oxidation-driven protein import into the intermembrane space of mitochondria

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In the bacterial periplasm and in the ER of eukaryotic cells, sulfhydryl oxidases catalyze the formation of disulfide bonds between cysteine residues in order to induce or stabilize protein folding. In contrast, other cellular compartments are assumed to be generally

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