Atp10p Assists Assembly of Atp6p into the F₀ Unit of the Yeast Mitochondrial ATPase*

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The F₀F₁-ATPase complex of yeast mitochondria contains three mitochondrial and at least 17 nuclear gene products. The coordinate assembly of mitochondrial and cytosolic translation products relies on chaperones and specific factors that stabilize the pools of some unassembled subunits. Atp10p was identified as a mitochondrial inner membrane component necessary for the biogenesis of the hydrophobic F₀ sector of the ATPase. Here we show that, following its synthesis on mitochondrial ribosomes, subunit 6 of the ATPase (Atp6p) can be cross-linked to Atp10p. This interaction is required for the integration of Atp6p into a partially assembled subcomplex of the ATPase. Pulse labeling and chase of mitochondrial translation products in vivo indicate that Atp6p is less stable and more rapidly degraded in an atp10 null mutant than in wild type. Based on these observations, we propose Atp10p to be an Atp6p-specific chaperone that facilitates the incorporation of Atp6p into an intermediate subcomplex of ATPase subunits.

The bulk of cellular ATP of eukaryote cells is synthesized by the F₀F₁-ATPase complex of the mitochondrial inner membrane. The ATPase is composed of two functionally and physically coupled parts, i.e. the membrane-embedded F₁ sector to which the hydrophilic F₁ sector is attached from the matrix side. In the yeast Saccharomyces cerevisiae, the F₀ sector consists of eight subunits (for reviews, see Refs. 1–3). An oligomer of Atp9p forms a ring-like structure that rotates in the membrane bilayer and, together with Atp6p, drives proton transfer across the inner membrane. Six other subunits, namely Atp8p, Atp4p, and the oligomycin sensitivity-conferring protein (OSCP) and its subunits d, f, and h, form the stator arm connecting F₀ to F₁. The γ, δ and ε subunits of F₁ are part of a central stalk linking it to the Atp6p ring. ATP synthesis depends on a rotation of this stalk within the static catalytic F₁ hexamer made up of three α and three β subunits. Seven other proteins, stably associated with the F₀F₁-ATPase, function in the regulation or oligomerization of the yeast complex (1, 4, 5).

Three components of the yeast F₀ sector, Atp6p, Atp9p, and Atp10p, are encoded in the mitochondrial genome, whereas the other subunits are products of nuclear genes whose synthesis occurs in the cytosol. Current ideas of how the ATPase complex is assembled are based on the analysis of mutants and on what is known about the subunit topology of the complex (1, 6–9). A model compatible with such information is depicted in Fig. 1. After synthesis on mitochondrial ribosomes, Atp9p oligomerizes to form a ring-like homo-oligomer composed of 10–12 subunits (3). The F₁ sector assembles independently of the F₀ subunits and attaches to the Atp9p ring (10, 11). This complex was reported to be further modified by the addition of Atp8p first (6), followed by Atp4p (8) and perhaps other components of the stator arm. Finally, assembly of the Atp6p subunit completes the process to yield a functional ATPase complex (6, 12). The addition of Atp6p at a late stage of assembly may prevent the exchange of protons across the membrane through the partially assembled F₀ sector. This scheme is still very fragmentary and is lacking a host of intermolecular interactions of other F₀ subunits needed to stabilize the final complex (1, 2, 13).

Biogenesis of F₁ requires the general chaperone Hsp60p (14) and at least three factors, Atp11p, Atp12p, and Fmc1p (15–17). Atp11p and Atp12p function as molecular chaperones that probably stabilize unassembled α and β subunits by shielding their hydrophobic surfaces (16, 18). The function of Fmc1p is unclear at present. Much less is known about assembly of F₀. The nuclear gene ATPI0 was identified in a genetic screen to be essential for the proper assembly of F₀ (19). In the absence of Atp10p, the mitochondrial encoded ATPase subunits are synthesized but not assembled into F₀, because the F₁-ATPase is only loosely attached to the membrane. Because Atp10p is not a subunit of the ATPase complex (19) and is not involved in post-translational processing or modification of Atp6p (20), it was proposed to be an Atp6p-specific chaperone (20). The ability of a mutation near the carboxyl terminus of Atp6p to suppress an atp10 null mutant (20) points at a role of Atp10p in folding, assembly, or maintenance of Atp6p. The matrix chaperone Hsp70p has also been shown to be necessary for biogenesis of the F₀ sector (21). This general chaperone binds to and facilitates the association of Atp6p with a partially assembled intermediate of F₀ containing the Atp9p oligomer.

To analyze the function of Atp10p in more detail, we studied the assembly of the ATPase complex following radiolabeling of the mitochondrial encoded subunits in isolated wild type and mutant mitochondria. Based on the results reported here and those of earlier studies, Atp10p is proposed to function as a chaperone that interacts physically with newly synthesized Atp6p and maintains it in an assembly-competent state.

MATERIALS AND METHODS

Yeast Strains and Growth Media—All strains used in this study were isogenic with the wild type strain W303-1A. Construction of the aW303ΔATP10 deletion mutant and isolation of the aW303ΔATP10/R1 and R3 revertants (independent revertants with the identical mutation

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19775
in ATP6p have been described (16, 20). Yeast cultures were grown at 30 °C in YP (1% yeast extract and 2% peptone) medium supplemented with 2% galactose or 0.1% KOH-buffered lactate (22). Mitochondria were isolated as described previously (22), except that the cultures were adjusted to 2 mM/ml chloramphenicol for 2 h prior to harvesting of the cells.

Labeling of Mitochondrial Translation Products—For in vivo labeling of mitochondrial translation products, cells were grown in YPgal medium (2% galactose, 1% yeast extract, 2% peptone). Cycloheximide (150 μg/ml) was added to block cytosolic protein synthesis. After the addition of [35S]methionine, cells were incubated with agitation for 15 min. Reactions were stopped by the addition of 40 mM puromycin and 25 mM cold methionine and incubation for 10 min. After different time periods, aliquots were removed. Proteins were extracted, precipitated by trichloroacetic acid, resolved by SDS-PAGE, and visualized by autoradiography.

Mitochondrial translation products were labeled in vitro as described previously (22, 23). Mitochondria (40 μg protein) were incubated in translation buffer (0.6 M sorbitol, 150 mM KCl, 15 mM KH2PO4, 13 mM MgSO4, 0.15 mg/ml all amino acids except methionine, 4 mM ATP, 0.5 mM GTP, 5 mM α-ketoglutarate, 5 mM phosphoenolpyruvate, 3 mM/ml fatty acid-free bovine serum albumin, and 20 mM Tris/HCl, pH 7.4) containing 0.6 units of pyruvate kinase and 10 mM cold methionine and incubation for 10 min. After different time periods, aliquots were removed. Proteins were extracted, precipitated by trichloroacetic acid, resolved by SDS-PAGE, and visualized by autoradiography.

RESULTS

Atp10p Is in Direct Contact with Newly Synthesized Atp6p—Previous evidence for a genetic interaction of Atp10p and Atp6p (20) suggested that the two proteins may also interact physically. To detect a complex of the two proteins, mitochondrial translation products were labeled with [35S]methionine in the presence of the non-cleavable cross-linker DSS. The labeled mitochondria were solubilized, and the extract was treated with antibody against Atp10p in the presence of protein A-Sepharose beads. Autoradiography of the proteins separated by SDS-PAGE indicated background signals of all the translation products, irrespective of the presence or absence of the cross-linker. This unspecific adsorption to Sepharose is due to the highly hydrophobic nature of the polypeptides. A band of an apparent size of 52 kDa was enriched by Atp10p antibodies (Fig. 2, asterisk). This band only occurred in the samples treated with the cross-linker and was absent in the Δatp10 mutant and the Δatp10 revertant (W303 ΔATP10/R1) (20). This indicated that one of the radiolabeled translation products is in the proximity of Atp10p during or directly following its synthesis in the mitochondria. The apparent size of the novel band (52 kDa) is consistent with it being an adduct of Atp6p (30 kDa) and Atp10p.
Atp10p complex and indicate that Atp10p is in the proximity of newly synthesized Atp6p. The Atp6p released from the adduct appeared to migrate slightly faster than the Atp6p that had not been cross-linked (Fig. 3A, lanes marked $T$, loaded with total mitochondrial proteins). This mobility shift is caused by the immunoprecipitation procedure, because it is also seen when Atp6p is immunoprecipitated with Atp6p-specific antibodies from mitochondrial extracts that were not treated with a cross-linker (Fig. 3B).

The Interaction of Atp10p with Atp6p Can Occur Post-translationally—Proteins involved in membrane insertion of mitochondrial translation products typically interact with their substrates co-translationally. This was shown to be the case for the Oxa1p translocase, which promotes insertion of the mitochondrially translated subunits of cytochrome oxidase into the inner membrane (25, 26). On the other hand, factors that coordinate oligomerization of completed and folded subunit polypeptides are more likely to bind to their substrates post-translationally. To assess if the Atp10p/Atp6p complex is formed co- or post-translationally, DSP was added to mitochondria either during or following the labeling reaction. No significant difference was noted in the amount of Atp6p associated with Atp10p under the two conditions (Fig. 4). This indicates a post-translationnal interaction of Atp10p with Atp6p but does not exclude that it can also occur co-translationally.

Assembly of the ATPase Complex in Isolated Wild Type Mitochondria—Assembly of the ATPase complex was reported to occur in a sequential and obligatory order (6–8) as depicted in Fig. 1. To gain better insights into the role of Atp10p in this process, we studied the assembly of the ATPase complex in isolated mitochondria. Assembly in this in vitro system was enhanced in cells allowed to grow in the presence of chloramphenicol for 2 h prior to isolation of the mitochondria. Under these conditions, mitochondrial translation is blocked, leading to increased pools of nuclear gene products available for interaction with the newly synthesized subunits made in organello.

In agreement with earlier findings (21), all eight mitochondrially encoded polypeptides and two slower migrating bands with apparent masses of 48- and 54-kDa are detected in the autoradiogram of the labeled mitochondria (Fig. 5A, lane 1). The 48-kDa and 54-kDa products represent oligomers of F$_1$, subunits that are incompletely depolymerized by dodecyl sulfate (21) but are converted to the monomeric constituents when proteins are treated with trichloroacetic acid prior to SDS-PAGE (Fig. 5A, lane 2). Both oligomers are associated with the F$_1$-ATPase, as they were co-immunoprecipitated by antibodies against the F$_1$$\alpha$ subunit (Fig. 5A, lane 3). The enrichment of the 54-kDa oligomer suggests that this form is more efficiently or more stably attached to the F$_1$ sector recognized by the F$_1$$\alpha$-antisemur. An antisemur against the Atp4p subunit also co-immunoprecipitated the three mitochondrially synthesized subunits that are incompletely depolymerized by dodecyl sulfate (21) but are converted to the monomeric constituents when proteins are treated with trichloroacetic acid prior to SDS-PAGE (Fig. 5A, lane 2). Both oligomers are associated with the F$_1$-ATPase, as they were co-immunoprecipitated by antibodies against the F$_1$$\alpha$ subunit (Fig. 5A, lane 3). The enrichment of the 54-kDa oligomer suggests that this form is more efficiently or more stably attached to the F$_1$ sector recognized by the F$_1$$\alpha$-antisemur. An antisemur against the Atp4p subunit also co-immunoprecipitated the three mitochondrially synthesized

![Fig. 3. Atp10p can be cross-linked to newly synthesized Atp6p.](Image)

![Fig. 4. Atp6p interacts with Atp10p post-translationally.](Image)
ATPase subunits. In this instance, however, more Atp6p was co-precipitated because of its direct interaction to Atp4p (2). This indicates that, following synthesis in isolated mitochondria, Atp6p, Atp9p, and most likely also Atp8p assemble, at least in part, into complexes that contain subunits of the stator arm and F1.

The composition of the 48- and 54-kDa oligomers was examined by separating labeled wild type mitochondria by SDS-PAGE in one dimension, partially dissociating the oligomers in a strip of the gel with trichloroacetic acid, and separating the proteins in the acid treated strip by SDS-PAGE in a second dimension. The trichloroacetic acid treatment caused the release of the 48-kDa complex of a radioactive protein that migrated like Atp9p, as well as the release from the 54-kDa complex of two proteins, one corresponding to Atp9p and the other to Atp6p (Fig. 5B). Nuclear encoded ATPase subunits that might be present in the two oligomers are not labeled and, therefore, cannot be detected by this method. The presence of Atp6p in the 54-kDa oligomer is supported by Western blot analysis of mitochondrial proteins probed with an Atp6p-specific antisera, which reacted with the 22-kDa Atp6p monomer in wild type mitochondria isolated from the wild type strain W303-1A (WT) or the atp10 null mutant aWT303ΔATP10 (ΔATP10) were separated by SDS-PAGE, transferred to nitrocellulose, and probed with an Atp6p-specific antibody. Body. B, translation products were radiolabeled mitochondria isolated from the wild type and the atp10 null mutant and were analyzed as described in the legend for Fig. 5B. C, mitochondrial translation products labeled in organello with [35S]methionine were co-immunoprecipitated with antibodies against the mitochondrial Hsp70p chaperone and pre-immune serum (p.i.). The samples in the lanes labeled TCA + were precipitated with trichloroacetic acid to dissociate the oligomeric ATPase complexes before depolymerization in sample buffer and separation by SDS-PAGE. D, translation products were labeled in wild type (WT) and the atp10 null mutant (ΔATP10) mitochondria for 10 or 30 min and subjected to co-immunoprecipitation with Hsp70p antibodies. The amount of immunoprecipitated Atp6p was quantified by densiometry and normalized to the amount of the total Atp6p synthesized.

Atp10p is required for assembly of the 54-kDa oligomer. A, Atp10p is required for stable accumulation of Atp6p in mitochondria. Mitochondria (100 μg) from the wild type strain W303-1A (WT) or the atp10 mutant aWT303ΔATP10 (ΔATP10) were separated by SDS-PAGE, transferred to nitrocellulose, and probed with an Atp6p-specific antibody. Positions of molecular mass standards are indicated. The bands marked 48K and 54K depict the 48- and 54-kDa complexes. B, translation reactions were labeled as described for section B and resolved by SDS-PAGE. The gel slice of the labeled proteins was cut out and incubated in 12% trichloroacetic acid to partially dissociate the ATPase complexes. The pH of the gel slice was adjusted to 6.8 and positioned on top of a new SDS gel for separation in a second dimension. The 48-kDa complex (48K) partially dissociated into monomeric Atp9p, and the 54-kDa complex partially dissociated into Atp9p and Atp6p. Positions of the molecular mass standards for both dimensions are indicated. C, Atp6p is part of the 54-kDa complex. Mitochondria were lysed in LiDS sample buffer and subjected to SDS-PAGE. The gel slice of the acid treated strip by SDS-PAGE in a second dimension. The trichloroacetic acid treatment caused the release from the 54-kDa complex of two proteins, one corresponding to Atp9p and the other to Atp6p (Fig. 5B). Nuclear encoded ATPase subunits that might be present in the two oligomers are not labeled and, therefore, cannot be detected by this method. The presence of Atp6p in the 54-kDa oligomer is supported by Western blot analysis of mitochondrial proteins probed with an Atp6p-specific antisera, which reacted with the 22-kDa Atp6p monomer and the 54-kDa oligomer (Fig. 5C). These data indicate that isolated mitochondria are capable of assembling two different subcomplexes of the ATPase, i.e. a 48-kDa complex lacking Atp6p and one of 54 kDa that contains Atp6p. Atp10p Is Required to Form the 54-kDa Complex—Impaired biogenesis of the F0 sector in the Δatp10 mutant was shown previously to markedly lower the steady-state levels of Atp6p (20). This was confirmed with an antibody against Atp6p, which failed to detect the monomeric or assembled form of Atp6p in the mutant (Fig. 6A). The F0 defect in Δatp10 mitochondria was studied in more detail by examining the oligomerization state of newly synthesized Atp6p. Pulse labeling of mitochondria isolated from the Δatp10 mutant showed that all three of the mitochondrially encoded subunits of the ATPase were synthesized at ~50% of the rate in wild type (Fig. 6B). Furthermore, whereas the 48-kDa oligomer was also present in the mutant, the 54-kDa oligomer was absent (indicated by the top arrow in Fig. 6B). Thus, although the Atp6p oligomer assembles independently of Atp10p, formation of the larger complex containing Atp6p is blocked in the absence of Atp10p. Earlier evidence indicated that the 48-kDa but not the 54-
Atp6p was higher in the mutant than in the wild type (Fig. 7A, see bands labeled TCA +). This confirms the presence of Atp9p, but not of Atp6p or Atp8p, in the 48-kDa oligomer. Atp10p was not required for the interaction of Hsp70p with the Atp9p oligomer, as similar amounts of Atp9p could be precipitated with Hsp70p antibodies in wild type and ∆atp10 mutants (Fig. 6D). Thus, Atp10p and Hsp70p appear to play distinct roles and interact with different pools of unassembled ATPase subunits, i.e. Atp10p with Atp6p and Hsp70p with a partially assembled complex containing the Atp9p oligomer.

Atp10p Partially Protects Newly Synthesized Atp6p against Proteolysis—In organello incorporation of [35S]methionine into Atp6p is reduced in the ∆atp10 mutant (Figs. 5A and 6B). This could be due to reduced synthesis or increased degradation of the protein by proteases. To distinguish between these possibilities, mitochondrial translation products were radiolabeled in yeast cells in the presence of cycloheximide for 15 min. Translation was stopped by the addition of puromycin, and the stability of the synthesized proteins was analyzed after various periods of chase (Fig. 7A). No significant difference was found in the turnover of newly synthesized Atp6p/9p in the wild type and the mutant during the 2 h period of chase. In contrast, turnover of Atp6p was higher in the mutant than in the wild type (Fig. 7A and B). Atp10p could be protecting Atp6p by directly shielding it from degrading proteases or by promoting the assembly of Atp6p into a protease-resistant complex.

**DISCUSSION**

Formation of hetero-oligomeric membrane complexes depends on the assistance of specific chaperones that interact with their cognate subunits. This may be necessary in part because of the hydrophobic nature of unassembled subunits, which may have to be stabilized by accessory factors to allow them to progress through the assembly pathway and minimize non-productive side reactions. The results reported here, together with those reported earlier, indicate that Atp10p has such a chaperone function in assembly of Atp6p, as evidenced by the following four observations. 1) Atp10p can be cross-linked to newly synthesized Atp6p. This interaction occurs post-translationally and appears to be transient, as Atp10p is not a subunit of the functional ATPase complex. 2) Atp10p is required for proper assembly of Atp6p into the F0 sector of the ATPase. A role of Atp10p for proteolytic maturation or post-translational modification of Atp6p was excluded (20). Binding of Atp10p to Atp6p is more likely to be necessary to maintain Atp6p in an assembly-competent state. 3) Atp6p is more prone to proteolytic degradation in the ∆atp10 mutant, indicating that it is directly or indirectly protected by Atp10p. 4) A point mutation in Atp6p can suppress ∆atp10 mutants (20). The increased stability of Atp6p in the suppressed strain may reflect a shift of the equilibrium from degradation toward assembly of Atp6p.

Based on these observations, we propose that following synthesis on mitochondrial ribosomes, Atp6p is bound to Atp10p. This interaction prevents degradation and/or aggregation of Atp6p and channels it to associate with a preformed subcomplex of the ATPase (Fig. 8). The integration of Atp6p into this intermediate complex may be dependent directly on Atp10p. Alternatively, Atp10p may shift the equilibrium from degradation/aggregation to assembly. The latter seems more likely in view of the observation that ∆atp10 mutants are leaky and...
display slow growth on non-fermentable carbon sources (20). In the atp10 mutant the equilibrium might be shifted further toward assembly as a result of the increased stability of Atp6p. It is also possible, however, that the suppressor mutation helps to increase the efficiency with which Atp6p is assembled, thereby sparing a larger fraction of the protein from degradation. Assuming that Atp6p is one of the last subunits to be incorporated into the complex as proposed previously (12), the intermediate complex with which Atp6p assemblies would be expected to contain most or all of the other ATPase subunits. Two oligomeric complexes are detected in mitochondria that are allowed to synthesize the three endogenously encoded subunits of the ATPase. The 48-kDa oligomer observed in wild type mitochondria is also detected in mitochondria lacking Atp10p.

In the absence of Hsp70p function, the 48-kDa subcomplex loses its competence to interact with Atp6p, and assembly of the final ATPase structure is arrested (21). The interaction with the Hsp70p chaperone may stabilize this intermediate for further progression in the assembly pathway. Thus, the final step of the pathway appears to rely on two chaperones, namely the classical chaperone Hsp70p, which binds to the 48-kDa oligomeric intermediate, and Atp10p, which interacts with newly synthesized Atp6p and promotes its incorporation into a late assembly intermediate.

Members of the Hsp70p and Hsp60p families of chaperones participate in numerous cellular processes by facilitating folding and assembly of a large number of different substrates. In contrast, some reactions employ factors that have a single substrate polypeptide as their target. Although several such specific assembly factors have been reported in recent years, little is known about the mechanisms by which they work. One well studied example, Ump1p, is a specific factor that associates with and helps to complete the assembly of proteasome intermediates (27). Ump1p tightly regulates proteasome biogenesis and is removed at the final assembly step. The interaction with Ump1p is thought to prevent the harmful effects of proteolytically active but incompletely assembled proteasome intermediates. Despite the differences in the physiological role of the proteasome and the ATPase, subunit-specific chaperones like Atp10p may provide similar protective functions. The partially assembled F1 sector of the ATPase could present potential problems for the cell because of its ability to dissipate the membrane potential. This might explain why the proton-conducting Atp6p subunit is integrated into the complex at a late assembly step (12). Atp10p might control this critical step by ensuring the maintenance of a pool of assembly-competent Atp6p subunits. Whereas the cellular levels of general chaperones that are not occupied by substrates vary greatly with different temperatures and physiological conditions, the pool of specific chaperones can be precisely controlled and adapted to the needs of a certain biological process. An important focus of future work will be to understand the regulation of substrate-specific chaperones, such as Atp10p, and to clarify the molecular mechanisms by which they bind and release their substrate proteins.

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