A cDNA carrying the Rip1 gene, which encodes the Rieske iron-sulfur protein of Schizosaccharomyces pombe, has been cloned by complementing the respiratory deficiency of a Saccharomyces cerevisiae strain in which the endogenous copy of the RIP1 gene has been deleted. The deduced amino acid sequences of the S. pombe and S. cerevisiae iron-sulfur proteins are 50% identical, with the highest region of identity being in the C termini of the proteins, where the 2Fe:2S cluster is bound. When expressed in the S. cerevisiae deletion strain, the S. pombe iron-sulfur protein restores 25-30% of the ubiquinol-cytochrome c reductase activity. The kinetics of cytochrome c reduction, the effects of inhibitors which act at defined sites in the cytochrome bc$_1$ complex, and the optical properties of cytochrome b in membranes from the S. cerevisiae deletion strain complemented with S. pombe iron-sulfur protein indicate that the S. pombe protein interacts with cytochrome b to restore an apparently normal ubiquinol oxidase site, but that interaction between the iron-sulfur protein and cytochrome c$_1$ is partially impaired. This is the first heterologous replacement of an electron transfer protein in a respiratory enzyme complex in S. cerevisiae.

The cytochrome bc$_1$ complex is an oligomeric electron transfer enzyme located in the inner membrane of mitochondria and the plasma membrane of numerous bacteria (1). The cytochrome bc$_1$ complex transfers electrons from ubiquinol to cytochrome c$_1$ by a protonmotive Q cycle mechanism (2), thus converting electrical energy into a proton motive force across the membrane in which the enzyme resides. An essentially identical enzyme, the cytochrome bc$_1$ complex, is located in the thylakoid membranes of chloroplasts, where it catalyzes plastocyanin-oxygen reductase activity.

The Rieske iron-sulfur protein is one of three redox proteins which participate in the electron transfer activities of the cytochrome bc$_1$ complex, the other two being cytochrome c$_1$ and a di-heme cytochrome b. In the protonmotive Q cycle mechanism, the iron-sulfur protein catalyzes the single electron oxidation of ubiquinol, transferring one electron to cytochrome c$_1$ and generating a strongly reducing ubisemiquinone anion, which reduces the low potential cytochrome b$_{566}$ heme. Although the role of the iron-sulfur protein in the protonmotive Q cycle is established, there are numerous unanswered questions regarding how the iron-sulfur protein functions (3). In order to better understand structure-function relationships in the Rieske iron-sulfur protein, we have undertaken a mutational analysis of this protein in Saccharomyces cerevisiae (4-7). Here we report the cloning of a cDNA encoding the Rieske iron-sulfur protein of Schizosaccharomyces pombe and show that the S. pombe iron-sulfur protein can functionally substitute for the S. cerevisiae iron-sulfur protein.

EXPERIMENTAL PROCEDURES

Materials—Restriction endonucleases and T4 DNA ligase were from New England Biolabs. The Sequenase Kit was obtained from U. S. Biochemical Corp. [α-32P]dCTP and [α-35S]dATP were from Amersham Corp. Nuc-Trap columns and QuickHyb from Stratagene were used for probe purification and hybridization. Kodak XAR-5 film was used with a DuPont Cronex Lightning Plus intensifying screen for autoradiography. SDS, acrylamide, and bisacrylamide were from Bio-Rad. Urea and agarose (UltraPure) were from Life Technologies, Inc. DNA was isolated from agarose gels using the Gene Clean Kit from Bio101. Nytran nylon membranes were from Schleicher & Schuell. Glass beads (0.5 mm) were obtained from Biospec Products. Horse heart cytochrome c, decylubiquinone, and amino acids were obtained from Sigma. Yeast extract, peptone, Bacto-agar, and tryptone were from Difco Laboratories.

Cloning by Complementation of Respiratory-deficient Growth—Construction of the stable S. cerevisiae iron-sulfur protein deletion strain, Rip1, and of the complementing 2μ plasmid Yep352 (dr-RIP1) carrying the S. cerevisiae RIP1 gene have been described previously (4). A S. pombe cDNA library was constructed and provided by M. Minet and F. Lacroute. The cDNA was subcloned into the yeast 2μ expression vector pFL61 containing the yeast-selectable marker URA3. The constitutive promoter of the phosphoglycerate kinase gene was used to control the expression of the cDNA. Yeast were transformed by a lithium acetate procedure (8). Transformants were selected on minimal SD medium (0.67% nitrogen base without amino acids + 2% dextrose or 1% yeast extract + 2% peptone + 1% galactose). Respiratory-competent transformants were selected by replica plating onto N3 plates (2% glycerol + 1% yeast extract + 1% peptone). The plasmids were recovered from yeast (9) and amplified in E. coli strain DH5α (10).

Northern Analysis—Total RNAs were isolated from S. cerevisiae strain FL100 (11) and S. pombe strain 972 h (12) growing exponentially in YPD medium (1% yeast extract, 2% peptone, 2% dextrose). Poly(A)$^+$ RNA was purified with oligo(dT)-cellulose (13), resolved by electrophoresis on a 1.2% agarose gel containing formaldehyde, and blotted to nitrocellulose. The nitrocellulose blot was probed with the 1.4 kb$^2$ cDNA insert carrying the S. pombe iron-sulfur gene, which was excised from cloned plasmid SP2 with NotI and labeled with [α-32P]dCTP with the random priming kit from Life Technologies, Inc. (14).

DNA Sequencing—Plasmids amplified in DH5α were prepared by

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‡ The abbreviations used are: kbp, kilobase pair(s); bp, base pair(s); kb, kilobase(s).

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Heterologous Complementation of a Rieske Iron-Sulfur Protein-deficient Saccharomyces cerevisiae by the Rip1 Gene of Schizosaccharomyces pombe

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Fig. 1. Complementation of a Rieske iron-sulfur protein-deficient S. cerevisiae by the Rip1 gene of S. pombe.

The S. cerevisiae iron-sulfur protein deletion strain, JPJ1, was transformed with plasmids carrying either the S. cerevisiae iron-sulfur protein (S. c. RIp1) or the S. pombe iron-sulfur protein (S. p. Rip1). The transformed cells were then streaked onto plates containing either a fermentable (YPD) or nonfermentable (YPEG) carbon source and incubated at 30°C.

Cloning of S. pombe Rieske Iron-Sulfur Protein—The S. cerevisiae RIP1 deletion strain, JPJ1, was transformed with the cDNA library of S. pombe, and a total of 110,000 Ura− inserts. These were then replica-plated to media containing a nonfermentable carbon source and incubated at 30°C. A total of 10,000 plates was analyzed, and of these, four clones grew on ethanol/glycerol as a carbon source. Transformation of the S. cerevisiae deletion strain with the S. pombe cDNA restores respiratory-dependent growth to a rate comparable to that restored by the S. cerevisiae RIP1.

Northern blot analysis under stringent conditions confirmed that the cDNA insert excised from the SP2 clone hybridized to a S. pombe transcript of approximately 1 kb, while no hybridization signal was detected with RNA prepared from S. cerevisiae as shown in Fig. 2. This confirms that the complementing SP2 clone carries S. pombe DNA and not a spurious contaminant from S. cerevisiae.

Western analysis of mitochondrial membranes prepared from the S. cerevisiae RIp1 deletion strain complemented with S. cerevisiae or S. pombe iron-sulfur protein gene revealed that the iron-sulfur protein monoclonal antibody prepared against the S. cerevisiae protein (6) does not cross-react with the S. pombe iron-sulfur protein (results not shown). This provides additional confirmation of the unique identity of the two proteins.

The 1.4-kbp insert in SP2 includes an open reading frame capable of encoding a 228-amino acid protein of 24,824 molecular weight. The sequence of the open reading frame and 54 bp of noncoding DNA at the 5’ end of the gene and the predicted amino acid sequence of the protein encoded by the open reading frame are shown in Fig. 3. The protein encoded by the SP2 cDNA is similar in size to the S. cerevisiae Rieske iron-sulfur protein, the sequences of the two proteins are 47–50% identical, and the hydrophathy profiles of the two proteins are also very similar, as shown in Fig. 4. The highest region of sequence identity is in the C termini of the proteins in the region where the iron-sulfur cluster is bound, and the S. pombe protein includes the four cysteines and two histidine residues which are conserved in all Rieske iron-sulfur proteins. The two histidines and two of the cysteines ligate the 2Fe:2S cluster, and it has been proposed that the remaining two cysteines form a disulfide bridge (6). On the basis of this comparison and the heterologous complementation of the S. cerevisiae petite phenotype resulting from the RIp1 deletion, we conclude that the SP2 clone carries the cDNA for the S. pombe Rieske protein.

Properties of S. cerevisiae Cytochrome b5 Complex Containing S. pombe Rieske Iron-Sulfur Protein—The ubiquinol-cytochrome c oxidoreductase activities of mitochondrial membranes from the S. cerevisiae RIp1 deletion strain expressing either the S. cerevisiae or the S. pombe Rieske protein are shown in Fig. 5. When expressed in the S. cerevisiae deletion strain, the S. pombe iron-sulfur protein restores 25–30% of the ubiquinol-cytochrome c reductase activity (Fig. 5a). Succinate-cytochrome c reductase and NADH-cytochrome c reductase activities were also measured, and the S. pombe Rieske protein restored 25% of activity. These results indicate that the Rieske iron-sulfur protein complements the iron-sulfur protein deletion in S. cerevisiae, and that the S. pombe Rieske protein can functionally substitute for the S. cerevisiae Rieske iron-sulfur protein to restore activities of the respiratory complex.

The second plasmid, SP4, contained apparently identical 1.4-kbp cDNA inserts. The plasmid was recovered from JPJ1/SP2 and JPJ1/SP5 and characterized by restriction enzyme analysis. The plasmids recovered from JPJ1/SP2 and JPJ1/SP5 possessed identical HindIII, PstI, and NotI restriction enzyme patterns and contained apparently identical 1.4-kbp cDNA NotI inserts. JPJ1/SP2 was selected for further analysis.

The ability of the SP2 plasmid carrying the S. pombe cDNA to restore growth of the S. cerevisiae RIp1 deletion strain on a nonfermentable carbon source is shown in Fig. 1. Absence of the iron-sulfur protein in the RIp1 deletion strain, JPJ1, results in a tight respiratory-deficient phenotype reflected by its inability to grow on ethanol/glycerol as a carbon source.

PREPARATION OF MITOCHONDRIAL MEMBRANES—Yeast were grown to mid-log phase in YPEG (1% yeast extract, 2% peptone, 4% ethanol, 3% glycerol), and mitochondrial membranes were isolated by the modified glass bead method (6). Prior to breaking the cells, 0.5 mM diisopropyl glycerol, and mitochondrial membranes were isolated by the modified glass bead method (National Diagnostics). Prior to breaking the cells, 0.5 mM diisopropyl glycerol (6). Prior to breaking the cells, 0.5 mM diisopropyl glycerol, and mitochondrial membranes were isolated by the modified glass bead method (National Diagnostics). Prior to breaking the cells, 0.5 mM diisopropyl glycerol, and mitochondrial membranes were isolated by the modified glass bead method (National Diagnostics).

Optical Absorption Spectroscopy—Mitochondrial membranes were suspended to a protein concentration of 2 mg/ml in 10 mM Tris-Cl, pH 7.4, 0.15 M KCl, 2 mM EDTA, 5 mM KCN, and 50 μM cyanocobalamin. Reduction of cyanocobalamin was initiated by addition of dithionite. The optical absorption spectra of the reduced versus oxidized samples were recorded in the split beam mode using an Aminco DW-2A spectrophotometer. Data points were collected at 0.1 nm intervals with a 1 nm band pass by the aid of a Nicolet digital oscilloscope and x-y recorder interfaced to the spectrophotometer.

RESULTS

Cloning of S. pombe Rieske Iron-Sulfur Protein—The S. cerevisiae RIp1 deletion strain, JPJ1, was transformed with the cDNA library of S. pombe, and a total of 110,000 Ura+ transformants were obtained from two independent transformations. These were then replica-plated to media containing a nonfermentable carbon source. After several days incubation at 28°C, 11 colonies appeared on the N3 plates. Plasmid segregation confirmed that respiratory competence of these colonies co-segregated with uracil prototrophy. Two of the respiratory competent transformants, JPJ1/SP2 and JPJ1/SP5, were chosen for further analysis.

The plasmids were recovered from JPJ1/SP2 and JPJ1/SP5 and characterized by restriction enzyme analysis. The plasmids recovered from JPJ1/SP2 and JPJ1/SP5 possessed identical HindIII, PstI, and NotI restriction enzyme patterns and contained apparently identical 1.4-kbp cDNA NotI inserts. JPJ1/SP2 was selected for further analysis.

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activities of the mitochondrial membranes carrying the S. pombe iron-sulfur protein were 65 and 70%, respectively, of those carrying the S. cerevisiae protein (results not shown). These activities are sufficient to complement the petite phenotype of the RIP1 deletion strain, indicating that the activity of the cytochrome bc₁ complex is not rate-limiting for growth on nonfermentable carbon sources (6).

The ubiquinol-cytochrome c reductase activity is inhibited by antimycin, myxothiazol, and UHDBT, and titrations of the inhibitors indicated that the efficacy of inhibition is identical when the bc₁ complex contained S. cerevisiae or S. pombe iron-sulfur protein (results not shown).

Plots of cytochrome c reductase activity versus ubiquinol concentration revealed that ubiquinol is inhibitory when the S. cerevisiae bc₁ complex contains S. pombe iron-sulfur protein. This substrate inhibition is most evident when the data are presented in the form of double reciprocal Lineweaver-Burk plots as shown in Fig. 5. The calculated hydrophilicity plots of the S. cerevisiae (YscRIP1p) and S. pombe (YpbRip1p) iron-sulfur proteins are shown at the bottom.

We have shown in previous work that the optical absorption spectrum of cytochrome b in yeast mitochondria is sensitive to the presence or absence of the Rieske iron-sulfur approtein but is insensitive to the lack of iron-sulfur cluster insertion (6). Deletion of the iron-sulfur protein results in a diminution of the cytochrome b absorbance in mitochondrial membranes from JP1, as shown by the top trace in Fig. 6. The cytochrome b
absorption spectrum is restored to normal in mitochondrial membranes of the S. cerevisiae deletion strain expressing the S. cerevisiae protein, as shown in the middle trace in Fig. 6. The spectrum of mitochondrial membranes from yeast expressing the S. pombe iron-sulfur protein (bottom trace) is essentially identical with that of the S. cerevisiae complemented strain, indicating that the S. pombe and S. cerevisiae iron-sulfur proteins interact with cytochrome b in a very similar manner.

**DISCUSSION**

In this study, we describe the first heterologous complementation of a respiratory protein in S. cerevisiae and characterize the S. cerevisiae cytochrome bc$_1$ complex in which the endogenous iron-sulfur protein is replaced by that from S. pombe. It is not surprising that the S. pombe iron-sulfur protein will function in the S. cerevisiae cytochrome bc$_1$ complex. Although the 50% overall identity of the two sequences is not unusually high, as can be seen in Fig. 4, the amino acid sequences surrounding the iron-sulfur cluster, between Gln-151 and Glu-201 of the S. cerevisiae sequence, are 86% identical and, if conservative exchanges are accounted for, this segment of the two proteins is 96% homologous. In a random mutagenesis of the S. cerevisiae iron-sulfur protein in which 35 unique nonfunctional or temperature-sensitive alleles were characterized (7), 23 of these were in this highly conserved stretch, and only 1 of these mutated amino acids, A170T, is not identical in the S. cerevisiae and S. pombe sequences (Fig. 4).

The S. pombe protein restores approximately 30% of the ubiquinol-cytochrome c reductase activity to the iron-sulfur protein-deficient S. cerevisiae cytochrome bc$_1$ complex under conditions where the activity is zero order with respect to ubiquinol and cytochrome c. The incomplete restoration of activity is at least partially due to inhibition by the ubiquinol substrate when the S. pombe protein is substituted for the S. cerevisiae protein. Electron transfer from ubiquinol to cytochrome c through the high potential redox centers of the cytochrome bc$_1$ complex consists of three electron transfer reactions as diagrammed in Fig. 7. Reaction 1, oxidation of ubiquinol by the iron-sulfur cluster, is a diffusion-controlled reaction when the ratio of ubiquinol to bc$_1$ complex is 3 or less (17). However, at 45 M ubiquinol (Fig. 5), the ratio of quinol to enzyme is 3000:1, and, under these conditions, reaction 1 is zero order with respect to ubiquinol. Similarly, the ratio of cytochrome c to enzyme is sufficiently high (>3000:1) that reaction 3 is also zero order. Thus, under the usual cytochrome c reductase assay conditions, the rate at which ubiquinol is oxidized is limited by reaction 2 (Fig. 7), reoxidation of the 2Fe:2S cluster by cytochrome c.

Electron transfer from the 2Fe:2S cluster to the C$_1$ heme normally proceeds with an estimated half-time less than 200 $\mu$s (17) and is an essentially solid state reaction (3), requiring an exact fit between the iron-sulfur protein and the cytochrome. Substitution of the endogenous iron-sulfur protein with the S. pombe protein appears to compromise this interaction, such that electron transfer from the iron-sulfur cluster to the cytochrome C$_1$ heme is retarded in the heterologous cytochrome bc$_1$ complex.
The lag in the onset of inhibition (Fig. 5) can be attributed to the fact that exposure of the mitochondrial membranes to oxygen in the presence of only limiting amounts of endogenous substrate maintains the 2Fe:2S cluster in the oxidized state prior to initiating the cytochrome $c$ reductase assay. Under these conditions, oxidized 2Fe:2S cluster is initially available to oxidize ubiquinol. Early in the course of the cytochrome $c$ reductase assay, the redox poise of the cluster changes to a more fully reduced state, owing to the presence of excess quinol and retarded reoxidation of the cluster by cytochrome $c_1$, and the reduced cluster cannot oxidize ubiquinol. In the traces shown in Fig. 5, we estimate that the cytochrome $bc_1$ complex undergoes approximately 300 turnovers before the inhibitory effect of 45 $\mu$M ubiquinol is evident. This decreases to approximately 150 turnovers at 180 $\mu$M ubiquinol.

We also considered the possibility that the inhibitory effect of ubiquinol could be explained by a model in which there is double occupancy of the center P site by ubiquinol (16). If the $S. pombe$ iron-sulfur protein permits access of the quinol substrate to half of the double occupancy site which is otherwise accessible only to ubisemiquinone generated as a transient intermediate during ubiquinol oxidation, this might block reduction of the cytochrome $b_{566}$ heme by the semiquinone. Although the semiquinone at center P is of sufficiently low potential that it would not effectively convert the interfering quinol itself to a semiquinone reductant for the cytochrome, it would be expected to escape from center P and either undergo dismutation or react spuriously with high potential oxidants. Either of the latter two possibilities should diminish the antimycin sensitivity of the cytochrome $c$ reductase reaction, but we found that the efficacy of inhibition by antimycin was unchanged by the $S. pombe$ iron-sulfur protein.

The efficacy of cytochrome $c$ reductase inhibitors myxothiazol and UHDBT is also unchanged by the $S. pombe$ protein, and the $S. pombe$ protein restores the optical spectrum of cytochrome $b$. These latter results indicate that the $S. pombe$ protein interacts with cytochrome $b$ to form a quinol oxidase site, center P, which is indistinguishable from that formed by the $S. cerevisiae$ protein. We thus conclude that, while our results do not argue against a double occupancy model, the inhibitory effect of ubiquinol is better explained by impaired electron transfer from the Rieske center to cytochrome $c$.

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