In the yeast *Saccharomyces cerevisiae*, the most important systems for conveying excess cytosolic NADH to the mitochondrial respiratory chain are the external NADH dehydrogenases (Nde1p and Nde2p) and the glycerol-3-phosphate dehydrogenase shuttle. In the latter system, NADH is oxidized to \( \text{NAD}^+ \) and dihydroxyacetone phosphate is reduced to glyceraldehyde-3-phosphate by the cytosolic Gpd1p. Subsequently, glyceraldehyde-3-phosphate donates electrons to the respiratory chain via mitochondrial glyceraldehyde-3-phosphate dehydrogenase (Gut2p). At saturating concentrations of NADH, the activation of external NADH dehydrogenases completely inhibits glyceraldehyde-3-phosphate oxidation. Studies on the functionally isolated enzymes demonstrated that neither Nde1p nor Nde2p directly inhibits Gut2p. Thus, the inhibition of glyceraldehyde-3-phosphate oxidation may be caused by competition for the entrance of electrons into the respiratory chain. Using single deletion mutants of Nde1p or Nde2p, we have shown that glyceraldehyde-3-phosphate oxidation via Gut2p is inhibited fully when NADH is oxidized via Nde1p, whereas only 50% of glyceraldehyde-3-phosphate oxidation is inhibited when Nde2p is functioning. By comparing respiratory rates with different respiratory substrates, we show that electrons from Nde1p are favored over electrons coming from Ndp1 (internal NADH dehydrogenase) and that when electrons come from either Nde1p or Nde2p and succinodihydronicotinamide, their use by the respiratory chain is shared to a comparable extent. This suggests a very specific competition for electron entrance into the respiratory chain, which may be caused by the supramolecular organization of the respiratory chain. The physiological consequences of such regulation are discussed.

The yeast *Saccharomyces cerevisiae* lacks transhydrogenase activity (1, 2), and the redox couple NADH/NAD+ cannot pass through the mitochondrial membrane. Hence, systems for NADH turnover in mitochondria as well as in the cytosol are required under both aerobic and anaerobic conditions. The reason for this is that several processes result in production of NADH, i.e., several processes are, contrary to ethanol fermentation, not redox neutral. The synthesis of 1 mol of glycerol, the second major by-product of *S. cerevisiae* cells fermenting glucose, results in the consumption of 1 mol of NADH, whereas other by-products such as acetate lead to the production of cytosolic NADH. The largest part of excess cytosolic NADH formation is connected to biomass production (3, 4). The synthesis of proteins and nucleic acids and even the highly reduced lipids is associated with assimilatory NADH production. In particular, NADH is generated in the biosynthetic pathways of amino acid synthesis (3, 4). Anaerobically, the only means by which *S. cerevisiae* can reoxidize surplus production of NADH is by glycolysis (2, 5). Aerobically, several systems exist for conveying excess cytosolic NADH to the mitochondrial electron transport chain in *S. cerevisiae* (6). The two most important systems in this respect seem to be the external NADH dehydrogenase (Nde1p/Nde2p) (7, 8) and the glyceraldehyde-3-phosphate shuttle (9). The Nde1p/Nde2p system, which is localized in the inner mitochondrial membrane with the catalytic sites projecting toward the intermembrane space, has been shown to directly oxidize cytosolic NADH (7, 8). The glyceraldehyde-3-phosphate shuttle system, which involves the FAD-dependent Gut2p (10), is situated in the inner membrane of the mitochondria with the catalytic site projecting toward the cytosol and has been shown to be active in maintaining a cytosolic redox balance (9). In this system, the co-factor NADH is oxidized to \( \text{NAD}^+ \) by the mitochondrial glyceraldehyde-3-phosphate dehydrogenase (Gpd1p) when catalyzing the reduction of dihydroxyacetone phosphate (DHAP) to glyceraldehyde-3-phosphate. Thus, glyceraldehyde-3-phosphate delivers its electrons to the respiratory chain via Gut2p with the subsequent regeneration of DHAP.

Previously, we have shown (10) kinetic interactions between Nde1p/Nde2p and Gut2p. Our data demonstrate that the deletion of either one of the external dehydrogenases caused an increase in the efficiency of the remaining enzyme. In addition, we have shown that the activation of NADH dehydrogenase inhibits the Gut2p in such a manner that, at a saturating concentration of NADH, glyceraldehyde-3-phosphate is not used as a respiratory substrate. This effect is not a consequence of a direct action of NADH on Gut2p activity because both NADH dehydrogenase and its substrate are needed for Gut2p inhibition (10).

The present study raised three distinct questions, which we address in this report. 1) Is the role of each of the external NADH dehydrogenases (Nde1p/Nde2p) equivalent in this process? 2) Is the kinetic regulation of the activity of one enzyme as a function of the rate of another enzyme having the same physiological function as that allowed by their association in the same supramolecular complex in the inner membrane, or is it regulated by competition in the supply of electrons to the respiratory chain? 3) Does this competition in electron supply between the two external NADH dehydrogenases and Gut2p...
also apply to the internal dehydrogenases (Ndhp and/or succinodehydrogenase)?

Using two single mutants, Δnde1 and Δnde2, we were able to show that Nde1p was the enzyme involved in the complete inhibition of Gut2p, whereas when Nde1p was absent, the respiratory rate was caused equally by Nde2p and Gut2p.

Moreover, when they were functionally isolated, all the considered dehydrogenases were insensitive to each another, pointing out that the inhibition during oxygen consumption was linked to a competition for electron supply to the respiratory chain. In the presence or absence of one of the external dehydrogenases, Ndhp activity was affected in a way that was comparable with Gut2p. When the activity of succinodehydrogenase was considered, succinate oxidation was increased in the absence of Nde1p. Moreover, although Nde2p was able to inhibit substrate oxidation for all other dehydrogenases considered, it was not able to inhibit succinate oxidation.

### EXPERIMENTAL PROCEDURES

#### Yeast Strains and Growth Conditions—The S. cerevisiae strains used in this study were all derived from W303–1A (Table I). The cells were cultivated as batch cultures in a defined medium, yeast nitrogen base (Difco), using 5 g/liter glucose as the carbon and energy source. The cells were harvested subsequent to glucose depletion but after the diauxic shift occurred during respiratory growth on ethanol.

#### Preparation of Mitochondria—Yeast mitochondria (S. cerevisiae) were prepared as described previously (11) and suspended in Medium 1 (0.65 M mannitol, 0.36 mM EGTA, 10 mM Tris-maleate, 5 mM Tris-phosphate, pH 6.8).

#### Respiration Assay—Oxygen consumption was measured at 28°C in a 2-ml thermostatically controlled chamber equipped with a Clark oxygen electrode (Gilson) connected to a microcomputer, which provided an on-line display of rate values. Mitochondria (0.5 mg/ml) were incubated in Medium 1. The substrate supply consisted of either various concentrations of NADH, glycerol 3-phosphate, ethanol, and succinate or an NADH-regenerating system (4 mM glucose 6-phosphate, 2 mM NAD⁺, and various amounts of glucose-6-phosphate dehydrogenase (EC 1.1.1.49) from Leuconostoc mesenteroides, which is able to work with NAD+ as a co-factor).

#### Enzymatic Activities—NADH-ferricyanide reductase and glycerol 3-phosphate-ferricyanide reductase were assayed in Medium 1 on mitochondria in the presence of 0.2 μg/ml antimycin and 1 μM KCN and different concentrations of NADH and in the presence or absence of a saturating concentration (75 mM) of glycerol 3-phosphate. The reaction was started by the addition of 1.5 mM ferricyanide, and its reduction was followed at 436 nm (ε = 0.21 mM⁻¹·cm⁻¹).

#### Measurement of DHAP—DHAP was measured spectrophotometrically in neutralized HClO₄/EDTA extracts as described previously by Bergmeyer (12). NAD⁺ was measured fluorometrically in neutralized HClO₄/EDTA extracts as described previously (12).

### RESULTS

#### Competition between Either Nde1p or Nde2p and Gut2p for Respiration—We have shown previously (10) that the activation of external NADH dehydrogenase inhibits Gut2p in such a manner that, at a saturating concentration of NADH, glycerol 3-phosphate is no longer used as a respiratory substrate. This effect is not a consequence of a direct action of NADH on Gut2p because both NADH dehydrogenase and its substrate are needed for this inhibition. Because NADH dehydrogenase activity is caused by Nde1p and Nde2p, the question arises as to the respective role of each of these enzymes in such an inhibition process. Using a NADH-regenerating system (see “Experimental Procedures”), different steady states of oxygen consumption could be obtained depending on the NADH concentration produced by the glucose-6-phosphate dehydrogenase. At a saturating concentration of glycerol 3-phosphate, the respiratory rate was modulated by adding different amounts of NADH-producing enzyme. For each experimental condition, the respiratory rate and the glycerol 3-phosphate consumption (i.e. the dihydroxyacetone phosphate formation flux) were determined (Fig. 1A). Because oxygen consumption in the presence of both NADH and glycerol 3-phosphate was a combination of these two substrates, we quantified glycerol 3-phosphate oxidation via dihydroxyacetone phosphate production (see Fig. 1A). The NADH-linked respiratory rate was evaluated by the difference between the total respiratory rate and the dihydroxyacetone phosphate formation flux. This technique is validated in Table II, which shows that when glycerol 3-phosphate is the sole substrate, the respiratory rate is equal to the dihydroxyacetone phosphate production flux. Fig. 1, B and C, shows typical experiments of non-phosphorylating respiratory rates of both glycerol 3-phosphate and NADH, using mitochondria isolated from either the Δnde1 strain (Nde2p) or the Δnde2 strain (Nde1p). When only Nde2p was present, the addition of increasing concentrations of glucose-6-phosphate dehydrogenase to mitochondria respiring on glycerol 3-phosphate led to a slight increase in the total oxygen consumption (Fig. 1B and Table II) and a decrease in glycerol 3-phosphate oxidation associated with an increase in NADH oxidation. At the saturating concentration of both substrates, the part of oxygen consumption linked to each substrate was comparable (Table II). Moreover, the remaining respiration represented 57% of the maximal respiration for glycerol 3-phosphate and 62% for NADH (Table II). When only Nde1p was present, the addition of increasing concentrations of glucose-6-phosphate dehydrogenase to mitochondria respiring on glycerol 3-phosphate led to a drastic increase in the total oxygen consumption (Fig. 1C and Table II) and a complete inhibition in glycerol 3-phosphate oxidation associated with an increase in NADH oxidation. It should be noted that in the presence of glycerol 3-phosphate, the optimal respiration on NADH was stimulated (26%) (Table II). These results showed that both enzymes behaved very differently and that in wild type mitochondria, Nde1p was the enzyme responsible for the total inhibition of glycerol 3-phosphate oxidation when NADH was present.

The competition between Nde1p and Gut2p for Respiration Is Not Linked to an Interaction between These Enzymes—Previous studies (13) have shown that Nde1p, Nde2p, and Gut2p are part of a supramolecular complex that can be isolated from yeast mitochondria. This implies that these enzymes are able to interact functionally with each other. Thus, when Nde1p oxidizes NADH, it is possible that it inhibits glycerol 3-phosphate oxidation by Gut2p. To test this hypothesis, we measured the activity of the dehydrogenases that were functionally isolated in the presence of antimycin A and KCN, to inhibit the respiratory chain. In these conditions, the reduction of an artificial electron acceptor (ferricyanide) reflects the activity of the external dehydrogenases. However, we observed that ferricyanide itself inhibited the NADH-regenerating system (data not shown). To bypass this problem, increasing concentrations of NADH were added directly to isolated mitochondria. To measure electron flux from NADH dehydrogenases when both Nde1p or 2, and Gut2p were functioning, we measured the NAD⁺ formation flux (Fig. 2A). Moreover, as shown in Fig. 2A, it must be stressed that in the presence of Fe(CN)₆⁻ plus antimycin A plus KCN, no electron could go through the respiratory chain, and we assessed the actual activity of each dehydrogenase. Moreover, as shown in Fig. 2A, NAD⁺ quantification al-

### Table I

| Strain            | Description                                      |
|-------------------|--------------------------------------------------|
| Wild type         | ade2–1, his3–11, 112trp1–1a, ura3–1, can100     |
| Δnde1             | ade2–1, his3–11, 112trp1–1a, can100, Nde1Δ:TRP1  |
| Δnde2             | ade2–1, his3–11, 112trp1–1a, can100, Nde2Δ:LEU2  |

S. cerevisiae strains used in this study

All strains were derived from W303–1A.
followed us to determine the activity of Nde1p or Nde2p; the Fe(CN)₆⁻ reduction rate represents both NADH oxidation and Gut2p activity. Thus, the difference between $\text{J}_{\text{Fe(CN)}_6}$ and $\text{J}_{\text{NADH}/\text{H}^+}$ represents $\text{J}_{\text{DHAP}}$. Fig. 2, B and C, shows that for any NADH concentration and any enzyme (Nde1p or Nde2p), glycerol 3-phosphate oxidation was constant. In these experimental conditions, no inhibition of Gut2p was observed, showing that the inhibition of glycerol 3-phosphate oxidation (see Fig. 1) did not come from physical interaction between these enzymes. Because the electron transfer between Gut2p and O₂ was inhibited by Nde1p, the only explanation is a competition in electron transfer to the respiratory chain.

**Internal Dehydrogenases Are Also Involved in the Competition for Electron Supply to the Respiratory Chain**—To further study this competition process, we asked whether internal dehydrogenases (i.e. internal NADH dehydrogenase, Ndip, and succinodehydrogenase) could be involved. Both the respiratory rate ($\text{J}_{\text{O}_2}$) and the NADH oxidation flux ($\text{J}_{\text{NADH}/\text{H}^+}$) were measured in the presence of an internal substrate (either ethanol or succinate) and NADH (see Fig. 3) on wild type mitochondria (Table III). When NADH was the sole substrate, $\text{J}_{\text{NAD}^+}$ was the exact measure of the electron flux from NADH to oxygen.
In the presence of both ethanol and NADH, the respiratory rate was essentially the result of external NADH oxidation, and the electron flux from Ndip was inhibited strongly (82%) (Table III). In contrast, in the presence of both NADH and succinate, the respiratory rate was inhibited drastically by external NADH dehydrogenase, whereas when succinodehydrogenase is active in conjunction with external dehydrogenases, it is inhibited in the same way as they are. Because electron fluxes from Nde1p and Nde2p play distinct roles in glycerol 3-phosphate oxidation inhibition, we asked whether their behavior was comparable in the case of succinate. Gut2p has a stationary behavior was not observed for succinate, supports the specific organization of the dehydrogenases and respiratory chain. Previous work (16–18) has shown that complexes III and IV are assembled into large supercomplexes. Our hypothesis is that these supercomplexes can interact specifically with either complex II to form a respiratory chain using succinate or with the other supercomplex containing most of the dehydrogenases, i.e. Nde1p, Nde2p, Ndip, and Gut2p. For the sake of comprehension, two distinct phenomena should be considered here. First, the present study was conducted under a non-phosphorylating respiratory rate condition, under which the respiratory rate compensated the energy waste. Thus, the respiratory rate cannot be increased indefinitely, which explains why the dehydrogenases have to share the electron flux. However, some of these dehydrogenases have a full priority in electron transfer to the respiratory chain. In regard to our hypothesis, this priority implies that the dehydrogenases involved belong to the same supermolecular complex. This is reinforced by the fact that the dehydrogenases themselves are not able to inhibit each other. Second, and this is a different situation, if one considers the case of succinate, the succinodehydrogenase can belong to a different supermolecular complex, and the electron flux is then shared between the different dehydrogenases involved.

### DISCUSSION

In yeast mitochondria, which lack a proton pumping complex I, there are two external NADH dehydrogenases (Nde1p and Nde2p) and one internal NADH dehydrogenase (Ndip). Both external dehydrogenases share the same function, leaving one to wonder about the role of this redundancy. Previous studies (8) have shown that Nde1p is the main enzyme necessary for cytosolic NADH reoxidation during growth on a non-fermentable carbon source. However, NDE2 gene expression is induced after the diauxic shift (15), i.e. for growth on ethanol. Thus, under these conditions, which were the conditions used in the present study, both enzymes were present. In this study, we showed that mitochondria bearing only the Nde1p or the Nde2p enzyme had a comparable oxygen consumption rate on NADH. Even though both enzymes share the same function, they did not have the same role in regulating oxygen consumption fluxes. We have shown that both Gut2p and Ndip were inhibited when functioning in conjunction with Nde1p, whereas Ndip2p did not have such an effect. This could have been caused either by enzyme interactions, because they have been shown to belong to the same supermolecular complex, or by electron transfer competition. Clearly, when it was functionally isolated, Gut2p was not inhibited by Nde1p, ruling out the enzyme interaction hypothesis. Thus, the only remaining hypothesis that can explain our results is competition between dehydrogenases for the electron supply to the respiratory chain. We have shown that electrons coming from Nde1p have the right of way over those coming from either Gut2p or Ndip. Finally, this shows that even though both Nde1p and Nde2p share the same function, they do not have the same role in providing electrons to the respiratory chain and/or reoxidizing NADH. Interestingly, when both NADH and succinate were used as substrates at the same time, the resulting oxygen consumption rate was the sum of 60% of each of the individual (NADH or succinate) respiratory rates. Under these conditions, there did not seem to be any priority in electron transfer. That electrons coming from Nde1p had the right of way over electrons coming from Gut2p and Ndip, although this peculiar behavior was not observed for succinate, supports the specific organization of the dehydrogenases and respiratory chain.

### Table II

| Strain | Substrates | \( J_{O_2} \) |JDHAP | \( J_{O_2} - J_{DHAP} \) |
|--------|------------|--------------|-------|---------------------|
| \( \Delta nde1 \) | NADH | 232 ± 19 | 0 | 232 ± 19 |
| | G, P | 223 ± 17 | 226 ± 21 | 3 ± 20 |
| | NADH + G, P | 270 ± 51 | 127 ± 15 | 143 ± 17 |
| | Ndip | 242 ± 26 | 0 | 242 ± 26 |
| \( \Delta nde2 \) | G, P | 158 ± 18 | 149 ± 30 | 9 ± 19 |
| | NADH + G, P | 304 ± 32 | 0 | 295 ± 34 |

(\( J_{O_2} \)). In the presence of both ethanol and NADH, the respiratory rate was essentially the result of external NADH oxidation, and the electron flux from Ndip was inhibited strongly (82%) (Table III). In contrast, in the presence of both NADH and succinate, the resulting respiratory rate accounted for both substrates in which the utilization was inhibited by only roughly 30% (Table III). As shown above for Gut2p, Ndip is inhibited drastically by external NADH dehydrogenases, whereas when succinodehydrogenase is active in conjunction with external dehydrogenases, it is inhibited in the same way as they are. Because electron fluxes from Nde1p and Nde2p play distinct roles in glycerol 3-phosphate oxidation inhibition, we asked whether their behavior was comparable in the case of Ndip. Table IV shows that Nde1p alone was able to inhibit ethanol oxidation in a way that was comparable with glycerol 3-phosphate oxidation (see Table II). In mitochondria isolated from the mutant \( \Delta nde2 \), the respiratory rates with different substrates were similar to the ones in wild type mitochondria, except when succinate was used as the substrate (30% stimulation). However, in these mitochondria, the respiratory rate with NADH plus succinate has characteristics comparable with the wild type mitochondria. Moreover, when only Nde2p was present, at a saturating concentration of ethanol and NADH, the part of oxygen consumption linked to each substrate was comparable (Table IV), which is similar to what was shown with glycerol 3-phosphate and NADH (see Table II). In mitochondria isolated from the \( \Delta nde1 \) strain, the respiratory rate with succinate as a substrate was increased compared with both wild type and \( \Delta nde2 \). When both Ndip and succinate were used as substrates, the respiratory rate was high, and succinate was the main substrate that was oxidized (Table IV).
FIG. 2. Absence of direct inhibition between Nde1-2p and Gut2p. A, the technique used to assess the various fluxes is shown. The enzymes were functionally isolated by respiratory chain inhibition with antimycin A and KCN. NADH oxidation flux was assessed via NADH production flux, as described under “Experimental Procedures.” Fe(CN)$_6^-$ represents the sum of NADH and glycerol 3-phosphate (G$_3$P) oxidation. Thus the glycerol 3-phosphate oxidation rate via Gut2p is the difference between the Fe(CN)$_6^-$ reduction flux (divided by 2 because Fe(CN)$_6^-$ only accepts one electron) and the NADH production flux. OUT, outer face of the inner membrane; IN, inner face of the inner membrane. NADH and glycerol-3-phosphate dehydrogenase activities as a function of NADH concentration are shown in mitochondria isolated from Δnde1 (B) or Δnde2 (C). Mitochondria (0.5 mg/ml) were incubated in Medium 1 in the presence of antimycin A and potassium cyanide (see “Experimental Procedures”). In a first set of experiments, ferricyanide reduction (▲) and NADH production (●) were measured in the presence of 75 mM glycerol 3-phosphate. In a second set of experiments, ferricyanide reduction was measured in the absence of glycerol 3-phosphate (△). Dehydrogenase activities were expressed in micromoles of the substrate (NADH or glycerol 3-phosphate), consumed·min$^{-1}$·mg protein$^{-1}$ (i.e. ferricyanide production flux/2). This figure represents a typical experiment representative of three such independent experiments.
Respiration rates and NAD\(^+\) production flux in isolated mitochondria from wild type strains

Mitochondria, 0.5 mg ml\(^{-1}\), were incubated in Medium 1 (see "Experimental Procedures"). Substrate supply was 1 mM NADH and/or 100 mM ethanol and/or 10 mM succinate. JO\(_2\), was expressed in natomes, O\(^{-}\) min \(^{-1}\) mg protein\(^{-1}\), and JNAD\(^+\) was expressed in nmol, NAD\(^+\) min \(^{-1}\) mg protein\(^{-1}\). Results are means \(\pm\) S.D. of at least three independent experiments performed on three different mitochondrial preparations.

| Substrates          | JO\(_2\)   | JNAD\(^+\) | JO\(_2\)–JNAD\(^+\) |
|---------------------|------------|------------|---------------------|
| NADH                | 233 \(\pm\) 9 | 234 \(\pm\) 45 | 0                   |
| EtOH                | 198 \(\pm\) 8 | 0          | 198 \(\pm\) 8       |
| NADH + EtOH         | 260 \(\pm\) 8 | 224 \(\pm\) 20 | 36 \(\pm\) 6        |
| Succinate           | 131 \(\pm\) 3 | 0          | 131 \(\pm\) 3       |
| NADH + succinate    | 246 \(\pm\) 8 | 161 \(\pm\) 20 | 86 \(\pm\) 12       |

and thus has the right of way over other substrates such as the matricial NADH. This tight organization, which favors the reoxidation of cytosolic NADH, has important consequences on the functioning of the Krebs cycle and possibly on the redux status of the mitochondrial matrix. Indeed, when cytosolic NADH concentrations are high, the functioning of the Krebs cycle might be altered (consequent to the inability of matricial NADH reoxidation), and it has been shown that in this condition the Krebs cycle does not operate as a cycle but rather as two branches, one oxidative and the other one reducing (20). Furthermore, when cytosolic NADH is lowered, the Krebs cycle returns to functioning as a cycle (20). Thus, cytosolic NADH concentration, through Nde1p, can regulate mitochondrial oxidative metabolism. Under physiological conditions, the kinetic constraints for electron transfer at the level of the respiratory chain should lead to a higher redux potential in mitochondria than in the cytosol.

It is worth noting that succinate oxidation is not inhibited by electron transfer from other dehydrogenases and seems to play a peculiar role in yeast. It has been shown that the mitochondrial fumarate/succinate exchange is essential for the growth of yeast on ethanol or acetate as the sole carbon source (14). Thus, the succinodehydrogenase activity is also essential in this condition.

In conclusion, under physiological conditions, in which any of the dehydrogenase substrate is available, our work shows that the metabolic organization of the respiratory chain is such that it allows a selection and a priority in electron supply. This is a new mechanism of regulation of the yeast oxidative metabolism, which should be reconsidered in light of these results, taking into account that there is a competition between the mitochondrial dehydrogenases.

### References

1. Bruinenberg, P. M., Jonker, J., van Dijken, J. P., and Scheffers, W. A. (1985) Arch. Microbiol. 142, 302–306
2. van Dijken, J. P., and Scheffers, W. A. (1986) FEMS Microbiol. Rev. 32, 199–225
3. Albers, E., Larsson, C., Lidén, G., Niklasson, C., and Gustafsson, L. (1996) Appl. Environ. Microbiol. 62, 1387–1395
4. Albers, E., Lidén, G., Larsson, C., and Gustafsson, L. (1998) Recent Res. Dev. Microbiol. 2, 253–279
5. Nordström, K. (1968) J. Inst. Brew. 74, 429–432
6. Bakker, B. M., Overkamp, K. M., van Maris, A. J., Kotter, P., Luttik, M. A., van Dijken, J. P., and Pronk, J. T. (2001) FEMS Microbiol. Lett. 20, 25–33
7. Luttik, M. A. H., van Dijken, K. M., Kotter, P., de Vries, S., van Dijken, J. P., and Pronk, J. T. (1998) J. Biol. Chem. 273, 24529–24534
8. Small, W. C., and McAlister-Henn, L. (1998) J. Bacteriol. 180, 4051–4055
9. Larsson, C., Pålman, L.-L., Larsson, C., Averet, N., Bønou, O., Boebeke, S., Gustafsson, L., and Rigoulet, M. (2002) J. Biol. Chem. 277, 27991–27995
10. Guérin, B., Labbé, P., and Somlo, M. (1979) Methods Enzymol. 55, 149–159
12. Bergmeyer, H. U. (1988) in Methods of Enzymatic Analysis (Bergmeyer, H. U., Bergmeyer, J., and Grabl, M., eds) Vol. VI, 3rd Ed., pp. 342–350, VCH Publishers, Weinheim, Germany
13. Grandier-Vazeille, X., Bathany, K., Chaignepain, S., Camougrand, N., Manon, S., and Schmitter, J. M. (2001) Biochemistry 40, 9758–9769
14. Palmieri, L., Lasorsa, F. M., De Palma, A, Palmieri, F., Runswick, M. J., and Walker, J. E. (1997) FEBS Lett. 417, 114–118
15. DeRisi J. L., Iyer, V. R., and Brown, P. O. (1997) Science 278, 680–686
16. Schagger, H., and Pfeiffer, K. (2000) EMBO J. 19, 1777–1783
17. Schagger, H. (2001) IUBMB Life 52, 119–128
18. Schagger, H. (2002) Biochim. Biophys. Acta 1555, 154–159
19. AveRET, N., Aguilaniu, H., Bunouf, O., Gustafsson, L., and Rigoulet, M. (2002) J. Bioenerg. Biomembr. 34, 499–506
20. Gombert, A. K., Moreira dos Santos, M., Christensen, B., and Nielsen, J. (2001) J. Bacteriol. 183, 1441–1451
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CEREVISIAE

MECHANISM OF OXIDATIVE METABOLISM IN SACCHAROMYCES

Competition of Electrons to Enter the Respiratory Chain: A NEW REGULATORY

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