Characterization of a High Capacity Calcium Transport System in Mitochondria of the Yeast *Endomyces magnusii*\(^*\)

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The Ca\(^{2+}\) transport system of *Endomyces magnusii* mitochondria has been shown previously to be activated by spermine. Here we report it to be regulated also by low, physiological ADP concentrations, by the intramitochondrial NADH/NAD\(^+\) ratio, and by Ca\(^{2+}\) ions. The combination of all these physiological modulators induced high initial rates of Ca\(^{2+}\) uptake and high Ca\(^{2+}\)-buffering capacity of yeast mitochondria, enabling them to lower the medium [Ca\(^{2+}\)] to \(-0.2\) \(\mu\)M. The mechanisms of stimulation by these agents are discussed.

In animal cells, Ca\(^{2+}\) acts as an important second messenger in signal transduction. The signal is relayed to the mitochondrial matrix by the calcium uniporter (1, 2), which in the fast mode may respond to increases of [Ca\(^{2+}\)] from the resting level of \(-100\) nM to a few hundred nM or more in calcium waves (3). An increased matrix [Ca\(^{2+}\)]\(^1\) then activates Ca\(^{2+}\)-sensitive enzymes, i.e. various dehydrogenases, pyrophosphatase, and ATP synthase. This constitutes a mechanism for the short term regulation of cellular respiration and oxidative phosphorylation (4).

Yeast and fungal cells also have elements of a Ca\(^{2+}\)-based signal transduction system that functions in a manner similar to that of animal cells, except that the vacuolar system seems to have replaced the endoplasmatic reticulum (5) as the major Ca\(^{2+}\)-sequestering organelle (6). In the yeast *Saccharomyces cerevisiae*, [Ca\(^{2+}\)]\(^1\) was found to be very low (100 nM) and tightly controlled by transport processes across the plasma membrane and tonoplast membranes (7). A transient increase in the [Ca\(^{2+}\)] was found to be an indispensable response of *S. cerevisiae* cells to mating pheromone (7, 8), and in initiating morphogenetic processes (9, 10). Participation of [Ca\(^{2+}\)] in signal transduction also in yeast is thus gaining acceptance.

Mitochondria from *S. cerevisiae* and *Candida utilis* yeast cells were able to accumulate Ca\(^{2+}\) only slowly, even from unphysiologically high (mM) concentrations (11), indicating that the uptake lacked physiological significance. In our laboratory, it was found that *Endomyces magnusii* yeast cells had mitochondria capable of accumulating Ca\(^{2+}\) from moderate concentrations. This yeast species has large (as implied by the name *magnusii*) polynucleated cells, with a fully competent oxidative phosphorylation system having three conservation sites in the respiratory chain like animal mitochondria and no alternative electron transfer pathways (12). The affinity of the Ca\(^{2+}\)-transporting system for Ca\(^{2+}\) ions was rather low, with an apparent \(K_m\) value of 150–180 \(\mu\)M (13, 14). The Ca\(^{2+}\) uptake process was energy-dependent since it was strongly inhibited by omission of oxidizable substrate or by the presence of a respiratory inhibitor or uncoupler. Ca\(^{2+}\) uptake was associated with a transient depolarization of the inner mitochondrial membrane, reversible oxidation of cytochrome \(b\), and stimulation of respiration. This implies an uniport mechanism, in which Ca\(^{2+}\) uptake is driven electrochemically by the membrane potential, negative on the matrix side. Evidence was subsequently presented that the affinity of the Ca\(^{2+}\) transport system was increased in the presence of low concentrations of polyanymes below 25 \(\mu\)M spermine or 100 \(\mu\)M spermidine (15, 16). This prompted us to search more systematically for naturally occurring modulators of the Ca\(^{2+}\)-transporting system in these mitochondria. Here we report that ADP, Ca\(^{2+}\) ions, and exogenous NADH also may serve as potent activators of the *E. magnusii* mitochondrial calcium transporter. Some of these data have been reported at meetings (17, 18).

**EXPERIMENTAL PROCEDURES**

*E. magnusii* yeast, strain VKM Y261, was cultivated in agitated Erlenmeyer flasks at 28 °C in batches of 100 ml in a medium (18) containing glycerol (0.6–1.0%, v/v) as the source of carbon and energy. Cells were harvested at the late exponential growth phase when the cell density corresponded to 10–13 g wet weight/liter. Mitochondria were isolated by the method designed in our laboratory. Cells were incubated at room temperature for 30 min in 50 mM Tris-HCl buffer, pH 8.6, containing 10 mM dithiothreitol, washed twice with distilled water, resuspended in 50 mM Tris-EDTA buffer, pH 5.8–5.9, containing 1.2 mM sorbitol and 50 mg of helicase per g of original cell (wet weight), and incubated at 30 °C under mild stirring for 15–20 min to form spheroplasts. After centrifugation at 400 \(\times\) \(g\) for 18 min, the pellets were washed twice with 1.2 mM sorbitol containing 0.4% (w/v) bovine serum albumin, and pH was adjusted to 6.0. The pellet was further homogenized with a Dounce homogenizer in a medium containing 10 mM Tris-HCl buffer, pH 7.2, 0.4 mM mannitol, 0.5 mM EDTA, 0.5 mM EGTA, and 0.4% bovine serum albumin. The homogenate was mixed with an equal volume of the same buffer, except that 0.4 mM mannitol was substituted for 0.6 mM mannitol, centrifuged for 10 min at 1200 \(\times\) \(g\), and the supernatant centrifuged at 6000 \(\times\) \(g\) for 18 min. The pellets from the second spin were gently resuspended in approximately 20 ml of washing medium (EDTA and EGTA were excluded), recentrifugated at 6000 \(\times\) \(g\) for 18 min, and resuspended in a minimal volume of washing medium. The mitochondria thus obtained met all known cri-

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\(^{1}\) The abbreviations used are: [Ca\(^{2+}\)], free intracellular Ca\(^{2+}\) concentration; CSA, cyclosporin A; MPT, mitochondrial permeability transition.

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Uptake of Ca\(^{2+}\) by Yeast Mitochondria—Fig. 1 shows energy-dependent Ca\(^{2+}\) uptake by *E. magnusii* mitochondria respiring on pyruvate + malate. The addition of 100 \(\mu\)M Ca\(^{2+}\) caused an abrupt decrease in absorbance, reflecting the formation of the Ca\(^{2+}\)-murexide complex, followed by an increase in absorbance due to Ca\(^{2+}\) accumulation by mitochondria until a steady state was reestablished. The net efflux rate of Ca\(^{2+}\) even after accumulation of 300 \(\mu\)M of Ca\(^{2+}\) in the presence of inorganic phosphate or acetate on inhibition of uptake by anaerobiosis was very low, 17–18 nmol/min \cdot mg\(^{-1}\), the efflux being inhibitable by addition of Mg\(^{2+}\), Mn\(^{2+}\), or La\(^{3+}\) (data not shown), indicating that changes in the initial rates of net Ca\(^{2+}\) uptake were due to changes in influx rates. All Ca\(^{2+}\) accumulated was released in response to addition of the specific Ca\(^{2+}\) ionophore A23187, indicating the intramitochondrial localization of the Ca\(^{2+}\) accumulation.

The Stimulation of Ca\(^{2+}\) Uptake in Yeast Mitochondria by ADP—The Ca\(^{2+}\) accumulation by yeast mitochondria was enhanced by addition of 25 \(\mu\)M ADP (Fig. 1, traces a and b, and Fig. 2A). The effect of ADP was specific (other examined mononucleotides, including AMP, cAMP, GMP, GDP, IDP, ATP, or GTP, were inactive; data not shown), saturated at very low concentrations (with a half-maximal stimulation at 2–3 \(\mu\)M) (Fig. 3). Addition of 100 mM KCl slightly increased the affinity for ADP (data not shown). The ADP-enhanced Ca\(^{2+}\) uptake by yeast mitochondria was strongly prevented by low concentrations of atractyloside (Fig. 4), a specific inhibitor of adenine nucleotide translocator (23), but not by CSA, a potent and seemingly specific inhibitor of the Ca\(^{2+}\)-induced, phosphate-dependent permeabilization (MPT, pore) of the inner mitochondrial membrane (Fig. 4). These data appear to indicate that ADP acted from the matrix side and stimulated the electrogenic Ca\(^{2+}\) uptake mechanism itself. In the presence of ADP, the uptake kinetics were changed from sigmoidal to hyperbolic (Fig. 2A), the Hill coefficient being lowered from 2.59 to 2.11, and the \(V_{\max}\) being substantially increased. ADP thus

![Diagram](https://example.com/diagram.png)
moderately improved the Ca\(^{2+}\)-buffering capacity of mitochondria, i.e. their ability to maintain a low extramitochondrial [Ca\(^{2+}\)] (Fig. 1, traces a and b; Fig. 2B).

The Stimulation of Ca\(^{2+}\) Uptake in Yeast Mitochondria by NADH—Normally, freshly isolated *E. magnusii* yeast mitochondria showed a rather low intramitochondrial NADH/NAD ratio, which, however, could be considerably increased by short term incubation with NADH (data not shown). Yeast mitochondria, unlike many animal mitochondria, readily oxidize exogenous NADH. The dehydrogenase responsible for its oxidation is located on the outer surface of the inner mitochondrial membrane and donates electrons to the respiratory chain at the level of coenzyme Q, bypassing the site I of energy conservation (12). In mitochondria from glycerol-grown *E. magnusii* cells, NADH along with glycerol 1-phosphate is the most rapidly oxidizable substrate “monopolizing” the respiratory chain and potently inducing reversed electron flow (24, 25). To have a higher NADH/NAD\(^+\) ratio in the matrix, the mitochondrial suspension was incubated with 4 m\(M\) NADH for 0.5 min (this is sufficient to effectively induce reversed electron flow; Ref. 25) and then used a small aliquot of the suspension (50-fold dilution) to avoid external NADH serving as an additional respiratory substrate. Mitochondria treated in this way exhibited very high rates of Ca\(^{2+}\) uptake (Fig. 5A) and a slightly improved Ca\(^{2+}\)-buffering capacity (Fig. 5B). The Ca\(^{2+}\) affinity of the transporter was increased, and half-maximal rate of uptake was obtained at 100 \(\mu\)M, while in the control 155 \(\mu\)M was required, but the Hill coefficient was not changed. The stimulatory effect of NADH was not due to ADP contamination in the NADH preparation used.

The Stimulation of Ca\(^{2+}\) Uptake in Yeast Mitochondria by Ca\(^{2+}\)-Ca\(^{2+}\)-ion-transport inhibitors were found to modulate the yeast mitochondrial Ca\(^{2+}\) transporter. Moderate amounts of Ca\(^{2+}\), added sequentially, progressively increased the uptake rates and lowered the external steady state Ca\(^{2+}\) concentrations (Fig. 6, A and B, left traces). Similarly, preincubation of mitochondria for 1 min with 20 \(\mu\)M Ca\(^{2+}\) accelerated the uptake of moderate (50 and 100 \(\mu\)M) Ca\(^{2+}\) concentrations (Fig. 1, trace c, and Fig. 7A), the [Ca\(^{2+}\)] at V\(_{1/2}\) being lowered to 63 \(\mu\)M without changing the Hill coefficient. Remarkably, such a pretreatment of mitochondria enabled them to take up virtually all Ca\(^{2+}\) from the incubation medium, lowering the steady state [Ca\(^{2+}\)] to a few micromolar at most (Fig. 1, trace c, and Fig. 7B).

![Diagram](http://www.jbc.org/)

**Fig. 5.** The effect of preincubation of yeast mitochondria with NADH on the initial rate of Ca\(^{2+}\) transport (A) and the steady state medium free Ca\(^{2+}\) (B). Closed circles, control; open triangles, the mitochondrial suspension was preincubated with 4 mm NADH in the incubation medium for 0.5 min and then a small aliquot (50-fold dilution) was used for the registration of Ca\(^{2+}\) transport.

The Stimulation of Ca\(^{2+}\) Uptake in Yeast Mitochondria by the Combined Action of the Various Modulators—The stimulatory effect of ADP was further potentiated by 25 \(\mu\)M spermine (Fig. 1, traces a and b; Fig. 2, A and B). Added together, these two physiological modulators provided high rates of Ca\(^{2+}\) uptake (Fig. 2A) and an improved Ca\(^{2+}\)-buffering capacity (Fig. 2B). Exceptionally high rates of Ca\(^{2+}\) uptake were obtained with a combination of all these modulators, i.e. ADP, spermine, NADH and Ca\(^{2+}\) (Fig. 8A and 9). The yeast mitochondria were then able, when exposed to 50–300 \(\mu\)M Ca\(^{2+}\), to maintain a constant steady state extramitochondrial [Ca\(^{2+}\)] close to the detection limit of the murexide technique (1–3 \(\mu\)M; Fig. 8B), and even that of Arsenazo III (Fig. 9), i.e. \(-0.2 \mu\)M. The level was maintained for 10–15 min, i.e. until the experiment was terminated (data not shown). It is noteworthy that yeast mitochondria supplemented with the physiological effectors preserved the improved kinetic properties of the Ca\(^{2+}\) transport system even in the presence of 0.5 mm Mg\(^{2+}\), 5 mm NaCl, and 100 mm KCl, mimicking the ionic composition of the cytosol (Fig. 8, A and B), the [Ca\(^{2+}\)] at V\(_{1/2}\) being 110, 96 \(\mu\)M, and the Hill coefficients 2.15.

**DISCUSSION**

The main finding in this study is a potent stimulation of mitochondrial Ca\(^{2+}\) transport in *E. magnusii* by ADP, Ca\(^{2+}\) itself, and NADH in addition to the previously known stimulation by polyamines (15, 16).

The Mechanism of Stimulation of Ca\(^{2+}\) Transport by ADP—The protective action of adenine nucleotides on mammalian mitochondria has long been known (26–28). The most likely explanation is that ADP counteracts the swelling induced by Ca\(^{2+}\), i.e. MPT (29–31) and the opening of the CSA-sensitive pore (32). We could not, however, find any evidence in favor of...
the existence of MPT in E. magnusii mitochondria. Therefore, it is unlikely that the effects of ADP and atractyloside are mediated by the modulation of pore opening, i.e. due to inhibition of Ca\(^{2+}\) efflux that is negligible, <20 nmol of Ca\(^{2+}\)/min/mg of protein. Rather, these data indicate that the stimulatory effect of ADP on the rate of Ca\(^{2+}\) accumulation was due to stimulation of the uptake mechanism itself. Rottenberg and Marbach (33, 34) have interpreted the lowering of the steady state [Ca\(^{2+}\)] by ADP in brain mitochondria as a stimulation of the uniporter via a conformation change of the adenine nucleotide translocator since the efflux rate for Ca\(^{2+}\) was low and CSA only slightly affected the steady state. The apparent lack of MPT in these yeast mitochondria would readily explain their ability to retain large amounts of Ca\(^{2+}\) and stay coupled for a comparatively long time.

The Mechanism of Stimulation of Ca\(^{2+}\) Transport by NADH—Yeast mitochondria offer a fertile field for evaluating Ca\(^{2+}\) transport as affected by gradual changes in the redox state of mitochondrial pyridine nucleotides. In animal mitochondria, the picture is complicated by MPT that is stimulated by oxidation of pyridine nucleotides, presumably because of interrelations between the redox states of pyridine nucleotides, glutathione, and pore vicinal SH groups (35). The mechanism by which NADH modulates mitochondrial Ca\(^{2+}\) transport in E. magnusii yeast is unknown; NADH may influence the transport directly or via the redox state of glutathione and SH groups. Oxidation of added NADH followed by reversed electron flow could conceivably effect reduction of matrix NAD\(^{+}\) that could enhance the Ca\(^{2+}\) uptake mechanism.

The Similarities between E. magnusii and Animal Mitochondrial Ca\(^{2+}\) Uptake—The Ca\(^{2+}\) uptake system of E. magnusii yeast mitochondria displays striking similarities with the well characterized animal mitochondrial calcium uniporter. In both systems, the driving force is the transmembrane potential, negative on the matrix side. Both transport systems are stimulated by polyamines and by Ca\(^{2+}\) itself (36–38). It may therefore be justified to call the E. magnusii yeast Ca\(^{2+}\) transporter a mitochondrial calcium uniporter as well. The concerted action of spermine, ADP, NADH, and small concentrations of Ca\(^{2+}\) ions may confer to these yeast mitochondria a similar role as for the animal uniporter (4), i.e. the short term regulation of mitochondrial respiration and oxidative phosphorylation. Yeast mitochondria may even be more important in the overall Ca\(^{2+}\) homeostasis than animal mitochondria. However, there is one striking difference in that the animal mitochondrial calcium uniporter is very sensitive to Ruthenium Red (39), while the E. magnusii one is less sensitive, if at all, and in this respect resembles plant mitochondria (40). Saccharomyces yeasts have a low activity, Ruthenium Red-sensitive cation transporter (41). We have even seen stimulation of Ca\(^{2+}\) up-charging by spermine and ADP in these mitochondria, while it was absent in animal mitochondria. The mechanism by which NADH modulates mitochondrial Ca\(^{2+}\) transport in E. magnusii yeast is unknown; it seems to be due to a modulation of the Ca\(^{2+}\) uptake mechanism by NADH.

Fig. 6. The effect of sequential additions of Ca\(^{2+}\) on Ca\(^{2+}\) transport by yeast mitochondria. In A, the addition of Ca\(^{2+}\) was 50 μM, and in B, 100 μM. The traces at the right correspond to control experiments in which the Ca\(^{2+}\) addition was the sum of Ca\(^{2+}\) taken up after the first addition at left plus the amount added in the second addition of Ca\(^{2+}\). The initial rates of Ca\(^{2+}\) uptake in nanomoles × min\(^{-1}\) × mg\(^{-1}\) protein were in A, after the first addition of Ca\(^{2+}\) 43, after the second 113, and in the control 86, while in B the corresponding rates were 90, 184, and 118, respectively.
take by Ruthenium Red in E. magnusii yeast mitochondria (42). Recently, stimulation of the fast mode of Ca\(^{2+}\) uptake by very low levels of Ruthenium Red in liver mitochondria was reported (3).

It is remarkable that E. magnusii so far is the only yeast species in which an efficient mitochondrial Ca\(^{2+}\) uptake system has been demonstrated. One may speculate that there is a close correlation between the capacity of mitochondria for Ca\(^{2+}\) transport and the structural organization of the respiratory chain, i.e., the functioning of complex I of the respiratory chain as a coupling site in oxidative phosphorylation, and the absence of alternative electron flow pathways bypassing this site (12). These two features contribute to establishing a high matrix NADH/NAD\(^+\) ratio. The sensitivity of the yeast mitochondrial Ca\(^{2+}\) transporter to modulation by physiological effectors makes it a promising model in searching for other potential physiological modulators of Ca\(^{2+}\) transport.

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