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Mechanisms of Passive Potassium Influx in Corn Mitochondria[1]

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ABSTRACT

Corn mitochondria in 100 millimolar KCl show accelerated passive swelling upon addition of uncoupler. This unusual response has been compared with swelling produced by valinomycin, tripropyltin, and nigericin. It is concluded that the driving force for swelling lies with the chloride gradient and a high P_{Cl}:P_K ratio, the chloride influx creating a negative membrane potential. The action of uncoupler is to facilitate K^+ influx via the endogenous H^+/K^+ antiporter. The antiporter is active over the pH range 6 to 8, is not sensitive to Mg^{2+} concentration, and is not inactivated by aging. It is not clear why corn mitochondria show this exceptional activity of the H^+/K^+ antiporter in K^+ influx. It is speculated that during isolation the antiporter may be exposed or activated, and that it contributes to cyclic K^+ transport and high State 4 respiration rates.

On the basis of passive swelling in isotonic salt solutions it appears that mitochondria from several plant sources are permeable to chloride and nitrate (9, 13, 15, 17, 20, 24, 28, 29), anions which are typically absorbed and transported readily by plant tissues. The rate of KCl or KNO_3 influx is commonly limited, however, by the relative impermeability of the inner mitochondrial membrane to K^+, as demonstrated by the marked increase in passive swelling upon addition of the K^+ ionophore, Val.[2] Figure 1A illustrates the now generally accepted explanation for this. A high value for P_{Cl} (permeability constant for Cl^-) compared to P_K, plus a large concentration gradient for Cl^-, establishes a diffusion potential, negative inside, down which K^+ "leaks" or rapidly fluxes when P_K is increased by valinomycin. In the usual experiment using 100 to 150 mM K^+ salt there is not likely to be a large K^+ concentration gradient since freshly isolated plant mitochondria (15, 18) are like liver (2) or heart (6) mitochondria in containing 120 to 170 nmol K^+/mg protein. If the initial matrix volume is estimated at about 1 μl/mg protein (2) the internal K^+ activity will not differ greatly from the external. The Cl^- content of plant mitochondria appears to be unreported; it is about 16 mM in rat liver mitochondria (2).

A potential alternative pathway for K^+ entry is illustrated in Figure 1B. Here the K^+ enters via an electroneutral K^+/H^+ antiport, which is widely accepted as the avenue for K^+ extrusion from plant mitochondria during energy-linked contraction (15–17, 20). If this antiporter is also operative in KCl influx the swelling rate will be governed by the rate of H^+ influx or OH^- efflux to relieve the pH gradient created and to charge compensate the electrogenic Cl^- influx. If so, the addition of a H^+-shuttling uncoupler, such as DNP or FCCP, or the Cl^-/OH^- exchanging ionophore TPT (26), should greatly accelerate the rate of salt entry and swelling. However, recent reports (17, 20, 24) indicate little if any KCl influx by this mechanism in plant mitochondria unless nigericin, an antibiotic carrying out neutral H^+/K^+ exchange, is introduced. The endogenous H^+/K^+ exchanger is believed to operate only in salt extrusion (20).

An exception to this generalization may be found in corn mitochondria. Early experiments on passive swelling in KCl showed uncouplers to accelerate swelling (12, 27). Analyses for KCl showed that swelling has an osmotic origin (22). Thus, it is possible that in corn mitochondria at least part of the KCl influx might be mediated through the H^+/K^+ antiport allowing K^+ entry.

We have utilized standard swelling techniques to examine this question. We conclude that in corn mitochondria the H^+/K^+ antiport is present and can act efficiently in KCl uptake. It is stable under varied conditions of pH, Mg^{2+} and aging. However, we suggest that it normally acts in K^+ extrusion, stabilizing mitochondrial salt loads, and in this capacity may contribute significantly to State 4 respiration rates.

MATERIALS AND METHODS

Mitochondria were isolated by the "cushion" technique from 3 day etiolated corn shoots (9). The isolation medium contained 0.4 M sucrose, 50 mM Tes, 1 mg/ml BSA, 5 mM ethylene glycol-*bis*(β-aminoethyl ether-*N*,*N*'-tetracetic acid, adjusted pH 7.6 with KOH. Oxidative phosphorylation was determined as described (9).

Swelling was determined by light transmission at 520 nm in a standard medium of 100 mM KCl, 4 mM $MgSO_4$, 25 μg/ml antimycin A, 1 μg/ml oligomycin, 1 mg/ml BSA, and 10 mM Tes buffer adjusted to the desired pH with Tris. Standard experimental additions to the medium were 10 μM FCCP, 1 μM TPT, 0.13 μg/ml Nig, 0.75 μg/ml Val. Variations in media or additives are given with the data. Swelling was started by addition of 0.1 ml of mitochondria suspension in 0.25 M sucrose (0.8–1.2 mg protein) to 3.8 ml of medium.

The endogenous Cl^- concentration of the mitochondria was too low for determination by methods at our disposal. Dr. Philip K.

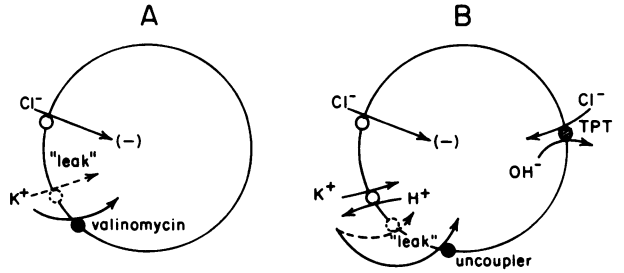

FIG. 1. Diagramatic illustration of avenues for K^+ influx during spontaneous, passive swelling of mitochondria in KCl solutions. (○), endogenous avenues of transport; (●), avenues provided by ionophores or uncouplers. "Leaks" represent endogenous avenues which may be introduced or exposed during isolation leading to spontaneous swelling. A, K^+ uniport; B, K^+/H^+ antiport.

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[2] Abbreviations: Val, valinomycin; FCCP, ρ-trifluormethoxy(carbonylcyanide)-phenylhydrazone; TPT, tripropyltin; Nig, nigericin.

Hopke, Environmental Research Laboratory, University of Illinois, made the determinations for us by neutron activation analysis. For this purpose we centrifuged down the final suspension of mitochondria (12.7 and 12.6 mg protein in two preparations) and analyses were made for K^+ and Cl^- on the total sample.

RESULTS AND DISCUSSION

Mitochondrial Integrity. Bonner (5) has suggested that spontaneous swelling in salt solutions may arise from mitochondria which are not intact, presumably having suffered membrane damage during isolation. By the usual criteria of respiratory control ratios and ADP:O ratios, the corn mitochondria preparations used here were tightly coupled and not generally inferior to other plant preparations (Fig. 2; see also ref. 9). This does not mean that the isolated mitochondria are as tightly coupled as *in vivo*, but it does suggest that corn mitochondria (and other plant mitochondria) compared to most animal mitochondria come through isolation procedures more permeable to H^+ or ions.

In chemiosmotic theory, high "substrate" (5) or State 4 rates signify that avenues exist for reentry of H^+ extruded by the respiratory chain. One direct avenue might be a simple H^+ "leak." Proton permeability is higher in plant than animal mitochondria (10). Moore *et al.* (25) have found for mung bean mitochondria that the proton motive force rises in State 4; it is conceivable that a higher potential might open a "voltage gate" for such a leak. Ducet (10) has shown that the proton conductance of potato mitochondria is greatly reduced in the presence of BSA. In all the experiments reported here, BSA was included in the isolation and reaction media.

Another source of H^+ leak might be through the coupling ATPase. When corn mitochondria are loaded with Pi, there is a component of the "acceptorless" or "substrate" respiration which can be blocked with oligomycin (9, 14), indicating a turnover of matrix Pi with the coupling ATPase which admits H^+. In addition, using oligomycin to block the H^+ leak through the ATPase increases the rate and extent of energy-linked ion uptake (1, 23). The use of chelating agents during isolation may remove Mg^{2+} essential to H^+ impermeability; in corn mitochondria Mg^{2+} will prevent the swelling associated with H_2SO_4 acidification (21). In the swelling experiments which follow we have routinely added oligomycin and Mg^{2+}.

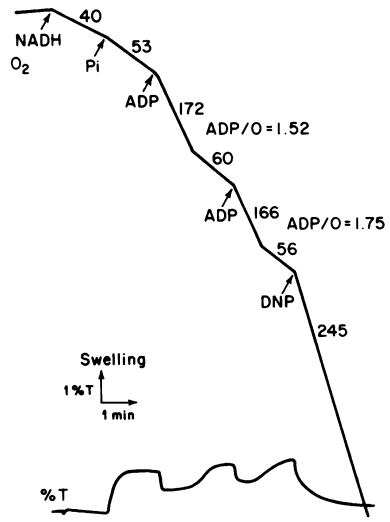

FIG. 2. Respiration and swelling traces for a typical preparation of corn mitochondria. Medium was 0.2 M sucrose, 20 mM Tes, 20 mM KCl, 4 mM $MgSO_4$, 1 mg/ml BSA (pH 7.5) with KOH, 0.1 ml mitochondria (0.68 mg protein). Additions were 1.25 mM NADH, 5 mM KH_2PO_4, 300 nmol ADP, 100 μM DNP.

In addition to H^+ leaks, high State 4 rates might result from recycling of H^+ in cyclic ion transport. Our laboratory has proposed cycling of K-phosphate under steady-state loading. As shown in Figure 2, corn mitochondria reswell in State 4. Swelling is due to uptake of K-phosphate (14), and steady-state swelling is maintained with no change in respiration rate unless ion transport is perturbed (14, 16). The cycling of salts in steady-state is postulated to include K^+ entry via uniport and exit via H^+/K^+ antiport (16). Corn mitochondria in Tes-Tris buffered sucrose have low NADH oxidation rates unless KCl is added (22), which also indicates that K^+ cycling at the expense of the proton motive force might be a component in the "loose coupled" respiration of corn mitochondria.

The available evidence suggests that high State 4 and swelling rates are much more likely to be due to the high activity of ion transport systems than of injured and leaky membranes. However, the possibility remains that isolation techniques expose or activate these transport systems. For example, Jung and Brierley (20) find high pH, which induces swelling, to activate the H^+/K^+ antiport for efflux in potato mitochondria, and Garlid (11) reports swelling to unmask the H^+/K^+ antiport activity in rat liver mitochondria. There are also differences in plant tissue to be accounted for; Jung and Brierley (20) found that mitochondria from fresh potatoes do not swell passively in KCl, while those from potatoes stored at 4 C for a few weeks do.

In this report on KCl swelling in corn mitochondria we consider that the mitochondria meet normal standards for membrane integrity, and that the spontaneous, passive swelling in KCl reflects the operation of normal avenues for ion transport.

K^+ and Cl^- Content. Table I gives the K^+ and Cl^- content of freshly isolated corn shoot mitochondria as determined by neutron activation analysis. The K^+ content is slightly lower than previously determined for corn and cauliflower mitochondria (about 140 nmol/mg protein [18]) possibly due to reisolation from the suspending medium, which effectively produces an additional washing. Matrix volume and matrix K^+ were not determined, but previous work where this was done showed that corn mitochondria suspended for 1 min in 120 mM sucrose plus 40 mM K-acetate had a matrix concentration of 130 mM K^+ (22). Collectively, the data indicate that initially there is very little difference in K^+ activity across the inner membrane when mitochondria are suspended in 100 mM KCl. In comparison, Cl^- concentrations differ by an order of magnitude, and might produce electrical potentials of −40 to −50 mv if the $P_{Cl}:P_K$ ratio is large. Thus, the analytical data support the supposition that the driving force for KCl swelling lies with the Cl^- gradient (and a membrane permeable to Cl^-).

Ionophore and Uncoupler Activated Swelling. Figures 3A and B are from a summary experiment which illustrates the principle findings of an extensive investigation of agents which accelerate swelling in KCl. Endogenous respiration was blocked with antimycin A (although this had no discernible effect on swelling) and possible H^+ leaks through the coupling ATPase were blocked with oligomycin. It was observed early that both rat liver (3) and corn (25) mitochondria swell more rapidly at high pH, and comparisons were made at pH 6.0 and 7.5.

In agreement with previous investigations of plant mitochondria, Val produced very rapid KCl swelling. This is swelling by the mechanism illustrated in Figure 1A, and demonstrates that K^+ permeation is the limiting factor in spontaneous swelling. Raising the pH increases both spontaneous and Val-activated swelling. In

Table I. *K^+ and Cl^- Content of Isolated Corn Mitochondria*

	K^+	Cl^-
	nmol/mg protein	
Prep. 1	122 ± 4	14 ± 0.6
Prep. 2	130 ± 4	11 ± 1.0

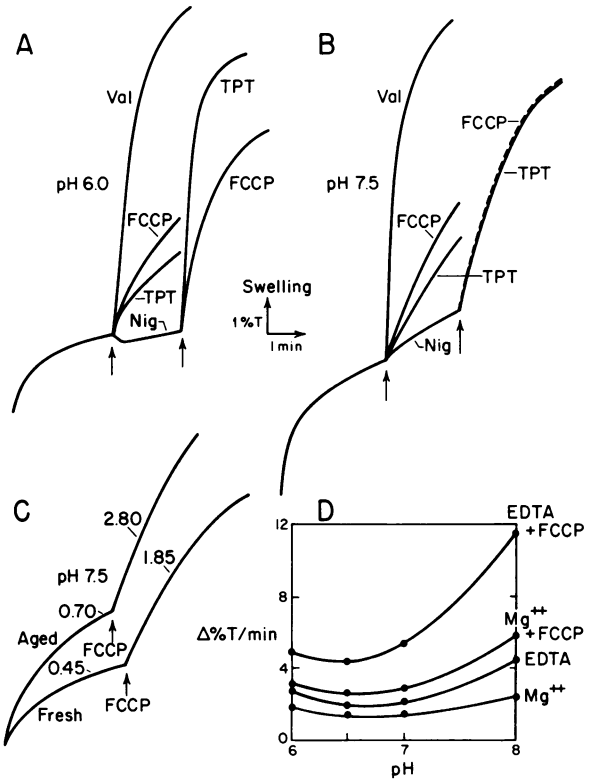

FIG. 3. Swelling of corn mitochondria in KCl. See under "Methods and Materials" for description of procedure, media, and additives. Additions made as indicated at arrows. A, swelling at pH 6.0; B, swelling at pH 7.5; C, effect of aging mitochondria on ice for 90 min (figures are Δ% T for 1 min before and after adding 10 μM FCCP); D, swelling rates as a function of pH in standard medium (4 mM Mg^{2+}), and medium lacking Mg^{2+} but containing 0.1 mM EDTA; FCCP = 10 μM.

liver mitochondria, Azzi and Azzone (3) attributed the effects of high pH to increased Cl^- permeability since Cl^- exchange was promoted at high pH and K^+(^{86}Rb) exchange was not. Assuming this applies to corn mitochondria, increasing the pH from 6.0 to 7.5 would increase P_{Cl}, and thus the electrical potential for driving K^+ influx. Brierley *et al.* (7) attribute rapid swelling of heart mitochondria with increasing pH to increased K^+ or Na^+ permeability on the basis that ionophores are more effective in producing swelling at low pH. In corn mitochondria, Val is effective at low and high pH (Fig. 3, A and B).

However, the action of valinomycin does not establish that spontaneous swelling (no Val) is due to an endogenous K^+ "leak." The addition of FCCP (or 2,4-dinitrophenol) or TPT also produced accelerated swelling, showing that the H^+/K^+ antiport mechanism of Figure 1B is present and functional. Spontaneous swelling thus might be ascribed to a H^+ leak as well as a K^+ leak.

One way of estimating the relative efficiency of the H^+/K^+ antiport in spontaneous swelling is to saturate this pathway by introducing Nig, the H^+/K^+ exchanging ionophore (Fig. 3, A and B). Nig had no effect on spontaneous swelling, but its presence increased the rate of swelling when FCCP was introduced to facilitate H^+ influx. Nonetheless, it is remarkable that the rate produced by FCCP without Nig was quite large, and demonstrates that the endogenous H^+/K^+ antiport possesses sufficient activity to be an effective avenue in K^+ transport.

With allowance for the increased spontaneous swelling as pH increases, FCCP has much the same effect at pH 6.0 and 7.5, even with Nig present. This was not true for TPT, which in the presence of Nig was more effective at pH 6.0. This result might be attributed to rising P_{Cl} with rising pH (3). At low pH, where P_{Cl} is lower, the introduction of the TPT-Cl^-/OH^- antiport can add significantly to the avenues for Cl^- influx, and when coupled with the Nig H^+/K^+ antiport produces a large KCl influx. At high pH the limiting factor is not Cl^- influx but H^+ influx (or OH^- efflux) (Fig. 1B), and TPT has no greater effect than FCCP (*cf.* Fig. 3, A and B).

A series of experiments with a preparation of mitochondria might take 1 h or more, during which time they were held on ice. It was consistently found that spontaneous swelling rates were higher in aged mitochondria (Fig. 3C); as previously reported, this is also true for State 4 respiration (14). This aging effect did not alter the proportionate response to FCCP (Fig. 3), and thus the relative activity of the endogenous H^+/K^+ antiport remained constant during aging. (A corollary of this is that aging cannot greatly increase any H^+ leak; if the mitochondria were endogenously uncoupled there should be less response to FCCP.)

In these experiments we routinely added Mg^{2+} which is helpful in maintaining the integrity of corn mitochondria in K-phosphate or sulfate transport (1). Mg^{2+} slightly retarded the KCl swelling rate (data not shown). A comparison was made of adding Mg^{2+} or EDTA over the pH range 6 to 8. As previously reported (27), corn mitochondria show minimum swelling at about pH 6.5 (Fig. 1D). Removal of endogenous divalent cations with EDTA had proportionately greater effect at high pH, and the response to FCCP was somewhat greater at high pH. The effect of removing divalent cations and high pH is probably best ascribed to increased chemical potential of K^+ at the membrane surface (4). For the purpose of this investigation, the important point is that the endogenous H^+/K^+ antiport was active over the entire pH range and was not critically dependent on the presence of divalent cations.

In animal mitochondria the cation$^+/H^+$ antiport is much more active with Na^+ than K^+, judging by rates of energy-linked salt efflux (6, 7). With spontaneous and uncoupled swelling of corn mitochondria, activity was greater with KCl than NaCl, although it is clear that the antiporter can accommodate both ions (Fig. 4, A and B). Yoshida and Sato (29) report spontaneous swelling of castor bean mitochondria to increase in the order $NH_4Cl < LiCl < NaCl < KCl$.

Figure 5 shows the effect of substituting slowly penetrating choline$^+$ for K^+. There is very little swelling response to uncoupler until K^+ is added. The slight swelling produced by FCCP might be due to limited uptake of HCl (*i.e.*, simple H^+ influx accompanying Cl^-), but there is insufficient potential in the Cl^- gradient to permit more than a small acidification of the matrix. Addition of K^+ permits recycling of H^+ via H^+/K^+ exchange and thus net salt influx.

Yoshida and Sato also report (29) that spontaneous swelling in K-halides increases in the order $F^- < Cl^- < Br^- < I^-$. This is the

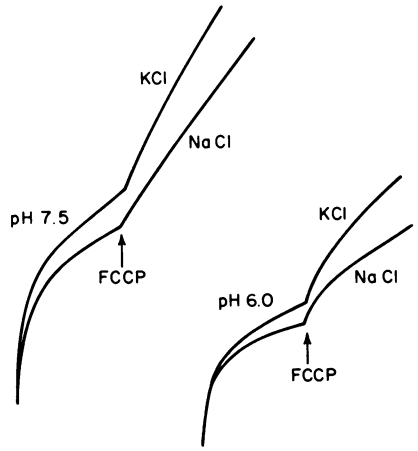

FIG. 4. Swelling in 100 mM KCl medium, and in medium substituting 100 mM NaCl.

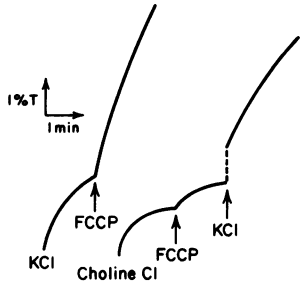

FIG. 5. Comparison of uncoupler-induced swelling in KCl and choline chloride. Standard medium at pH 7.5 with the substitution of 100 mM choline chloride for KCl as indicated. Additions were 10 μM FCCP and 0.5 ml of 1 M KCl (the instantaneous increase in transmission upon addition of KCl is due to dilution of the mitochondrial suspension).

Table II. *Swelling Rates of Corn Mitochondria in K Halides as Affected by Uncouplers and Ionophores*

Determinations of swelling rates made in 100 mM KCl, KBr, or KI at pH 6.0 as in Figure 3A. Rates were determined by change in per cent T for 1 min preceding and 1 min following the addition of FCCP, TPT, or Val.

	Initial	+ FCCP	+ TPT	+ Val
		Δ% T/min		
KCl	0.2	2.6	1.7	5.1
KBr	0.6	3.3	2.1	6.6
KI	1.2	3.0	2.7	6.7

order of increasing atomic radius, decreasing ionization potential, and thus decreasing potential for ion hydration. Data for spontaneous swelling in Cl^-, Br^-, and I^- are given in Table II. Results with F^- were erratic and are not reported. The increase in spontaneous swelling with Br^- and I^- compared to Cl^- can be ascribed to increased concentration gradients and permeability constants which will increase the membrane potential for driving electrophoretic K^+ influx. In parallel, there is proportionately less response to FCCP and TPT. That is, as the membrane potential rises there is greater tendency for K^+ to enter via uniport (Fig. 1A) than by H^+/K^+ antiport (Fig. 1B). Also, under the greater potential the apparent resistance to K^+ entry via uniport appears to be lower (less proportionate response to Val). Experiments with KSCN, which penetrates very readily (29), gave swelling rates 10-fold more rapid than KCl, with no response to addition of uncoupler (data not shown).

Swelling in K-Acetate. Another means of detecting K^+ influx via the H^+/K^+ antiport might be by swelling in K-acetate. Acetate is believed to penetrate the membrane as the undissociated acid in energy-linked or NH_4^+-gradient driven uptake (15). If acetic acid also is the penetrating molecule in spontaneous swelling an H^+/K^+ exchange would be required to give net K-acetate uptake. In such a system Val should not give accelerated swelling since acetic acid influx would produce ΔpH across the membrane rather than $\Delta\psi$. However, in both turnip (24) and mung bean mitochondria (17) Val somewhat increases K-acetate swelling, suggesting an electrogenic penetration of acetate, similar to Cl^-.

As with KCl, spontaneous swelling in K-acetate increases with pH (Fig. 6), and since there is a pronounced response to valinomycin, especially at pH 7.5, an electrogenic influx of acetate is indicated. Spontaneous swelling is more rapid than in KCl, and there is much less response to uncoupler. Since acetic acid also functions in H^+ transport, the K-acetate medium is already furnished with a weak uncoupler, accelerating swelling rates and diminishing response to further addition of uncoupler. Nig produces a sharp increase in swelling, particularly at pH 6.0 where the acetic acid concentration is higher. The large difference in

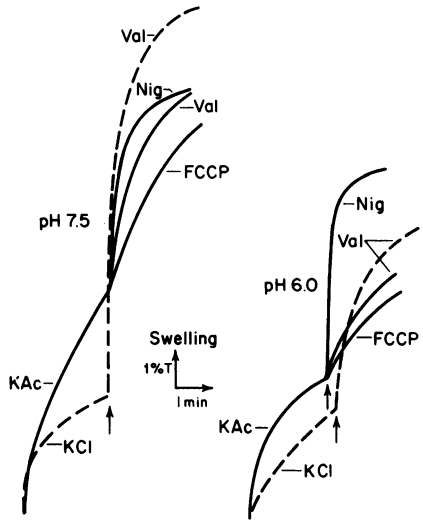

FIG. 6. Swelling in 100 mM K-acetate medium. Additions as in Figure 3. Swelling of the same mitochondrial preparation in 100 mM KCl is shown for comparison (- - -).

swelling rates produced by Nig and Val at pH 6.0 indicates that electrogenic penetration of acetate makes less proportionate contribution to swelling than at pH 7.5.

It appears that at the lower end of the physiological pH range, spontaneous swelling is partially mediated by H^+-acetate$^-$ symport plus H^+/K^+ antiport. With diminishing H^+ there is increasing permeability to acetate$^-$, and a greater share of the salt influx involves electrophoretic K^+ influx. The restriction on spontaneous swelling at pH 6.0 may lie with partial inactivation of the H^+/K^+ antiport at low pH, and only upon addition of Nig is the full potential for acetic acid penetration realized.

Conclusions. These experiments confirm previous observations (12, 27) that corn mitochondria show accelerated swelling in KCl upon addition of uncouplers. Within the framework of generally accepted chemiosmotic theory and the action of ionophores and uncouplers (Fig. 1) this result is due to the presence of an endogenous H^+/K^+ antiport active in salt influx. It does not have to be activated by swelling or respiration-linked pH gradients (17, 20). Judging by the response to FCCP, the antiport appears to function over the physiological pH range and is relatively indifferent to the presence of Mg^{2+} (Fig. 3D). Its activity is not noticeably altered by the changes in KCl permeability that accompany aging (Fig. 3C). The antiport will function in Na^+ influx, although not so effectively as with K^+ (Fig. 4). At lower pH values the antiport may function in K-acetate swelling (Fig. 6). To the best of our knowledge no other species of mitochondria show spontaneous activity of the H^+/K^+ antiport in passive salt influx. A suggestion is made that turnip mitochondria transport Na^+ or K^+ inwards by this antiport, but they do not show accelerated swelling with uncoupler (24).

What is not revealed in these experiments, and cannot be by present methodology, is what proportions of KCl influx in spontaneous swelling (no added ionophores or uncouplers) are due to K^+ "leak" (Fig. 1A) and H^+ "leak" (Fig. 1B). As indicated, these "leaks" govern the rate of swelling and are responsible for high State 4 respiration. The fact that the presence of an active H^+/K^+ antiport is indicated by addition of uncoupler does not establish that the antiport is functioning in spontaneous swelling. The ADP:O ratios do not indicate a significant H^+ permeability. Acidification of the medium produces stable pH values despite large swelling or contraction (21). Extrapolating from the increase in swelling rates with increased pH and increased halide permeability, in which K^+ influx appears to be increasingly by uniport, we suspect that spontaneous swelling is largely by means of electro-

phoretic K^+ influx (see also ref. 20). This is the generally accepted means of K^+ entry during energy linked salt uptake by plant (15) and animal (6) mitochondria, and the primary distinction in passive swelling is that the electrical gradient produced by anion permeability is much smaller.

It is likely that the normal *in vivo* function of the H^+/K^+ antiport is in stabilizing the salt content of the matrix, as suggested for K-phosphate (16). Jung and Brierley (20) have made a similar suggestion. Huber and Moreland (17) report that the antiport is activated for efflux by respiration. Ready penetration of K^+ by uniport and rapid efflux by H^+/K^+ antiport can account for high State 4 respiration. Cycling of K^+ in State 4 has been shown for heart mitochondria (8, 19).

We do not know why corn mitochondria readily demonstrate H^+/K^+ antiport activity in KCl influx, while others do not. There is no theoretical reason why an antiport should not operate in either direction given suitable potential gradients. If antiport activity is normally expressed only in K^+ efflux, despite favorable gradients and permeabilities for influx, there must be biological controls. Perhaps part of this control is lost during isolation of corn mitochondria, or by exposure to high concentrations of salt. As previously reported, however, some control exists during efflux pumping, suggestive of a lipid barrier (16).

Lastly, although the above conclusions are consistent with swelling experiments and salt transport theory as illustrated in Figure 1, some caution should be expressed. We have not determined net K^+ fluxes, and there is no direct demonstration that the increased swelling with uncoupler, TPT, or Val results from increased influx of K salts. Neither is it shown that an H^+/K^+ antiport exists as a specific exchange transporter characteristic of the inner membrane. Under some circumstances, an "antiport" might consist of a uniport admitting K^+ down a potential created by uncoupler-mediated efflux of H^+. Additional and more sophisticated experimentation is needed to establish the nature and activity of the H^+/K^+ antiport.

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