Characteristics of the Movement of \( K^+ \) across the Mitochondrial Membrane and the Inhibitory Action of \( Tl^+ \)

(Received for publication, December 23, 1974)

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SUMMARY

The incubation of mitochondria in mixtures that contain phosphate, \( NaCl \), oxidizable substrate, and ethylenediaminetetraacetate induces the efflux of \( K^+ \). This process depends on electron transport and on the cyclic movement of phosphate across the membrane. Sodium ions, \( Li^+ \), or \( Ca^2+ \) to a smaller extent, are required for maximal release of \( K^+ \). Potassium ions do not induce net efflux of internal \( K^+ \), but instead prevent the \( Na^+ \)-induced release of \( K^+ \). Significant \( K^+ \) influx takes place in \( K^+ \)-depleted mitochondria through a process with characteristics which are almost identical with those in which \( K^+ \) release takes place. As \( Na^+ \) inhibits the uptake of \( K^+ \), it is suggested that the movement of \( K^+ \) across the membrane is controlled by the cationic environment. Thallous ion, at concentrations that do not affect oxidative phosphorylation, was found to be an effective inhibitor of the influx and the efflux of \( K^+ \). The inhibitory effect of \( Tl^+ \) seems to be specific for \( K^+ \) since it does not affect the movement of \( Na^+ \). Mitochondria bind 10 to 15 nmmol of \( ^{204}Tl^+ \) per mg of protein through an energy-independent process.

Although ionophore-mediated translocation of monovalent cations across the mitochondrial membrane has been extensively studied (for review see Refs. 1 and 2), little work has been done on the intrinsic capacity of the mitochondria to move \( K^+ \) across its inner membrane. Perhaps this is due to the finding that mitochondria maintain their \( K^+ \) against a concentration gradient (3), which suggested that the membrane was impermeable to \( K^+ \). Moreover, mitochondria retain \( K^+ \) in conditions in which extensive loss of \( Na^+ \) takes place (4). However, in relatively long periods of incubation at 38°, mitochondria released a significant amount of \( K^+ \) that could be taken up again provided an energy source was introduced (5, 6); also Gamble (7) found that digitonin particles possess an energy-dependent \( K^+ \) transport. It has also been observed that EDTA increases the permeability of the mitochondria to monovalent cations (8–10), and more recently, Gamble (11) found that citrate could mediate the uptake of \( K^+ \). Therefore, these findings suggest that \( K^+ \) movements in mitochondria are subject to some type of control.

This is of importance since monovalent cations (12–16) and their movement (17–20) across the membrane affect oxidative phosphorylation and related reactions. In this work we describe some characteristics of the energy- and cation-dependent translocation of \( K^+ \) in mitochondria. Also the inhibitory effect of thallous ion on the movement of \( K^+ \) is described. A possible inhibitory effect of \( Tl^+ \) on \( K^+ \) translocation was studied because its atomic radius is similar to that of \( K^+ \) (1.40 and 1.33 A, respectively), but it binds more strongly to proteins (21, 22).

MATERIALS AND METHODS

Rat liver mitochondria were prepared as described elsewhere (14) in 0.25 \( \text{M sucrose plus 1 mM EDTA adjusted to pH 7.3 with Tris base} \). In some cases mitochondria were prepared in the absence of EDTA. The release of \( K^+ \) was measured as described before (15). Our isolated intact mitochondria had 100 to 130 nmmol of \( K^+ \) per mg of protein. Experiments with preparations with less than 100 nmmol were discarded. In some experiments inorganic phosphate was determined in the 6\% trichloroacetic extract of a mitochondrial suspension according to Sumner (23).

Uptake of \( K^+ \) was measured by incubating mitochondria depleted of \( K^+ \) for 3 min in mixtures of variable composition (see under "Results"); subsequently the mixture was diluted with sucrose with or without EDTA and centrifuged at 10,000 \( \times g \) for 5 min. The mitochondria were washed twice with sucrose or sucrose plus EDTA. The \( K^+ \) content of these mitochondria was measured by flame photometry as described elsewhere (15).

Oxygen uptake was measured polarographically (Yellow Springs Instrument Co.), and ADP to oxygen ratios were measured according to Estabrook (24).

The binding of \( ^{204}Tl^+ \) (Amersham/Searle) to mitochondria was determined by incubating the particles in mixtures that contained various concentrations of \( ^{204}Tl^+ \) and thereafter filtered through Millipore. The filter was washed with 2.0 ml of 0.28 \( \text{M sucrose} \) and transferred to a scintillation vial that contained 10 ml of Bray's solution. The samples were counted with a Packard scintillator with a \( ^{4}C \) window. As the Millipore filters bound \( ^{204}Tl^+ \), a correction was made by subtracting the radioactivity found in filters through which aliquots of the reaction mixture (without mitochondria) had been passed and washed with 2.0 ml of sucrose. Protein was determined according to Lowry et al. (25).

RESULTS

Characteristics of Movement of \( K^+ \) across Membrane—Although the uptake and release of \( K^+ \) by mitochondria were measured independently and under different conditions the results are presented simultaneously to facilitate a comparison of the characteristics of the two processes. Maximum efflux and influx of \( K^+ \) require phosphate and EDTA (Table 1). Outward and inward translocation of \( K^+ \) also require electron transport, as evidenced by the sensitivity of the two processes to rotenone (26).
The effect of various cations and changes in the concentration of Na\(^+\) is required for maximal release of K\(^+\) (Table I). Therefore, the mitochondria were washed twice with sucrose. In Experiment 2, the conditions were as in Experiment 1, except that the mitochondria were pre-incubated in sucrose-EDTA. In the uptake experiments K\(^+\)-depleted mitochondria were incubated for 3 min under vigorous shaking in 10 mM of a mixture that contained 2.5 mM glutamate, 2.5 mM phosphate, 1 mM EDTA, 100 mM sucrose, and 50 mM KCl. Where indicated the mixtures contained 6.3 \(\mu\)M rotenone. The indicated components were omitted from the complete mixture.

| Experiment | Reaction media | Release of K\(^+\) | K\(^+\) uptake |
|------------|----------------|-------------------|---------------|
| 1          | Complete       | 113               | 46            |
|            | Glutamate      | 20                |
|            | NaCl           | 74                |
|            | Phosphate      | 79                | 21            |
|            | EDTA           | 65                | 14            |
| 2          | Complete       | 107               | 25            |
|            | Complete + rotenone | 63       | 0             |

The effect of various chelating agents on K\(^+\) movements is shown in Table II. EDTA was withdrawn or substituted by 1 mM ethylene glycol bis(\(\beta\)-aminoethyl ether) N\(_2\), N\(_4\)-tetraacetic acid (EGTA), \(\alpha\)-phenanthroline, or citrate. In Experiment 2, the mixtures with 1 mM EDTA were supplemented with the indicated concentrations of Mg\(_2^+\).

| Experiment | Additions | K\(^+\) released | K\(^+\) uptake |
|------------|-----------|------------------|---------------|
| 1          | EDTA      | 54               | 20            |
|            | EGTA      | 87               | 54            |
|            | \(\alpha\)-Phenanthroline | 40 | 25            |
|            | Citrate   | 38               | 18            |
|            | 0.5 mM Mg\(_2^+\) | 40       | 30            |
|            | 1.0 mM Mg\(_2^+\) | 85       | 33            |
| 2          | 0.6 mM Mg\(_2^+\) | 37       | 14            |
|            | 1.2 mM Mg\(_2^+\) | 22       | 16            |
|            | 3.0 mM Mg\(_2^+\) | 9        |               |

The effect of Li\(^+\), K\(^+\), and Cs\(^+\) to that of Na\(^+\). Cs\(^+\) induced a release of K\(^+\) that amounted to about 30% of that induced by Li\(^+\) or Na\(^+\). External K\(^+\) did not support K\(^+\) loss, even though its atomic radius lies between that of Na\(^+\) and Cs\(^+\). Moreover, external K\(^+\) effectively prevented the Na\(^+\) induced release of K\(^+\) (Table III, Experiment 3).

These findings suggested that external cations control the fluxes of K\(^+\). Accordingly, the effect of Na\(^+\) on K\(^+\) influx was studied. Na\(^+\) effectively prevented K\(^+\) influx (Table IV).

An attempt to study the role of phosphate on K\(^+\) movements was made by studying the effect of mersalyl on K\(^+\) efflux. The concentration of mersalyl used was that known to block the phosphate carrier (27, 28). The change in the concentration of phosphate in mitochondria was measured simultaneously with the amount of K\(^+\) lost (Table V). Mersalyl inhibited the efflux of K\(^+\) by more than 50% which indicates that the movement of phosphate across the membrane is required for maximal release. Moreover, it may also be concluded that phosphate undergoes a cyclic movement across the membrane during K\(^+\) release since intramitochondrial phosphate does not change significantly.

It should be pointed out that we have observed significant variability in the uptake of K\(^+\). In 36 experiments that have been carried out in strictly identical conditions with 50 mM K\(^+\) in the uptake mixture, it has been observed that two preparations of

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**Table I**

**Requirements for release and uptake of K\(^+\) in mitochondria**

In the release experiments mitochondria (approximately 90 mg of protein) prepared in 0.25 M sucrose at pH 7.3 were incubated in 12.0 ml of a mixture that contained 75 mM NaCl, 10 mM \(\text{H}_3\text{PO}_4\) (adjusted to pH 7.3 with Tris base), 10 mM glutamate, and 100 mM sucrose (complete reaction media) for 3 min at room temperature with vigorous shaking. The suspension was centrifuged and the pellet washed twice with sucrose. In Experiment 2, the conditions were as in Experiment 1, except that the mitochondria were prepared in sucrose-EDTA. In the uptake experiments K\(^-\) depleted mitochondria were incubated for 3 min under vigorous shaking in 10 mM of a mixture that contained 2.5 mM glutamate, 2.5 mM phosphate, 1 mM EDTA, 100 mM sucrose, and 50 mM KCl. Where indicated the mixtures contained 6.3 \(\mu\)M rotenone. The indicated components were omitted from the complete mixture.

**Table II**

**Effect of various chelating agents and Mg\(_2^+\) on release and uptake of K\(^+\)**

The experimental conditions were as in Table I, except those in Experiment 1. From the complete incubation media 1 mM EDTA was withdrawn or substituted by 1 mM ethylene glycol bis(\(\beta\)-aminoethyl ether) N\(_2\), N\(_4\)-tetraacetic acid (EGTA), \(\alpha\)-phenanthroline, or citrate. In Experiment 2, the mixtures with 1 mM EDTA were supplemented with the indicated concentrations of Mg\(_2^+\).

**Table III**

**Effect of Na\(^+\) and other cations on release of K\(^+\)**

The experimental conditions were as in Table I except that the complete mixture (see Table I) contained the indicated concentrations of the various salts.

| Experiment | Salt added | K\(^+\) released |
|------------|------------|------------------|
|            | K\(^+\) released |
| 1          | 10 mM Na\(^+\) | 62               |
|            | 20 mM Na\(^+\) | 67               |
|            | 40 mM Na\(^+\) | 71               |
|            | 75 mM Na\(^+\) | 72               |
|            | 100 mM Na\(^+\) | 71              |
| 2          | 75 mM Li\(^+\) | 113              |
|            | 75 mM Na\(^+\) | 118              |
|            | 75 mM K\(^+\) | 58               |
|            | 75 mM Cs\(^+\) | 88               |
|            | 75 mM Na\(^+\) + 10 mM K\(^+\) | 102 |
|            | 75 mM Na\(^+\) + 30 mM K\(^+\) | 87 |
|            | 75 mM Na\(^+\) + 60 mM K\(^+\) | 57 |
|            | 75 mM Na\(^+\) + 90 mM K\(^+\) | 36 |

**Table IV**

**Effect of NaCl on uptake of K\(^+\) by K\(^+\)-depleted mitochondria**

The incubating conditions were as in Table V except that the indicated concentrations of NaCl were added to the complete mixture with 50 mM KCl.

| NaCl | K\(^+\) uptake |
|------|----------------|
|      | nmol K\(^+\)/mg |
| 1    | 67             |
| 10   | 53             |
| 30   | 48             |
| 70   | 30             |
| 100  | 25             |

The effect of various chelating agents on K\(^+\) movements is shown in Table II, Experiment 1. EDTA was the only chelating agent that supported K\(^+\) influx and efflux; ethylene glycol bis(\(\beta\)-aminoethyl ether) N\(_2\), N\(_4\)-tetraacetic acid, \(\alpha\)-phenanthroline, and citrate did not significantly effect K\(^+\) movements. Mg\(_2^+\) prevented K\(^+\) movements at concentrations lower than EDTA (Table II).

An important difference between K\(^+\) influx and efflux is that Na\(^+\) is required for maximal release of K\(^+\) (Table I). Therefore, the effect of various cations and changes in the concentration of Na\(^+\) was studied on K\(^+\) efflux. Sodium ions induced maximal K\(^+\) efflux at 40 mM. Table III, Experiment 2 compares the effect of Li\(^+\), K\(^+\), and Cs\(^+\) to that of Na\(^+\). Cs\(^+\) induced a release of K\(^+\) that amounted to about 30% of that induced by Li\(^+\) or Na\(^+\). External K\(^+\) did not support K\(^+\) loss, even though its atomic radius lies between that of Na\(^+\) and Cs\(^+\). Moreover, external K\(^+\) effectively prevented the Na\(^+\) induced release of K\(^+\) (Table III, Experiment 3).

These findings suggested that external cations control the fluxes of K\(^+\). Accordingly, the effect of Na\(^+\) on K\(^+\) influx was studied. Na\(^+\) effectively prevented K\(^+\) influx (Table IV).

An attempt to study the role of phosphate on K\(^+\) movements was made by studying the effect of mersalyl on K\(^+\) efflux. The concentration of mersalyl used was that known to block the phosphate carrier (27, 28). The change in the concentration of phosphate in mitochondria was measured simultaneously with the amount of K\(^+\) lost (Table V). Mersalyl inhibited the efflux of K\(^+\) by more than 50% which indicates that the movement of phosphate across the membrane is required for maximal release. Moreover, it may also be concluded that phosphate undergoes a cyclic movement across the membrane during K\(^+\) release since intramitochondrial phosphate does not change significantly.

It should be pointed out that we have observed significant variability in the uptake of K\(^+\). In 36 experiments that have been carried out in strictly identical conditions with 50 mM K\(^+\) in the uptake mixture, it has been observed that two preparations of...
K⁺-depleted mitochondria did not show K⁺ uptake. In nine preparations more than 60 nmol of K⁺ per mg of protein were taken up by the mitochondria, while in the rest of the experiments the uptake was between 25 and 50 nmol of K⁺. It is not possible at this time to explain this variability.

Tl⁺ Inhibits Movement of K⁺ across a Membrane—The preceding experiments show that K⁺ can move across the mitochondrial membrane, and apparently the inward and outward movements of K⁺ seem to possess similar characteristics. The following experiments describe the inhibitory action of thallous ion on the movement of K⁺ across the membrane. To avoid precipitation of TlCl in the various reaction mixtures, the experiments with Tl⁺ were carried out in the presence of sulfate salts.

The results showed that at 8 mM Tl⁺ the leakage of K⁺ is reduced by about 50% (Fig. 1A) and that the effect of Tl⁺ occurs at all of the concentrations of Na⁺ employed (Fig. 1B) which suggests that Na⁺ does not compete with Tl⁺.

It is possible that Tl⁺ affected the loss of K⁺ by inhibiting the action of Na⁺, by blocking the pathway through which K⁺ is released, or by acting on both systems. However, Tl⁺ at a concentration of 5 mM did not affect the previously described (14, 16) Na⁺-induced release of oxygen uptake (Fig. 2). Apparently Tl⁺ prevents the movement of K⁺, but not that of Na⁺.

The effect of Tl⁺ on the uptake of K⁺ by K⁺-depleted mitochondria was also studied. Tl⁺ inhibits the uptake of K⁺, but the results shown in Fig. 3 indicate that the extent of the inhibition depends on the relative concentrations of K⁺ and Tl⁺. The inhibitory action of Tl⁺ is more marked at the higher Tl⁺:K⁺ ratios, which suggests that a competition exists between the two cations. Unfortunately, due to the nature of the experimental system employed a detailed kinetic analysis of the behavior of K⁺ and Tl⁺ cannot be made at the present.

The preceding experiments suggested that Tl⁺ acts on a system responsible for the inhibition of K⁺ movements across the membrane. Thus the amount of Tl⁺ that binds to mitochondria was measured. The data of Fig. 4 show the amount of 204Tl⁺ that appears in mitochondria incubated in sucrose (+ rotenone) or in the regular K⁺-depletion mixture. As in both systems almost identical results were obtained, the amount of Tl⁺ that appears in mitochondria probably corresponds to "bound" Tl⁺ and not Tl⁺ that has accumulated. At 8 nm Tl⁺, the concentration that induces maximal inhibition of K⁺ movements, about 12 nmol of Tl⁺ per mg of protein are bound.

The effect of Tl⁺ on K⁺ movements could be secondary to its action on electron transport or on the phosphorylation reaction. However, this possibility seems unlikely since concentrations of Tl⁺ that have accumulated. At 8 mM Tl⁺, the concentration that induces maximal inhibition of K⁺ movements, about 12 nmol of Tl⁺ per mg of protein are bound.

The release of K⁺ was measured in the complete incubation media as in Table I, with mitochondria prepared in sucrose, EDTA except that phosphate was omitted or 12.5 nmol of mersalyl per mg of protein were added to the mixture. The amount of phosphate was measured in the same mitochondrial suspension. The results are expressed as the amount of K⁺ and Pi (phosphate) that remained in the mitochondria after incubation.

![Fig. 1](http://www.jbc.org/)

**Fig. 1.** Effect of Tl⁺ on the release of K⁺. A, the experimental conditions were as in Table I with the complete incubation mixture, except that sulfate salts were employed instead of chloride salts, and the indicated concentrations of Tl⁺ (as the sulfate salt) were added. B, the experimental conditions were as in A except that the mixture contained the indicated concentration of Na⁺ (as the sulfate salt) and 8 mM Tl⁺ as shown. The results are expressed as the amount of K⁺ that remained in mitochondria.

| Reaction media | Mitochondrial K⁺ | Mitochondrial P₀ |
|----------------|-----------------|-----------------|
| Complete       | 21              | 23              |
| Complete − P₀  | 72              | 25              |
| Complete + mersalyl | 52        | 15              |

**Role of phosphate in release of K⁺**

The role of phosphate in the release of K⁺ was studied in the following way: in experiment A, the potassium-depleted mitochondria did not show K⁺ uptake. In nine preparations more than 60 nmol of K⁺ per mg of protein were taken up by the mitochondria, while in the rest of the experiments the uptake was between 25 and 50 nmol of K⁺. It is not possible at this time to explain this variability.

**Table V**

| Reaction media | Mitochondrial K⁺ | Mitochondrial P₀ |
|----------------|-----------------|-----------------|
| Complete       | 21              | 23              |
| Complete − P₀  | 72              | 25              |
| Complete + mersalyl | 52        | 15              |

**Fig. 2.** Polarographic traces of the effect of Tl⁺ on the Na⁺-stimulated oxygen uptake. The incubating conditions were 10 mM glutamate, 10 mM H₂PO₄ (adjusted with Tris base to pH 7.3), 10 mM Tris-sulfate (pH 7.3), 100 mM sucrose, and 1 mM EDTA in a final volume of 3.0 ml. Where shown the mixture contained 5 mM Tl⁺ as the sulfate salt Na⁺ was added at 75 mM as the sulfate salt. The numbers on the side of the trace indicate the rate of respiration (nanomols of oxygen min⁻¹ mg⁻¹).

**Fig. 3.** Effect of Tl⁺ on the uptake of K⁺. The experimental conditions were as in Table I, except that sulfate salts were employed as the chloride salts. In Experiment A the concentrations of added K⁺ (as the sulfate salt) were 25 mM and 50 mM. The results of this experiment are expressed as the percentage of inhibition of the uptake by the indicated concentrations of Tl⁺. In B, identical incubating conditions were employed, except that the concentration of K⁺ was varied as shown, and Tl⁺ was assayed at a concentration of 8 mM.
FIG. 4. Binding of $^{205}$Tl$^+$ to mitochondria. Mitochondria (5 mg of protein) were incubated in 1.0 ml of 0.25 M sucrose, 1 mM EDTA, and 8.6 $\mu$M rotenone at pH 7.3 (+---+) or in the complete K$^+$-depletion mixture (Table I) (O--O). After 3 min of incubation an aliquot was filtered, and the amount of $^{205}$Tl$^+$ in the mitochondria was measured.

FIG. 5. Effect of Tl$^+$ on the ADP to oxygen ratio and respiratory control of mitochondria. The incubating conditions were 10 mM glutamate, 10 mM phosphate (pH 7.3), 10 mM Tris-sulfate (pH 7.3), 10 mM K$^+$ (as the sulfate salt), 80 mM sucrose, and 1 mM EDTA in a final volume of 3.0 ml. ADP was added at a concentration of 0.33 mM. Tl$^+$ (as the sulfate salt) was added at the concentrations shown.

Tl$^+$ which suppress K$^+$ movements do not affect the respiratory rates or ADP to oxygen ratios of mitochondria (Fig. 5).

DISCUSSION

The results of this work show that under optimal conditions and in a period of 3 min close to 100 nmol of K$^+$ are released per mg of mitochondrial protein and that a significant, although variable, amount of K$^+$ can move into the mitochondria. K$^+$ movements of this magnitude have been observed only in the presence of ionophores (29) and uncouplers (30) and in prolonged periods of incubation (5, 6). Optimal movements of K$^+$ take place in mixtures that contain EDTA and phosphate and in which electron transport occurs; in the case of K$^+$ release, a monovalent cation other than K$^+$ is also required. In the absence of either of these components, the movement of K$^+$ is diminished, but not abolished. This suggests that electron transport, phosphate, EDTA, and the cationic environment contribute individually to poise the direction and the magnitude of K$^+$ movements. As efflux and influx of K$^+$ have similar characteristics except for their metal ion requirements, i.e. prevention of the Na$^+$-induced efflux by K$^+$ and inhibition of K$^+$ uptake by Na$^+$, it is logical to assume that the relative concentrations of Na$^+$ and K$^+$ govern the direction of the flux of K$^+$ across the membrane.

With respect to the events that occur during the movement of K$^+$, a mechanism is suggested in Scheme 1. Internal OH$^-$ was generated during electron transport exchanges for external phosphate (31, 32). Once on the inside, phosphate would induce the influx of an external cation. In a second step, internal phosphate and an internal cation (K$^+$) would be released from the mitochondria. This set of events would account for the observations that during the loss of K$^+$ from the mitochondria, phosphate undergoes a cyclic movement across the membrane (Table V), and also of the requirement for electron transport. Nevertheless, other alternatives are equally possible.

It has been reported (8-10) that EDTA reduces the Mg$^{2+}$ content of the mitochondria (8); thus it has been suggested that bound Mg$^{2+}$ is important for controlling the permeability of the membrane. However, K$^+$ influx in mitochondria which had been depleted of K$^+$ in the presence of EDTA also requires EDTA. Therefore, other factors besides the reported decrease in Mg$^{2+}$ content, such as binding of EDTA to the membrane, should be responsible for the increase in the permeability of the membrane to monovalent cations.

An objective of this work was to study whether or not movement of K$^+$ is affected by Tl$^+$, Tl$^+$, because of its ability to bind more strongly than K$^+$ to protein (21, 22), was considered a possible inhibitor of K$^+$ movements. Moreover, Tl$^+$ was recently reported to form complexes with valinomycin (33), the highly specific K$^+$ ionophore (29). Also Tl$^+$ may substitute for K$^+$ in the activation of the (Na$^+$-K$^+$)-activated ATPase (34) and pyruvate kinase (22), and it is accumulated by erythrocytes (34).

The inhibition of the influx and efflux of K$^+$ by Tl$^+$ indicates that both the inward and outward movements of K$^+$ take place through the same molecular system. Apparently, the same molecular entity reacts with external and internal K$^+$. Since Tl$^+$ does not inhibit the Na$^+$-stimulated oxygen uptake, the system seems specific for K$^+$ and may be concluded that Na$^+$ movements across the membrane occur through a system that is different from that through which K$^+$ is translocated.

The amount of bound Tl$^+$ required to inhibit K$^+$ translocation is of the order of 10 to 15 nmol per mg of mitochondrial protein. Since in our experimental conditions nonspecific binding cannot be excluded, this value is probably higher than the actual concentration of entities responsible for K$^+$ translocation. Moreover, we cannot discard the fact that phospholipids play a role in K$^+$ translocation.
According to the presently described experiments, we propose that K⁺ is translocated across the membrane through a system that critically depends on the cationic environment. Also, we propose that the influx and efflux of K⁺ are mediated through the same molecular system and that this is specific for K⁺.

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Characteristics of the movement of K+ across the mitochondrial membrane and the inhibitory action of Tl+.
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J. Biol. Chem. 1975, 250:5370-5374.

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