TABLE I

KINETIC CONSTANTS OF POTASSIUM ACCUMULATION BY SERIAL CORN ROOT SECTIONS *

ROOT SECTION, MM FROM TIP	K_s, MEQ/L		V_{max}, μEQ K × HR^{-1}			
			× GM FRESH WT^{-1}		× MG PROTEIN N^{-1}	
	− Ca	+ Ca	− Ca	+ Ca	− Ca	+ Ca
0– 5	0.34	0.89	8.73	3.01	1.48	0.51
5–15	0.44	0.44	2.36	0.77	3.60	1.18
15–25	0.47	0.32	1.63	0.71	3.17	1.38
25–35	0.55	0.11	1.77	1.01	2.74	1.57
Entire root	0.51	0.19	2.50	1.99	...	...

* Mean values from 4 expts. Values for entire primary roots were taken from a previous report (2) and inserted here for purpose of comparison.

absence of calcium it rises with increasing cell maturity; conversely, in the presence of calcium it falls. In the tip section calcium decreases the affinity between potassium and its carrier; in the more basal sections the affinity is increased.

On a fresh weight basis, V_{max}—the maximum velocity at which the tissue can accumulate potassium under these conditions—is maximal in the tip region and minimal in the 15 to 25 mm region. On a protein nitrogen basis, V_{max} is maximal in the 5 to 15 mm region in the absence of calcium and in the 25 to 35 mm region in its presence. Both with and without calcium the minimum velocity occurs in the root tip. Calcium depresses V_{max} less in the basal sections than in the tip section. Although these sections are larger and encompass a greater root length than those used by Brown and Cartwright, the data verify their observation that on a protein basis the immature cells are not as effective in potassium accumulation.

The changes in the kinetic constants and in their response to calcium with cell maturation can be explained in various ways. A number of carrier systems can be operative in potassium accumulation, each with different characteristics, the measured kinetic values being but the mean of the contribution to accumulation made by each carrier. During the ontogeny of the root cell the proportion or activity of the several carriers may change—and perhaps new carriers are introduced—with a resultant alteration of the constants of accumulation. On the other hand, a single carrier system with sequential steps may be altered in its biochemical and biophysical properties by the alteration of one or more of the steps. Kinetic studies are of limited value in deciding between these or other alternatives as they give only the overall characteristics of the individual reactions involved, and are insufficient to determine the complexity and diversity of the individual steps. We can only guess as to the steps occurring between the initial and the final phase, with the sole knowledge that the complex reaction has to obey the Michaelis-Menten kinetics. A similar difficulty involving the adenosine triphosphate activation of myosin has been reviewed by Morales, Botts, Blum and Hill (3).

SUMMARY

The kinetics of potassium accumulation by serial sections from corn root tips have been investigated. The kinetic constants, K_m and V_m, change with growth and maturation, and the nature and extent of the change is conditioned by the presence or absence of calcium ion. The velocity of potassium accumulation per unit protein nitrogen is least in the meristematic region. It is deduced that K is accumulated by a complex of carriers operating independently or sequentially, and that elements of the complex are changed during growth and maturation.

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OXIDATION OF MALONATE BY PEANUT MITOCHONDRIA [1]

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For many years malonate was considered a classical competitive inhibitor of succinic dehydrogenase (1). The assumption was made that it is a metabolically inert substance. However, the utilization of malonate by microorganisms (2–6), mammals (7–9), and fishes (10) suggests that this acid may play a role in organic acid metabolism. Studies with a partially purified enzyme obtained from *Pseudomonas* sp. (6) and mitochondria from rat kidney (9) suggest that malonate degradation proceeds by way of malonyl CoA, which is then decarboxylated to acetyl CoA and CO_2.

Malonate has been reported in the leaves of a

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TABLE I

COFACTOR REQUIREMENTS FOR OXIDATION OF MALONATE-1,3-C^{14}

COMPONENTS	% OXIDATION
Complete	24
- ATP	2
- CoA	2
- DPN	14
- TPN	16
- GSH	17
- $MnSO_4$	11

The complete reaction mixture contained 1 micromole of malonate-1,3-C^{14}, 5800 cpm, 0.5 ml of mitochondria (approximately 22 mg of protein) in 0.2 M TRIS-0.5 M sucrose, pH 7.2, with about 5×10^{-3} BAL, 10 micromoles of phosphate buffer, pH 7.1, 50 micromoles of KCl, 1 micromole of ATP, 0.3 micromole of CoA, 0.2 micromole of DPN, 0.1 micromole of TPN, 5 micromoles of GSH, 1 micromole of αKG, 1 micromole of $MnSO_4$, 0.2 ml of 20% KOH in the center well, 0.3 ml of 10 M H_2SO_4 in the sidearm, final volume 1.7 ml. Time of incubation, 2 hrs; temperature, 25° C; gas phase, air; % oxidation = $BaC^{14}O_3$ (cpm) × 100/substrate (cpm).

number of plants (11). However, the unequivocal utilization of malonate by plant tissues has not been demonstrated (12).

In the course of an investigation on the oxidation of propionate to CO_2 and acetate by peanut mitochondria (13) we observed a rapid release of $C^{14}O_2$ from 1 micromole of malonate-1,3-C^{14} (table I). An absolute requirement for ATP[4] and CoA was demonstrated. DPN, TPN, GSH, Mn^{++} and α-ketoglutarate enhance the rate of $C^{14}O_2$ released. While 1 micromole of malonate-1,3-C^{14} is oxidized 24 % in 2 hours, malonate-2-C^{14} under the same conditions is oxidized only 4.5 %. This would be expected if malonate is oxidized via acetyl CoA which then enters the Krebs cycle.

The path of malonate oxidation was determined by incubating malonate-2-C^{14} with peanut mitochondria in the presence of all the cofactors shown in table I. The reaction products were examined for Krebs cycle acids by standard paper chromatographic techniques.

[4] The following abbreviations are used: adenosine triphosphate, ATP; coenzyme A, CoA; diphosphopyridine nucleotide, DPN; triphosphopyridine nucleotide, TPN; glutathione, GSH; α-ketoglutarate, αKG; 2,3-dimercaptopropanol, BAL; 2-amino-2-hydroxymethyl-1,3-propanediol, TRIS.

When 0.1 micromole of malonate-2-C^{14} was oxidized, the Krebs cycle acids, citrate, malate and succinate became radioactive. It would appear from this evidence that at low concentrations, malonate is readily metabolized by the following scheme:

$$\text{malonate} \xrightarrow{\text{ATP, CoA}} \text{malonyl CoA} \rightarrow CO_2 + \text{acetyl CoA} \rightarrow \text{Krebs cycle}$$

The details of this sequence are now under investigation.

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