The Effect of Potassium on the Intestinal Transport of Glucose*

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ABSTRACT The rate of absorption of glucose, galactose, and 3-*O*-methylglucose was studied in the rat's small intestine perfused *in situ* with isosmotic solutions containing these sugars and Na_2SO_4 or K_2SO_4. The presence of high $[K^+]$ in the lumen enhances absorption of glucose but not that of galactose or of 3-*O*-methylglucose. The potassium stimulation is apparent at higher glucose concentrations where primarily carrier-mediated diffusion is involved in the translocation. In this case potassium stimulates transport even if it is the only cation in the lumen. The potassium-stimulated intestine produces more glycogen with higher specific activity than the control gut. Lactic acid production by the intestine is markedly enhanced if the intestinal lumen is perfused with a solution containing glucose and high $[K^+]$. It is concluded that potassium does not affect permeability or the specific sugar transport system of the gut, but enhances intracellular metabolic disappearance of glucose thereby creating a larger luminal intracellular concentration gradient which in turn enhances the rate of carrier-facilitated entry.

Simple sugars are absorbed by a specific transport mechanism from the intestine. The specificity of the mechanism is due to the carrier with which sugar molecules combine temporarily while passing across the lumen-facing membrane of the mucosal epithelium. The driving force responsible for translocation can be either a concentration difference between the lumen and the blood ("carrier-mediated diffusion"), or a "pump" which causes sugar to move against a higher concentration ("active transport"), or a combination of the two. Active sugar transport ("sugar pump") is dependent upon the presence of Na^+ in the lumen of the gut (for references see reference 1). The isolated surviving small intestine pumps sugar if Na^+ is the only cation in the bath and so far there is no evidence that the presence of any other cation is essential for this function.

Riklis and Quastel (2) described an interesting effect of K^+ upon absorption

* Dedicated to Professor F. Verzár on the occasion of his 80th birthday.

of glucose in the isolated small intestine of the guinea pig. In this preparation, the increase of K^+ concentration in the bath to 15 mM enhanced the mucosal to serosal translocation of glucose if this sugar was placed into the lumen at an initial concentration of 14 mM and was not present initially in the serosal compartment.

The present study was undertaken to examine potassium enhancement of glucose transport in some detail. Theoretically four possible mechanisms are available whereby augmentation of the transport rate of glucose can come about: (*a*) by increasing the passive permeability of the gut, (*b*) by stimulating the pump, (*c*) by increasing the amount or capacity of the carrier, and (*d*) by increasing glucose metabolism in the intestinal epithelium and thereby enhancing its concentration gradient between the lumen and the interior of the cell. The experiments described in this report indicate that enhancement of intestinal transport of glucose by potassium is due to augmentation of the intracellular glucose metabolism.

MATERIALS AND METHODS

Analytical grade glucose and galactose were used. 3-*O*-methylglucose was a gift from Ayerst, McKenna and Harrison, Ltd., and contained a small (<0.5%) amount of glucose as a contaminant. Radioactive glucose used was uniformly ^{14}C-labeled. Na_2SO_4 and K_2SO_4 were analytical grade preparations.

Reducing sugars were determined by the Nelson copper reduction method (3). Glucose was determined with glucose oxidase (4). Lactic acid was determined with the Barker-Summerson method in barium hydroxide–zinc sulfate–treated filtrates (5), and the Conway microdiffusion method adapted by Ryan (6) was utilized in the determination of its specific activity.

Glycogen in the intestinal tissue was determined by a modification of the method of Seifter et al. (7) as follows: 5 ml of 30% KOH was placed in a 15 ml Pyrex centrifuge tube, covered with aluminum foil, weighed on an analytical balance, and placed in a hot water bath. The tissue was rapidly excised, placed in the hot KOH, covered with aluminum foil, and reweighed. Thereafter the tubes were heated in a boiling water bath for 3 hr, with occasional stirring. After cooling to room temperature, 8 ml of absolute ethanol was added to each tube. The contents were mixed and kept in a refrigerator overnight, then centrifuged cold at 3000 RPM for 10 min. The supernatant was discarded, the pellet was stirred up with 10 ml of 95% ethanol, centrifuged; the sediment was treated with 10 ml of 60% ethanol and recentrifuged. Thereafter, the pellet was stirred again with 3 ml of water and centrifuged; the supernatant was carefully transferred into a container graduated to 10 ml. This water extraction procedure was repeated once, collecting the supernatant again in the same container. Finally, volume was adjusted to 10 ml, and aliquots were taken for analysis by the anthrone procedure.

^{14}C was assayed in a liquid scintillation spectrometer using the following scintillation liquid: 40 mg of 2-5-diphenyloxazole (PPO) + 1 mg of 1,4 *bis*-2(5-phenylox-

azolyl)-benzene (POPOP) in 10 ml of toluene + 6 ml of absolute methanol. 0.1 to 0.2 ml water solutions could be introduced into this medium.

Male Sprague-Dawley white rats were anesthetized with a subcutaneous injection of 1.5 g per kg of urethan. A 6 to 8 cm long loop of upper jejunum was cannulated and perfused with appropriate isosmotic solutions using either of the two methods described elsewhere (unless otherwise indicated, method I was empolyed as routine in these experiments, 8). From time to time, samples were taken from the perfusate and the sugar was analyzed. The disappearance of sugar from the perfusate was cal-

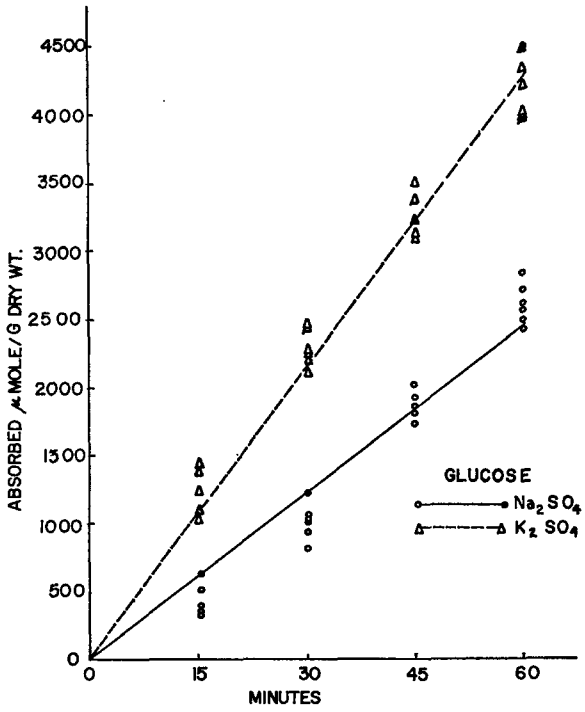

FIGURE 1. Absorption of glucose (148 mM) from solution made isosmotic with Na_2SO_4 or K_2SO_4.

culated as absorbed micromoles per gram dry gut weight; whereby, assumption was made that, in a given anatomical region, the dry weight of the gut is proportional to the absorptive surface.

RESULTS

1. *Effect of Potassium upon the Intestinal Transfer of High Concentration Sugar*

The perfusing fluid (40 ml) was composed of an equal mixture of an isosmotic solution of sugar (148 mM) and isosmotic Na_2SO_4 or K_2SO_4. Results depicted in Fig. 1 show that intestinal absorption of glucose is markedly enhanced when

potassium is the single cation in the lumen. However, Figs. 2 *a* and *b* indicate that neither the influx of galactose nor that of 3-*O*-methylglucose is influenced by the presence of a high [K^+] in the perfusate.

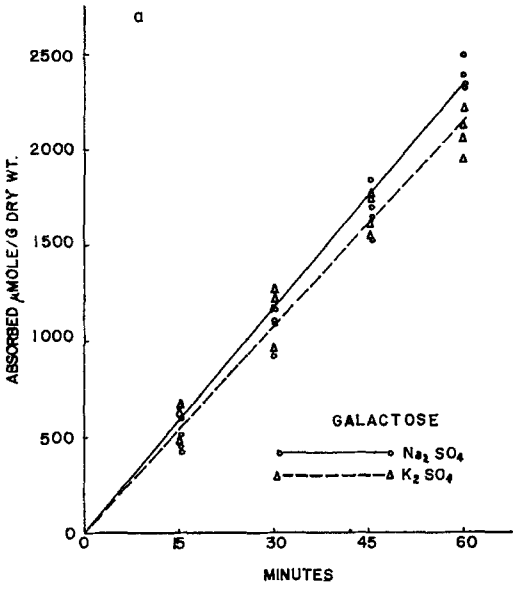

FIGURE 2 *a*. Absorption of galactose (148 mM) from a solution made isosmotic with Na_2SO_4 or K_2SO_4.

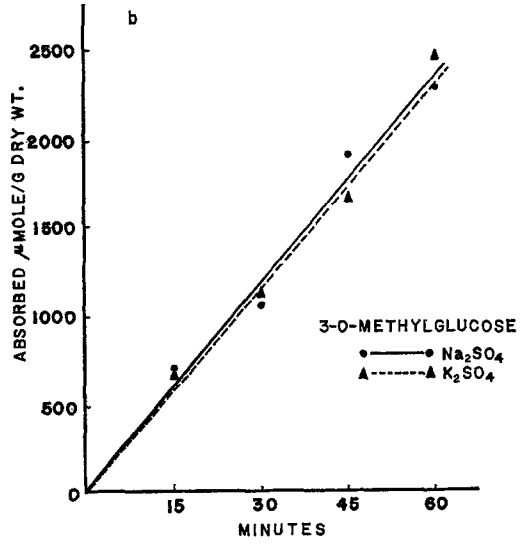

FIGURE 2 *b*. Same of 3-*O*-methylglucose (145 mM).

Phlorizin inhibits glucose influx from either sodium- or potassium-containing medium (Fig. 3 *a* and *b*), and potassium-stimulated transport is somewhat more sensitive to inhibition by the glucoside.

2. *Relationship between the K^+ Content of the Perfusing Medium and the Transport of Glucose*

Fig. 4 summarizes results of experiments in which the concentration of glucose in the lumen (2.78 mM) was kept lower than in the blood (>5 mM). In this case, absorption can take place only against a concentration gradient, viz. by active transport. Transport is almost completely inhibited if K^+ is the only

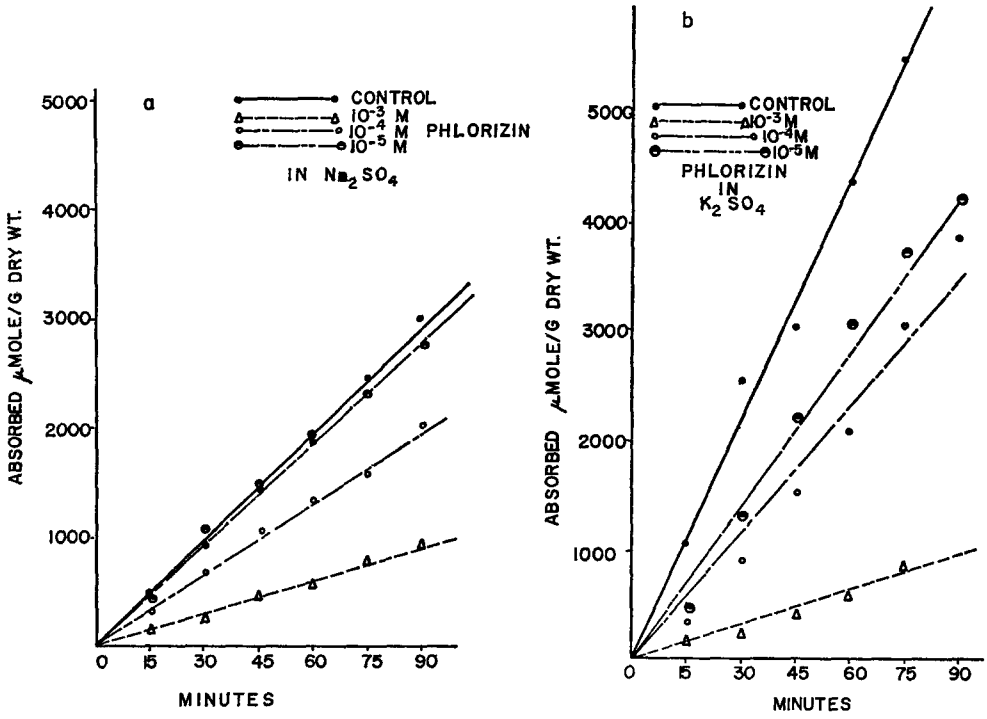

FIGURE 3 *a*. Effect of the presence of phlorizin in the lumen of the gut on the absorption of glucose (148 mM) from an isosmotic solution with Na_2SO_4.
FIGURE 3 *b*. Same in K_2SO_4.

cation in the perfusate, thereby corroborating previous observations (9). If the gut is perfused with mixtures of isosmotic Na_2SO_4 and K_2SO_4 in varying proportions, containing always 2.78 mM of glucose, the rate of active sugar transport is increased with the increase of the relative concentration of Na^+. K^+ in these experiments does not show a stimulating effect upon the glucose transport.

If, on the other hand, the glucose concentration is high (148 mM) so that absorption proceeds primarily via carrier-mediated diffusion, potassium ions already in low concentration stimulate transport and the stimulation is in-

creased proportionally with the increase of the relative concentration of potassium (Fig. 5).

3. *Effect of Concentration of Glucose upon the Stimulation of Intestinal Absorption by Potassium*

The stimulating effect of potassium depends on the relative concentrations of glucose, sodium, and potassium as illustrated in Figs. 6 and 7. Stimulation

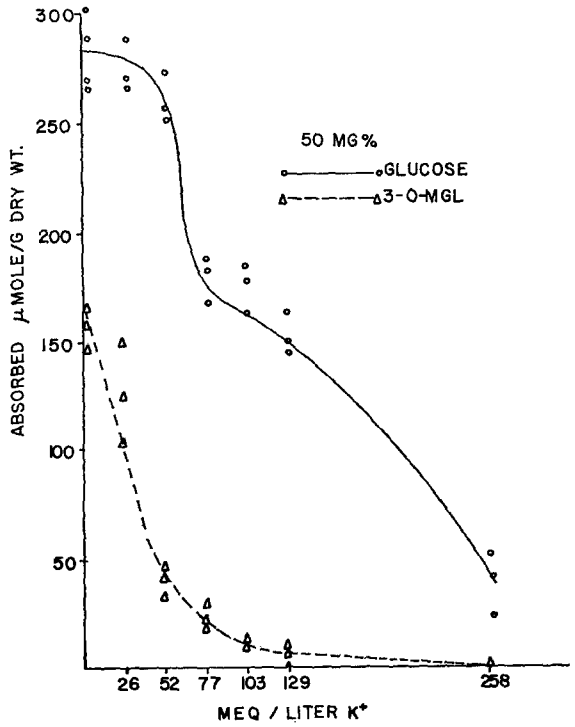

FIGURE 4. Effect of potassium on the active intestinal transport of glucose and 3-*O*-methylglucose. The perfusing fluid was a mixture of Na_2SO_4 and K_2SO_4 kept isosmotic (258 mEq/liter). Ordinate, transported sugar in 1 hr. Abscissa, concentration of K^+ (as the $[K^+]$ increased, the $[Na^+]$ decreased. At 0 $[K^+]$ the $[Na^+]$ was 258 mEq/liter).

seems to be maximal when glucose is present in approximately 140 mM concentration. This maximum is the same whether potassium is the only cation in the perfusate or whether sodium is also present. As long as the luminal glucose concentration is 110 mM or more, the potassium-produced stimulation is independent of the simultaneous presence of sodium. At concentrations <110 mM however, potassium stimulates only if sodium is also present. In this case, if potassium is the only cation in the lumen, absorption is not stimulated, but inhibited.

4. *Effect of Potassium upon Intracellular Glucose Metabolism*

In order to ascertain the changes in mucosal glucose metabolism, Intestinal glycogen and lactic acid production in the gut wall were studied.

For these studies the portal vein was drained with a continuous "transfusion" of the rat as described by Kiyasu et al. (10). A loop of the upper small intestine was perfused, using method II (8), with 145 mM of glucose, with or without ^{14}C-label, dissolved in either Na_2SO_4 or K_2SO_4 solution. The glycogen content and the specific activity of the glycogen in the gut tissue were deter-

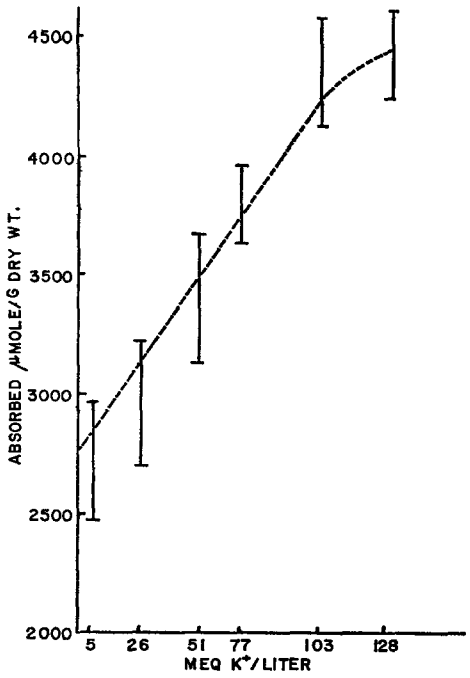

FIGURE 5. Effect of potassium on the absorption of glucose (148 mM) in 1 hr. The ratio of Na_2SO_4–K_2SO_4 was kept 0.5 times isosmotic (128 mEq/liter).

mined. In order to avoid a loss of glycogen during the separation of the mucosal layer, the entire loop of the gut was analyzed. In this way the tissue was introduced into the hot alkaline digestion medium within a few seconds following the excision.

Lactic acid was determined in the portal blood draining from intestines perfused with glucose or mannitol (for control) dissolved either in Na_2SO_4 or K_2SO_4. Net lactic acid production was calculated by the arterial-venous difference. In a separate set of experiments the intestine was perfused with ^{14}C-tagged glucose in Na- or K-containing medium and the specific activity of the portal lactic acid was determined by assaying for radioactivity the trapped acetaldehyde produced by its oxidation in the Conway microdiffusion chamber.

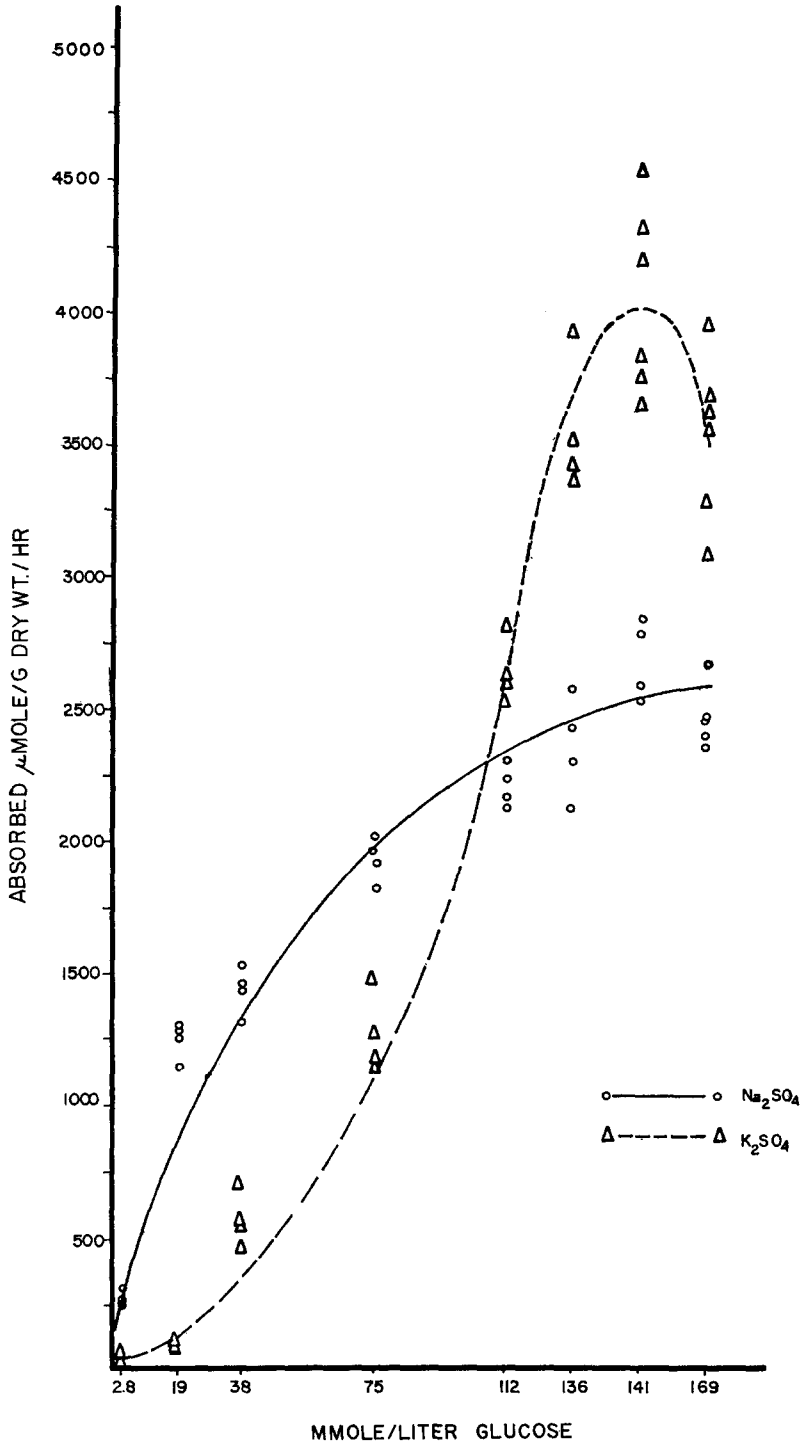

FIGURE 6. Absorption of glucose from perfusates containing increasing initial concentrations of glucose and made isosmotic with either Na_2SO_4 or K_2SO_4.

Portal drainage in these experiments was complete as indicated by failure to detect radioactivity in arterial plasma during and at the end of each experiment.

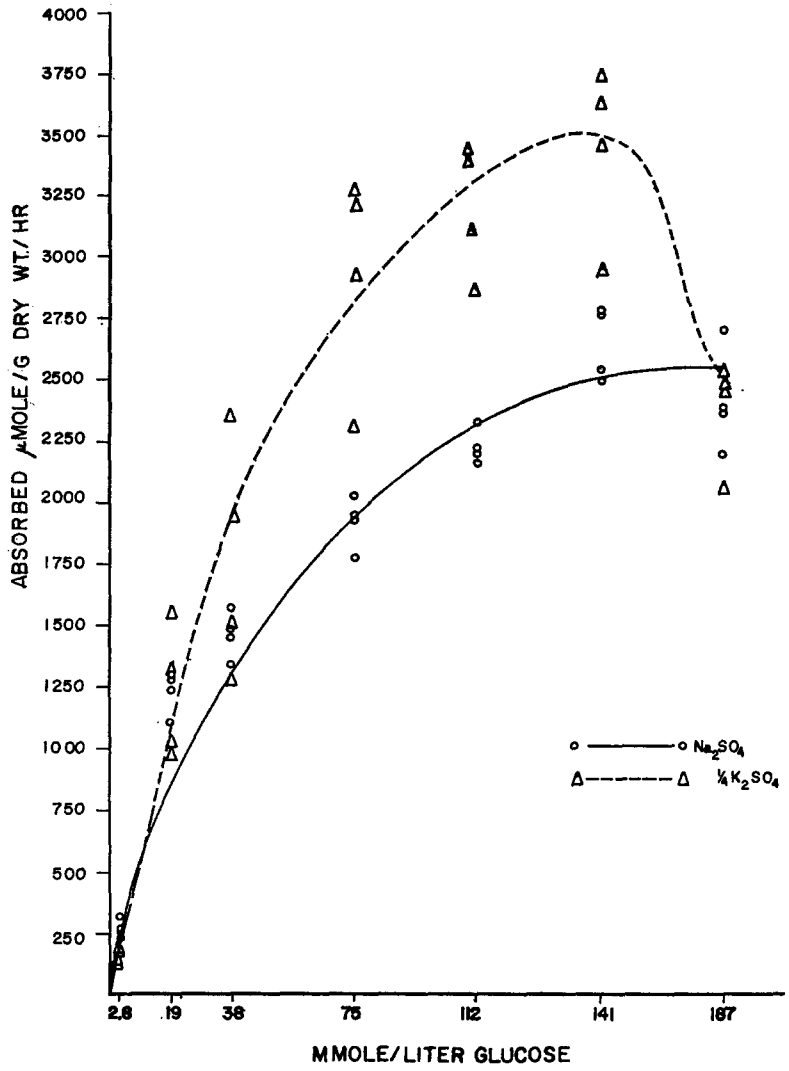

FIGURE 7. Absorption of glucose from perfusates containing increasing initial concentrations of glucose and made isosmotic either with Na_2SO_4 (unbroken line) or with a mixture of Na_2SO_4 and K_2SO_4; the concentration of the latter kept constant, 64 mEq/liter (broken line).

When potassium was the principal cation in the lumen, as compared with the control containing sodium, the absolute amount of glycogen increased only slightly in the gut tissue but its specific activity was enhanced significantly (Table I).

TABLE I

GLYCOGEN CONTENT OF THE SMALL INTESTINE AFTER 1 HR PERFUSION WITH 148 mM ^{14}C-GLUCOSE MADE ISOSMOTIC WITH EITHER Na_2SO_4 OR K_2SO_4

Animal No.	Intestinal perfusing solution		Absorbed	Glycogen		R^*
	Electrolyte	Glucose specific activity		In gut tissue water	Specific activity	
		CPM/mg	*mg/hr*	*μg/ml*	*CPM/mg*	
1	Na_2SO_4	15,220	28.1	81	7,930	0.52
2	Na_2SO_4	16,140	30.4	188	5,480	0.34
3	Na_2SO_4	20,470	29.5	66	7,000	0.34
4	Na_2SO_4	20,930	18.3	97	7,600	0.36
Average				108		0.39
5	K_2SO_4	15,000	37.6	124	12,300	0.82
6	K_2SO_4	13,000	42.5	213	10,800	0.83
7	K_2SO_4	15,760	40.6	214	15,670	0.99
8	K_2SO_4	15,200	50.4	182	13,100	0.86
Average				183		0.88

$$^*R = \frac{\text{Specific activity of glycogen}}{\text{Specific activity of glucose in the lumen}}.$$

Total lactic acid produced by the intestine filled with a potassium-rich glucose-containing medium was significantly greater than in the gut filled with a sodium-rich solution. In addition, the specific activity of lactic acid in the blood drained from the potassium-containing gut segment was also enhanced (Tables II and III).

TABLE II

LACTIC ACID IN THE PORTAL VEIN

The intestines were perfused with isosmotic solutions of various compositions.

Animal No.	Intestinal perfusate	Total blood collected from portal vein in 1 hr	Net production of lactic acid by the intestine in 1 hr
		ml	*mg*
1	145 mM mannitol + Na_2SO_4	12.0	0.0
2	145 mM mannitol + Na_2SO_4	13.5	1.76
3	145 mM glucose + Na_2SO_4	21.2	8.90
4	145 mM glucose + Na_2SO_4	16.2	2.42
5	145 mM mannitol + K_2SO_4	28.4	10.20
6	145 mM mannitol + K_2SO_4	17.0	5.27
7	145 mM glucose + K_2SO_4	25.8	31.73
8	145 mM glucose + K_2SO_4	15.9	20.67

DISCUSSION

Results of these experiments indicate that potassium enhances intestinal absorption of glucose and that stimulation of the translocation is proportional with the concentration of potassium. If sugar is present in <110 mM initial concentration, potassium stimulates only if sodium is also present in the perfusing fluid. If, on the other hand, the intraluminal concentration of glucose is high, so that the absorption proceeds primarily via carrier-mediated diffusion, potassium stimulates sugar transport even if it is the sole cation present in the lumen.

TABLE III

SPECIFIC ACTIVITY OF THE LACTIC ACID IN THE PORTAL BLOOD PLASMA

The intestine was perfused with isosmotic solution containing 146 mM of (135,000 CPM/ml) ^{14}C-labeled glucose and Na_2SO_4 or K_2SO_4

Animal No.	Cation in the perfusate	Total lactic acid produced in 1 hr	Specific activity of lactic acid	R^*
		mg	*CPM/mg*	
1	Na	9.56	39,000	0.29
2	Na	4.11	61,000	0.45
3	Na	4.72	68,000	0.50
Average				0.41
4	K	16.43	104,000	0.77
5	K	28.73	116,000	0.86
6	K	20.00	112,000	0.83
Average				0.82

$$^*R = \frac{\text{Specific activity of lactic acid}}{\text{Specific activity of glucose in the lumen}}.$$

It is significant that only intestinal transfer of glucose is enhanced by potassium and not that of galactose or 3-*O*-methylglucose. Such a high degree of substrate specificity excludes the possibility that potassium increased the passive permeability of the mucosal epithelium. Furthermore, the finding that potassium-enhanced glucose absorption is inhibited by phlorizin suggests that this transport is not a simple diffusion but mediated by carrier. The effect of phlorizin is rather specific in inhibiting the aldose-carrier combination (11, 12).

Yet, the increase of the rate of glucose absorption by potassium is not the result of a direct effect upon the transport carrier or upon the active transport system. Both the carrier and the pump are shared by glucose, galactose, and

3-*O*-methylglucose whereas potassium enhances only the translocation of glucose. It is noteworthy in this connection that active transport of 3-*O*-methylglucose is not only unstimulated but is totally suppressed even if the perfusing fluid is composed of an equal mixture of isosmotic K_2SO_4 and Na_2SO_4 (Fig. 4).

The fourth possibility, that potassium affects the intracellular metabolism of glucose, is substantiated by the finding that, when radioactive glucose is placed in the gut lumen together with high concentrations of potassium, the specific activity of glycogen in the epithelium increases. At the same time there was a sizeable increase in production of lactic acid with a higher specific activity. All this points to a stepped up glycogen and lactic acid formation in the presence of a high K^+ level. That K^+ increases the formation of glycogen in the liver is a known phenomenon. The findings that the absolute increase of intracellular glycogen was not as significant as the increase of its specific activity indicate a rapid turnover of glycogen. Perhaps the glycogen formation is an intermediary step in the production of lactic acid.

Based upon present results, the stimulating effect of potassium on the glucose absorption could be visualized in this way: glucose enters the mucosal epithelial cells with the aid of the carrier. This process is not affected by K^+. Having overcome the lipid diffusion barrier with the help of the carrier, the process of entry becomes essentially a diffusion (within a definite concentration range, when the carrier is still "unsaturated"); consequently, its rate is governed by the concentration gradient between the lumen of the gut and the intracellular boundary of the lumen-facing membrane. Potassium, by enhancing intracellular metabolic transformation of glucose, increases the concentration difference, hence the increased rate of passage of sugar. It is noteworthy that the K^+-caused enhancement was observed even at glucose concentrations (<110 mM) at which the sugar pump was functioning (provided that the sodium requirement for this function was met, see Fig. 8). This indicates that the entry and the concentrative "pumping" of sugar are two distinct processes and that carrier-mediated entry is independent of luminal ionic environment. This assumption is further supported by data presented in Figs. 6 and 7. It is clear from these that enhancement by potassium of glucose absorption at luminal sugar concentrations of <110 mM requires the simultaneous presence of sodium. As glucose concentration is increased, the sodium requirement gradually decreases until a glucose concentration of about 110 mM is reached; above this level, glucose absorption is more rapid from a pure potassium sulfate medium than if sodium ions are also present. It is interesting to note that 110 mM is about the glucose concentration at which the intestinal glucose carrier becomes saturated.

The data outlined above have been further analyzed graphically based upon the following consideration: should diffusion alone be involved in a given

transport process, the ratio of flux over initial concentration plotted against the initial concentration of the substrate would yield a straight line relationship. If, however, another reaction is superimposed upon the diffusion, the relationship would deviate from linearity, provided that the rate of the superimposed reaction does not increase proportionally with the concentration of the substrate. As indicated in Fig. 8, an approximately linear relationship was obtained for glucose transport if potassium was the only cation in the medium in the intestinal lumen. This indicates that, from a potassium-containing medium, glucose is absorbed by a process which is essentially diffusion (mediated by the carrier). If, however, sodium is also present, the ratio of

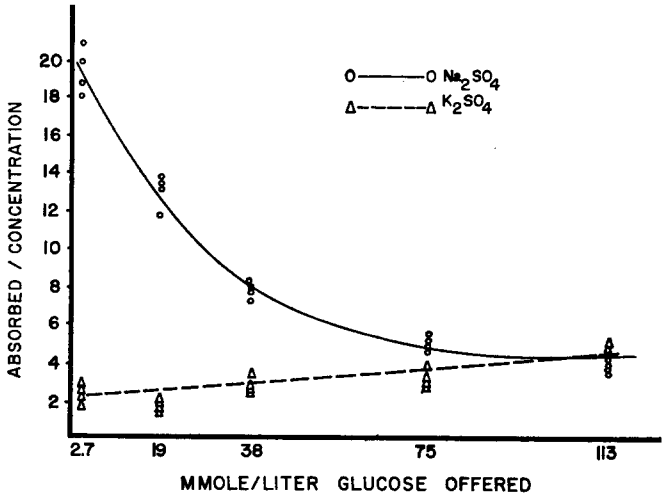

FIGURE 8. Ordinate, ratio absorbed in μmole/g dry gut/hr over initial glucose concentration. Abscissa, initial glucose concentration in the perfusate. The perfusate was made isosmotic with either Na_2SO_4 or K_2SO_4. For further explanation see text.

flux/concentration increases inversely with the concentration of glucose. Thus sodium stimulates another process which is superimposed upon the mediated diffusion. This second process is obviously the functioning of the pump. However, at a certain glucose concentration the two curves meet. At this point, active transport is dwarfed by the rapid rate of mediated diffusion and the transport is the same, both in a pure potassium- and a pure sodium-containing medium. This is the same concentration (about 110 mM) at which, as mentioned above, the transport mechanism becomes saturated.

Experimental findings as well as graphical analysis indicate that when glucose is offered to the intestinal epithelium in a concentration of 110 mM or less, two processes are involved in absorption: a carrier-mediated diffusion and an active transport. The active process takes place only if sodium is present at the lumen-facing membrane of the epithelium. The carrier-mediated entry, how-

ever, proceeds even from a sodium-free medium. Present study thus corroborates the previous assumption that sodium is needed primarily for the functioning of the pump while the carrier-mediated diffusion is not sodium-dependent (13).

The original finding of Riklis and Quastel (2) with regard to the enhancement of glucose absorption by potassium is confirmed by the present experiments; moreover in the light of the present experiments a logical explanation for potassium stimulation can be offered. It should be pointed out, however, that Riklis and Quastel studied absorption in general and did not have the ideal experimental setup for the study of active transport proper. In their experiments sugar was present initially only in the mucosal compartment so that transport took place along the gradient thus favoring carrier-mediated diffusion; consequently it is difficult to draw the clear-cut conclusion that purely active sugar transport was functioning in those experiments. Thus, it is not clear whether the study of Riklis and Quastel proved either that potassium stimulates or that a lack of sodium inhibits active transport. The first of these assumptions is definitely disproved by the present study while the second assumption was proven subsequently by experiments more adequately suited to the study of active transport proper (14).

The present experiments were conducted *in situ* in the intestine with its undisturbed normal circulation carrying continuously a plasma containing a relatively high amount of sodium to the intestinal epithelium. One could expect that, if the lumen is perfused with an isosmotic potassium sulfate solution, the mobile sodium ion may leak out into the lumen and be again rapidly absorbed, thus escaping detection in the perfusate. Yet, when the perfused fluid was actually analyzed only traces of sodium could be detected after perfusing with isosmotic K_2SO_4 for 1 hr or more. Although the possibility of a rapid leakage and then reabsorption of sodium cannot be entirely eliminated, the possible significance for sugar transport of a small amount of sodium present for short periods of time at the brush border can be assessed by examining Fig. 4. In this figure, absorption of 3-methylglucose, placed in a very low concentration in the lumen, was examined at various potassium levels. The potassium level was varied by changing the sodium/potassium ratio in the isosmotic concentration. The concentration of sugar was low enough to enable the reasonable assumption that the pump was involved in transport. The data indicate clearly that even if sodium sulfate constituted 50% of the perfusing fluid the sugar pump was not yet activated. This proves that far more than trace amounts of sodium are needed for the functioning of the pump. Consequently, even if trace amounts of sodium leak into the lumen during *in situ* experiments, one can assume that this would not affect the transport mechanism.

In view of the present study it is interesting to speculate as to what extent

intracellular glucose metabolism does contribute to the rate of absorption. The theory that intracellular metabolism may be responsible for preferential absorption of glucose is rather old. It was clearly formulated by Verzár (15) who proposed that glucose was "selectively" absorbed because a large concentration gradient of free sugar between the lumen and the intracellular space was created through its phosphorylation in the mucosal epithelium. Recent findings, particularly concerning the functioning of the specific carrier and the mechanism of active transport, suggest that phosphorylation (or some other type of intracellular metabolic change) is probably not the immediate cause of selective glucose absorption. Nevertheless, present study indicates that, under certain conditions, intracellular sugar metabolism can contribute to the factors which determine the rate of absorption from the intestine.

All experiments described above were performed with potassium placed in the lumen and no attempt was made to correlate the effects with concentration changes in tissue potassium. However, recent preliminary experiments in this laboratory indicate that various treatments which cause an increment of intracellular $[K^+]$ in intestinal mucosa produce the same effect as high intraluminar $[K^+]$, which, incidentally, also causes an intracellular increase of this cation in the epithelium (16). This could indicate that the effects observed in this study are due to increased intracellular potassium.

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