Active Transport of D-Xylose in the Isolated Small Intestine of the Bullfrog

ULRIK V. LASSEN and T. Z. CSÁKY

From the Department of Pharmacology, University of Kentucky College of Medicine, Lexington

ABSTRACT Fluxes of D-xylose-1-C^{14} (xylose) across the wall of the isolated intestine of the bullfrog were studied. When sodium was the principal cation in the mucosal bathing fluid, the transport rate of xylose from the mucosa to the serosa was about 5 times greater than the transport rate from the serosa to the mucosa, indicating an active intestinal transport for this sugar. With potassium as the principal cation on the mucosal side, the transport rate of xylose from the mucosal to the serosal compartment is reduced about 5 to 6 times without appreciable change in the serosal to mucosal transport. The asymmetry was also considerably reduced when ouabain was added to the mucosal and serosal compartments. The data confirm the in vitro and in vivo observations indicating active transport of xylose and are also in accord with the earlier findings that active transport of sugars in the intestine is dependent upon the presence of sodium ions in the mucosal compartment and is inhibited by cardioactive steroids. Since the chemical constitution of xylose does not meet the requirements which were hitherto considered necessary for active transport of sugars in the intestine, this structural requirement has to be revised.

INTRODUCTION

The intestinal transport of D-xylose (xylose) is characterized by a much slower rate than that of glucose or galactose and by an apparent proportionality between the sugar concentration and the rate of absorption. Earlier this behavior was interpreted as an indication of diffusion (1); however, Wilbrandt and Rosenberg (2) pointed out that this kinetics is consistent with a carrier transport with high K_m. Salomon et al. (3) produced good evidence for the presence of a mobile xylose carrier in the intestine, as they showed inhibition of the xylose movement by glucose in the isolated rat gut and a "counterflow" of xylose against a glucose gradient. Such kinetic behavior could indicate a common mobile carrier for the two sugars by reasoning parallel to the sugar

transport in the red cell (4, 5); but, as in the erythrocyte, these experiments did not give any indication of an active or "up-hill" transport of xylose.

Under the assumption that xylose could serve as an indicator for the equilibrating part of the "pump and leak" transport system for actively transported sugars, studies were undertaken of fluxes of xylose in the isolated intestine of the bullfrog. It soon became apparent that the transport of xylose itself was asymmetrical. The present report describes in detail these experiments, which lead to the conclusion that the transport of xylose in the isolated intestine of the frog must be active, as judged from a steady asymmetry of transport in the two directions (6). Similar to other active transport systems, the asymmetrical transport of xylose is dependent upon the presence of sodium ions in the mucosal bathing fluid and is reduced under the influence of a "pump" poison such as digitalis. The present experiments, together with the observations described previously (7), that xylose actually accumulates in the serosal compartment against a higher concentration in the isolated intestine of the frog and that this accumulation is also dependent on sodium and inhibited by digitalis, are accepted as evidence of active xylose transport in the small intestine.

MATERIALS AND METHODS

The experiments were performed in the isolated small intestine of the bullfrog (*Rana catesbeiana*) during the spring and summer months (except the experiments in Table III which were performed in the fall). The frogs, weighing 300 to 350 g, were decapitated and their spinal cords pithed. The abdomen was immediately opened, and the lower part of the duodenum and the upper part of the jejunum were excised and mounted in a plexiglass flux chamber described by Csáky and Thale (8). The length of the intestine between the two points was about 6 cm. By this method both compartments were agitated, the serosal by a small animal respirator, the mucosal by a bubble device which also kept the bathing solution aerated. The procedure permitted repetitive sampling from both compartments.

All incubating media were modified frog Ringer's solutions, as described previously by Csáky (9). The principal anion was sulfate to minimize fluid movements, and the principal cations were either sodium ("Na_2SO_4-Ringer's") or potassium ("K_2SO_4-Ringer's"). The media were modified by the addition of 11 mM/liter of Tris-buffer to maintain a pH of 7.3.

D-Xylose-1-C^{14} was obtained from Nuclear Research Chemicals, Orlando, Florida. According to the manufacturer, the sugar was synthesized from threose and $NaC^{14}N$ and was claimed to have a chromatographic radiopurity of better than 99%. The method of synthesis excluded any contamination by other pentoses or by hexoses.

At "zero time" the labeled xylose, having a specific activity of 0.29 to 0.62 mc per mmole, was added to either the mucosal or the serosal compartments. The molar concentrations of xylose ranged from 0.2 to 2 mM, except in one series of experiments (presented in Table VII) in which the sugar concentration was considerably higher.

After the addition of the labeled sugar to one of the compartments, 50 μl samples were taken from the opposite compartment at 10 to 15 min intervals. For calculation

of the transport rate constants, initial and final samples were taken also from the compartment of the C^{14} addition. Radioactivity was measured in the Packard Tricarb Liquid Scintillation Spectrometer. To each sample was added 12 ml of scintillation fluid of the following composition: 4 g PPO, 100 mg of POPOP, 1 liter of toluene, 600 ml of absolute methanol.

The following procedure was employed for ascertaining whether the radioactivity in these experiments really represented unchanged xylose. At the end of a typical flux experiment, the fluids of the mucosal and serosal compartments were carefully collected. The gut was then removed from the flux chamber and extracted with boiling distilled water. The extract was combined with the mucosal-serosal fluids and assayed for radioactivity. 93 to 98% of the originally introduced radioactivity was recovered in the combined solutions. In another set of experiments the mucosal fluid, tissue extracts, and serosal fluid were separately evaporated to dryness. The residue was extracted with methanol and chromatographed on Whatman No. 1 filter paper with the top layer of a solvent mixture composed of 3 parts ethyl acetate, 1 part glacial acetic acid, and 3 parts water. A reference xylose spot was developed simultaneously and localized by spraying with silver-nitrate ammonia. The dry filter paper strips were cut into 4 to 5 cm lengths, placed in a scintillation vial, 12 ml of scintillation fluid added, and counted in the liquid scintillation spectrometer. 85 to 95% of the radioactivity in the initial spot was recovered in the combined fractions, and all the radioactivity was localized in a spot which was located identically with the reference xylose spot.

All gut segments were tested for leakage at the end of the experiments by the addition of a high concentration of methylene blue on the mucosal side. The gut was then carefully observed for 15 min with the serosal agitation stopped. This way even minor leaks were detected.

The experiments were performed at room temperature of 23–25°C.

CALCULATIONS

From the steady-state slope of increase in counting rate in the compartment opposite to the one to which labeled xylose was added, (ΔCPM/hr), the volume in milliliters of this compartment (V_{trans}) and the mean of initial and final counting rates in the compartment of C^{14} addition (CPM_{cis}), a rate constant for the transport of radioactive xylose per length of intestine (ca. 6 cm) was calculated:

$$k = \frac{\Delta \text{ CPM/hr}}{\text{CPM}_{cis}} V_{trans}$$

As no correlation was found between the transport rate and the concentration of "carrier-free" labeled xylose (0.2 to 2 mM), these concentrations must be well below saturation of the transport system. The "k" is a usable arbitrary figure to express the steady rate by which xylose passes from one compartment to the other, provided that $CPM_{trans} \gg CPM_{cis}$ even at the end of the experi-

ment so ΔCPM_{cis}/hr is nearly constant during the experiment. As seen from the tables and Fig. 1, these two conditions are found in the experiments.

Statistical figures and conclusions are based on standard formulae (10).

RESULTS

For the calculation of the rate constants it was necessary to have a steady-state transport over a period of time. Fig. 1 shows the serosal counting rate after the addition of labeled xylose (2.5 μc) to the mucosal compartment containing 15 ml of Na_2SO_4-Ringer's. In this experiment the increase in counting rate in the serosal compartment followed a straight line after an initial delay of approximately 40 min. In other similar experiments it was found that a steady flow was obtained usually between 30 and 100 min. For this reason the

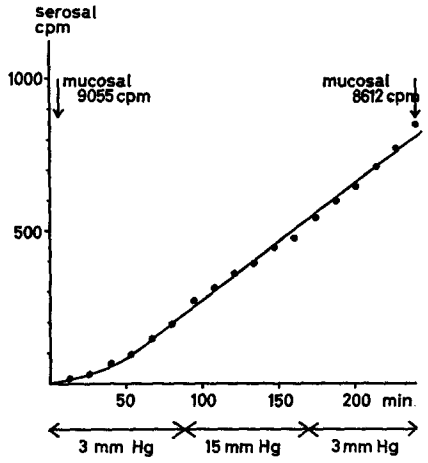

FIGURE 1. Serosal counting rate (counts per minute = CPM) after addition of 2.5 μc xylose-1-C^{14} to 15 ml Na_2SO_4-Ringer's in the mucosal compartment. Serosal compartment contained 9 ml Na_2SO_4-Ringer's. The pressure differences between mucosal > serosal compartments are shown below the time scale. The mucosal counting rates at beginning and end of the experiment are indicated at the arrows.

slope of the curve between 100 and 200 min after addition of the labeled xylose was used for calculation of the transport. The figure also shows the initial and final counting rate in the mucosal compartment (CPM_{cis}).

With the method employed, a small pressure difference had to be maintained between the two sides of the gut to keep it distended and to allow ample circulation in the mucosal compartment. The pressure difference was usually about 3 mm Hg. Wilson (11) found that, in experiments with everted sacs of intestine, the contraction and the resulting higher pressure in the inside of the sac may affect the transport rate. At pressures as low as mentioned above, vigorous peristalsis was frequent; but no effect on the transport rate could be seen. To investigate whether a change in pressure would affect the transport in the present type of experiment, the pressure distending the gut in the experiment in Fig. 1 was transiently raised from about 3 mm Hg to about 15 mm Hg. Despite considerable expansion and loss of peristalsis of the gut at the higher pressure, no effect could be seen on the rate by which xylose passed from the

mucosal into the serosal compartment. In all the succeeding experiments the pressure in the mucosal compartment was maintained about 3 mm Hg positive to the serosal compartment.

From experiments similar to the above, the transport rate constants for xylose-1-C^{14} from mucosal to serosal ($M \rightarrow S$) were determined. The results

TABLE I

TRANSPORT OF D-XYLOSE-1-C^{14} FROM MUCOSA TO SEROSA

Both mucosal and serosal compartments contained Na_2SO_4-Ringer's.

Experiment No.	Serosal volume	ΔCPM hr^{-1} (serosal)	CPM_{cis} (mucosal)	k*
	ml			
1	8	300	13747	0.175
2	9	90	4150	0.198
3	9	74	4536	0.148
4	9	138	9129	0.136
5	9	235	8834	0.240
6	9	155	7517	0.186
			Mean	0.181 hr^{-1}

*k calculated according to equation (1).

TABLE II

TRANSPORT OF D-XYLOSE-1-C^{14} FROM SEROSA TO MUCOSA

Both mucosal and serosal compartments contained Na_2SO_4-Ringer's.

Experiment No.	Mucosal volume	ΔCPM hr^{-1} (mucosal)	CPM_{cis} (serosal)	k*
	ml			
1	10	39	12549	0.031
2	11	13	12435	0.011
3	15	36	6900	0.076
4	15	31	8660	0.054
5	15	9	6407	0.022
6	15	25	15022	0.025
7	15	35	17817	0.029
			Mean	0.035 hr^{-1}

* k calculated according to equation (1).

are summarized in Table I. The table lists the individual values and the arithmetic mean. In these experiments both compartments contained Na_2SO_4-Ringer's.

Using the Na_2SO_4-Ringer the transport of labeled xylose from the serosal to the mucosal compartments ($S \rightarrow M$) was determined in comparable experiments. The results are summarized in Table II. It may be seen that the $M \rightarrow$

S transport rate of xylose is 5.2 times greater than the transport in the opposite direction, namely, $S \rightarrow M$. This difference is highly significant ($p < 0.001$).

Using the same technique as that described above, the two-way transports of H_2^3O were also investigated in a separate series of experiments. Na_2SO_4-Ringer's was placed in both mucosal and serosal compartments. Table III summarizes the results. It is shown that the flux of tritiated water was nearly the same in both directions, in the same system in which a marked transport asymmetry was noted for xylose.

TABLE III

TRANSPORT OF H_2^3O AND D-XYLOSE-1-C^{14} IN THE ISOLATED SMALL INTESTINE OF THE TOAD

Both mucosal and serosal compartments contained Na_2SO_4-Ringer's, 10 ml each.

(*a*) Radioactivity added to mucosal compartment

	H_2^3O			D-Xylose-1-C^{14}		
Experiment No.	ΔCPM hr^{-1} $\times 10^3$	CPM *cis* $\times 10^3$	k*	ΔCPM hr^{-1}	CPM_{cis}	k*
1	156	1,121	1.39	550	27,890	0.197
2	253	1,020	2.48	980	28,145	0.348
3	262	1,161	2.26	1,500	30,895	0.486
4	195	1,189	1.64	1,000	31,070	0.322
		Mean	1.94 hr^{-1}		Mean	0.338 hr^{-1}

(*b*) Radioactivity added to serosal compartment

	H_2^3O			D-Xylose-1-C^{14}		
Experiment No.	ΔCPM hr^{-1} $\times 10^3$	CPM_{cis} $\times 10^3$	k*	ΔCPM hr^{-1}	CPM_{cis}	k*
1	190	1,195	1.59	210	28,355	0.074
2	281	1,200	2.34	300	31,280	0.096
3	244	1,228	1.99	450	29,175	0.154
4	179	1,160	1.54	250	31,000	0.081
		Mean	1.87 hr^{-1}		Mean	0.101 hr^{-1}
		r‡	1.04		r‡	3.35

* k calculated according to equation (1).

‡ $r = \dfrac{\text{Transport rate constant from mucosa to serosa}}{\text{Transport rate constant from serosa to mucosa}}$.

The rate constants for the xylose transfer in Table III differ somewhat from those in the other tables in this work. This finding may be correlated with the fact that the experiments in Table III were performed in the late fall, instead of spring and early summer, as in the other experiments.

The presence of sodium ions in the medium bathing the mucosal surface of the gut is essential for the active transport of sugars, amino acids, and pyrimidines (8, 9, 12, 13). If sodium is replaced by potassium or lithium, the active transport of these substances is drastically diminished.

To see whether the xylose transport is dependent upon the presence of sodium ions, the unidirectional transports of radioactive xylose were studied in intestines bathed with a Ringer solution in which the sodium was replaced

TABLE IV

EFFECT OF K_2SO_4-RINGER'S IN THE MUCOSAL COMPARTMENT ON TRANSPORT OF D-XYLOSE-1-C^{14}

The serosal compartments contained Na_2SO_4-Ringer's.

(*a*) Radioactivity added to mucosal compartment

Experiment No.	Serosal volume	ΔCPM hr^{-1} (serosal)	CPM_{cis} (mucosal)	k^*
	ml			
1	9	40	15934	0.023
2	9	60	16021	0.033
3	9	47	16322	0.026
4	9	30	8297	0.032
5	9	32	10296	0.028
			Mean	0.028 hr^{-1}

(*b*) Radioactivity added to serosal compartment

Experiment No.	Mucosal volume	ΔCPM hr^{-1} (mucosal)	CPM_{cis} (serosal)	k^*
	ml			
6	15	48	14058	0.051
7	15	40	15087	0.040
8	15	51	14205	0.055
			Mean	0.049 hr^{-1}

* k calculated according to equation (1).

by potassium (K_2SO_4-Ringer's). In the experiment described in Table IV the mucosal compartment contained K_2SO_4-Ringer's, the serosal compartment Na_2SO_4-Ringer's. It is seen that the replacement of sodium by potassium on the mucosal surface drastically decreased the transport $M \rightarrow S$, while the transport $S \rightarrow M$ had not changed considerably. The ratio between transports $M \rightarrow S/S \rightarrow M$ is 0.57, in contrast to the 5.3 ratio of the experiments when the gut was bathed in sodium containing Ringer's. This result strongly suggests that an active transport of xylose is eliminated if sodium is replaced by potassium in the medium bathing the mucosal surface. Table V shows the

results of experiments when the mucosal compartment contained Na_2SO_4-Ringer's and the solution bathing the serosal surface of the gut contained K_2SO_4-Ringer's. The results of these experiments are very similar to those described in Tables I and II with a resulting high $M \rightarrow S/S \rightarrow M$ ratio. Therefore the replacement of sodium in the *serosal* compartment did not considerably alter the transport ratio. The results of these experiments are very similar

TABLE V

EFFECT OF K_2SO_4-RINGER'S IN THE SEROSAL COMPARTMENT ON THE TRANSPORT RATES OF D-XYLOSE-1-C^{14}

The mucosal compartments contained Na_2SO_4-Ringer's.

(*a*) Radioactivity added to mucosal compartment					
Experiment No.	Serosal volume	ΔCPM hr^{-1} (serosal)	CPM_{cis} (mucosal)	k*	
	ml				
1	9	240	18988	0.114	
2	9	167	8281	0.182	
				Mean 0.148	hr^{-1}
(*b*) Radioactivity added to serosal compartment					
Experiment No.	Mucosal volume	ΔCPM hr^{-1} (mucosal)	CPM_{cis} (serosal)	k*	
	ml				
3	15	26	14690	0.026	
4	15	37	14277	0.039	
5	15	34	14263	0.036	
6	15	108	30585	0.053	
7	15	90	31326	0.043	
8	15	96	31000	0.046	
				Mean 0.049	hr^{-1}

* k calculated according to equation (1).

to those obtained earlier with regard to the actively transported 3-*O*-methylglucose in the isolated intestine of the toad (8).

Cardioactive steroids are potent inhibitors of active sodium transport in a number of tissues (14, 15). These steroids also inhibit the active intestinal transport of sugars, amino acids, and pyrimidines (16, 17). It was therefore of interest to investigate the effect of a cardiac steroid, ouabain, on the xylose transport. In the experiments described in Table VI, ouabain was added to both the mucosal and serosal compartments to make a final concentration of 10^{-5} M. In the experiments described in Table VI *a* and VI *b* both mucosal and serosal compartments were bathed with Na_2SO_4-Ringer's whereas in the

experiment under Table VI *c* the mucosal compartment contained K_2SO_4-Ringer's. The presence of ouabain inhibited the transport $M \rightarrow S$ if sodium was present on the mucosal surface; viz., if active transport was maintained. The transport $S \rightarrow M$ was not significantly inhibited. The transport $M \rightarrow S$ which was already decreased by the replacement of sodium by potas-

TABLE VI

EFFECT OF OUABAIN (10^{-5}M IN BOTH SEROSAL AND MUCOSAL COMPARTMENTS) ON TRANSPORT RATES OF D-XYLOSE-1-C^{14}

(*a*) Mucosa: Na_2SO_4-Ringer's
Serosa: Na_2SO_4-Ringer's

	Radioactivity added to mucosal compartment			
Experiment No.	Serosal volume	ΔCPM hr^{-1} (serosal)	CPM_{cis} (mucosal)	k^*
	ml			
1	9	257	18652	0.124
2	9	183	18584	0.089
3	9	215	17464	0.111
			Mean	0.108 hr^{-1}

(*b*) Mucosa: Na_2SO_4-Ringer's
Serosa: Na_2SO_4-Ringer's

	Radioactivity added to serosal compartment			
Experiment No.	Mucosal volume	ΔCPM hr^{-1} (mucosal)	CPM_{cis} (serosal)	k^*
	ml			
4	15	48	15034	0.048
5	15	43	14494	0.045
6	15	65	14664	0.067
			Mean	0.053 hr^{-1}

(*c*) Mucosa: K_2SO_4-Ringer's
Serosa: Na_2SO_4-Ringer's

	Radioactivity added to mucosal compartment			
Experiment No.	Serosal volume	ΔCPM hr^{-1} (serosal)	CPM_{cis} (mucosal)	k^*
	ml			
7	9	45	18215	0.022
8	9	48	18764	0.023
9	9	47	18738	0.023
			Mean	0.023 hr^{-1}

* k calculated according to equation (1).

sium on the mucosal surface was not further inhibited by ouabain (Table VI *c*, compare with Table IV *a*).

As in reactions in which specific enzymes are involved, transport processes often show a saturation phenomenon. Thus the rate constant will decrease with increasing concentration of the substrate. Such a saturation phenomenon has been observed repeatedly in connection with the intestinal transport of sugars. In Table VII experiments are described in which the xylose concentration in the mucosal compartment was increased by mixing isosmotic xylose solution with the sodium sulfate–Ringer leaving a final xylose concentration of 0.1 M. In this experiment a type of transport was obtained which was considerably different from that presented in Table I. The rate constant was now in the same order of magnitude as in the experiments in which the mucosal

TABLE VII

EFFECT OF HIGH XYLOSE CONCENTRATION ON THE TRANSPORT OF D-XYLOSE-1-C^{14} FROM MUCOSA TO SEROSA

The mucosal compartments contained 7.0 ml 3.23% xylose and 8 ml Na_2SO_4-Ringer's giving a final xylose concentration of 0.10 M. The serosal compartments contained Na_2SO_4-Ringer's.

Experiment No.	Serosal volume	ΔCPM hr^{-1} (serosal)	CPM_{cis} (mucosal)	k*
	ml			
1	9	75	19768	0.034
2	9	98	19662	0.045
			Mean	0.040 hr^{-1}

* k calculated according to equation (1).

compartment contained K_2SO_4-Ringer's. At this concentration the active transport system of xylose seemed to be saturated. It should be noted that in these experiments, because of the high xylose concentration, the sodium ion concentration had to be lowered to maintain the isotonicity of the solution. However, such a sodium concentration is sufficient to maintain active sugar transport (18). Also, it is known that at this concentration level the active transport of sodium itself is not heavily affected (15).

DISCUSSION

It is generally assumed that xylose is absorbed unchanged from the intestine and that it is metabolized at a very slow rate in the animal body. However, in these experiments in which the intestinal transport of very small amounts of xylose was studied, the problem of a possible metabolic change during the transport process was reinvestigated. With a total recovery of about 90% or better, all the radioactivity in both *cis* and *trans* compartments as well as in

the gut tissue was localized in a chromatogram spot located identically with the reference xylose. Therefore it is safe to assume that the transport of radioactivity in these experiments is a true measure of the transport of xylose.

The considerable deviation of the transport ratio (mucosal-serosal over serosal-mucosal) from unity could be accepted as a strong indication that the substance is transported, provided the asymmetry cannot be accounted for by "solvent drag." Andersen and Ussing (19) have shown that the active movement of sodium ions followed by chloride ions and water can carry small molecules through the epithelial membrane, thus simulating an asymmetry in transport. If the freely movable chloride ions are replaced by sulfate ions (for which most plasma membranes are nearly impermeable), this solvent drag effect of active sodium transport is minimized. In a series of experiments in the surviving toad intestine, no measurable net movement of fluid was found in either direction (8). This finding was confirmed in the present work, as it was found that no appreciable transport asymmetry of H_2^1O was detectable when Na_2SO_4-Ringer's was the bathing medium for the gut. Consequently, the asymmetry of the xylose transport must be indicative of active transport. The actual thermodynamical proof of active transport, viz. the net movement against a concentration gradient both in vitro and in vivo, has been presented in a previous communication (7).

Wilson and Vincent (20) have performed a transport study of xylose in the hamster intestine using a xylose concentration of 30 mM and employing a chemical determination for xylose. They found little indication of asymmetry of the transport system. If it is permissible to compare the intestine of the frog with that of the hamster, the active transport system for xylose seems to be saturated somewhere between 2 mM (the highest concentration of xylose in most of the present study) and 30 mM.

At concentrations between 0.2 and 2 mM no correlation was found between mucosal and serosal transport rate, and it was concluded that the active transport system for xylose was far from saturation. This circumstance permitted an estimate of the ratio between the serosal and the mucosal concentration at steady state, in other words, when no net transport occurred. This final ratio is the ratio between the two rate constants ($M \rightarrow S$ and $S \rightarrow M$). However, to reach this point would require a very long time and it is therefore not feasible experimentally.

The simple arguments and equations used are invalid when saturation phenomena occur. Wilbrandt and Rosenberg (2) have given a full account of active transport systems superimposed upon equilibrating systems and with a common "carrier" acting in the two systems in two different forms.

Csáky (13) has proposed a similar mechanism in which sodium ions are essential for the carrier-pump combination to operate to produce active transport. The equilibrating and the active transport systems have a carrier in

common. Provided that the number of carrier sites are fixed, more of this carrier should be available for the equilibrating system under conditions where active transport is absent. Active transport of xylose is diminished or inhibited under the condition where no active sodium transport from the mucosal to the serosal side takes place. In this case (Tables IV and VI) the transport of xylose from the serosal to the mucosal side actually appears to be accelerated, even though the differences from the control (Table II) are hardly significant in the present small series. As this phenomenon may shed some light on a possible connection between active and equilibrating transport systems, it deserves further investigation.

Recently, Lawrence (21) reported a study of the transport of various sugars in the isolated intestine of the bullfrog. He did not find significant net transport of xylose against a concentration gradient. This finding was expected in view of the present results. Lawrence used a procedure whereby the serosal fluid was removed every hour for the measurements of the xylose by a chemical method. The initial mucosal concentration of xylose was 62.5 mg % (= 4.2 mM), a concentration at which the transport presumably is not saturated, as judged from the figures of the present paper with concentrations up to 2 mM. A net transport may be expected; but, as the rate constant for xylose flux is small under similar experimental conditions and with a comparable length of intestine, the net transport per hour is too small to be determined by a chemical method. So the apparent controversy is well explained by the different experimental approaches in the study of Lawrence and in the present paper.

The finding described in this paper and in a preliminary communication (7) of selective intestinal transport of xylose, has been confirmed by Alvarado (22) using the uptake of xylose in everted hamster intestine as a measure of transport. It was found that even though xylose was not accumulated, the concentration in the tissue was affected by agents affecting active transport of sugars (phlorizin, 2,4-dinitrophenol).

A considerable amount of effort was made to establish a structural prerequisite for the active transport of a sugar. It has been suggested (11, 23) that, among others, the presence of a hexose structure is essential for a sugar to be actively transported. The present paper, together with the previous communication (7), provides essential proof that xylose is actively transported. This finding will necessitate a revision of the previously established structural requirements for active sugar transport.

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