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Gastrointestinal transit and digestibility of maltitol, sucrose and sorbitol in rats: a multicompartmental model and recovery study

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Abstract. Using data obtained with a dye marker and the gavage technique, the kinetics of gastrointestinal transit of different loads of sugar substitutes (maltitol, sorbitol) and sugar (sucrose) in the rat were analysed using a linear multicompartmental model over a range from the realistic to the non-physiologic high, of carbohydrate intake levels and using only a few experimental time points. The model gave detailed insight into intestinal propulsion and gastrocecal transit time. Rate constants of transport between the compartments investigated were determined; they showed characteristics which could be related to the substance and the dosage administered. Analyses of the gastrointestinal content and calculations of the intestinal net water movement showed that the digestibility and absorption of the disaccharide sugar alcohol, maltitol, in the small gut depended inversely on the dose ingested. For all substances tested, caloric availability in the small intestine was calculated. At a physiological low level of maltitol intake, the results also indicated an insignificant calorie-saving effect in comparison to sucrose, an effect based mainly on the slow absorption rate of the maltitol cleavage product sorbitol.
Key words. Linear multicompartmental model; gastrointestinal tract; sugar substitutes; maltitol; sucrose; sorbitol; absorption; small intestine; caloric value.

It is of physiological and nutritional interest to characterize the gastrointestinal transit of digesta in humans and animals[1]. Several methods are available to measure the digestive flow through distinct intestinal regions[2–5], and attempts have been made to describe this mathematically[6–8].

The rate of intestinal passage is influenced by various factors[9,10] and has physiological consequences. For instance, increased orocecal transit time was shown to be closely related to carbohydrate malabsorption in the small intestine[11,12].

This may be of importance when investigating the caloric availability of alternative carbohydrates, e.g. the sugar substitutes; they are known to influence the gastrointestinal transit time by exerting dose-related osmotic effects[13,14].

Estimates of the calories available from a new sugar substitute of the disaccharide sugar alcohol type, i.e. maltitol (D-glucopyranosido-1,4-sorbitol), exist for humans and animals[15,16]. A reduction of caloric utilization of up to 50% of that of the normal dietary carbohydrate sucrose (D-glucopyranosido-1,2-fructofuranosid, 4 kcal/g) has been suggested[17,18]. However, the results concerning the fractions of maltitol cleaved and absorbed in the small intestine, or transformed by the colonic microflora to absorbable fermentation acids, are contradictory[15,16,19,20].

In vitro studies using small intestinal disaccharidases from rats and maltitol have questioned the postulated low physiologic calorie yield[21]. In vivo studies with different animal species suggested that the slow rate of absorption of the sorbitol moiety of maltitol is the decisive mechanism limiting its availability via the small intestine[22,23]. Feeding experiments with rats (including isocaloric starch replacement) revealed a significantly retarded weight increase in the group fed with maltitol[24]. However, the latter study, like most former experiments, involved introducing huge, non-physiological amounts of maltitol; such conditions are obviously unsuited for estimating the caloric availability of disaccharide sugar alcohols at a realistically low intake[13].

Therefore, this study aimed to develop a compartmental model describing the gastrocecal transit and the sites of absorption of different doses of sugar and sugar substitutes. Based on experimental data obtained with rats and using a dye marker to determine intestinal transit, the dose range screened included small-, medium- and non-physiological high doses of the tested carbohydrates. Ad-

ditionally, parallel gastrointestinal recovery studies and calculations of the intestinal net water movement were performed to elucidate the caloric value of maltitol in the small intestine as compared to sucrose and sorbitol.

Material and methods

In vivo experiments. Four different doses of crystalline maltitol (Roquette Frères) – 150, 300, 600 and 1200 mg/kg body weight (= mg/kg b.wt) – were administered by gastric gavage to male juvenile Wistar rats weighing about 100 g. The maltitol was given in a hypotonic electrolyte solution containing 0.075% (w/v) phenol red (Merck) as a transit marker. Sucrose and sorbitol (Merck) were the reference substances. Details of the experimental design have already been published[13,25]. 0.5, 1, 2 and 3 h after administration, the rats were anaesthetized and killed[14]. The gastrointestinal tract was quickly removed and subdivided into six compartments: stomach, first half, third quarter and distal quarter of the small intestine, cecum and colon. The contents were flushed with cold saline and analysed colorimetrically for phenol red[26]. Aliquots were frozen at – 20 °C until carbohydrate analysis was done. Maltitol, free glucose and sorbitol were determined by an HPLC procedure[27], sucrose and its monomers by an enzyme assay (Boehringer). Using the recovery data and assuming the caloric content of maltitol, sucrose and sorbitol to be 4 kcal/g with equal parts spread among the monomer fractions of maltitol and sucrose, the caloric contribution delivered to the small intestine within the first hour after ingestion was estimated. Gastrointestinal net water movement was calculated as described previously[13,14].

Statistics and mathematics. Unless otherwise indicated, in each case the results of the randomized investigation are presented as the mean and the standard deviation of at least 6 experiments. Dose effects on gastrointestinal marker distribution were examined by an analysis of variance (one-way classification). Significant differences between the dosages tested were checked by Tukey's multiple comparison method. All tests were realized with $\alpha = 0.05$ (two-sided). The calculations were performed with the software packages SAS V. 6.04[28], PCNONLIN V. 3[29] and MATHEMATICA V. 1.20[30] on a PC-80386 using MS-DOS V. 4.01.

The development of the compartmental model of gastrointestinal transit was based on the observed marker distribution and assuming a unidirectional marker flow. Given that the marker was administered as a bolus, the initial (a) and final (b) conditions for the model were:

(a) $t = 0$, $x_1 = 1$ (= 100% of the marker recovered is present in the proximal gastrointestinal compartment x_1, i.e. the stomach),
and $x_2 = x_3 = \ldots.. x_6 = 0$ (= 0% of the marker recovered is present in any of the distal compartments).

(b) $t = \infty$, $x_1 = x_2 = \ldots. x_5 = 0$ and $x_6 = 1$,

where the state variables x_1, $x_2, \ldots x_6$ represented the relative amounts of phenol red distributed in the compartment concerned at the given time points t [hours]. Constants characterizing the transport rates (per h) between the intestinal compartments ($k_1 \ldots k_5$) were calculated by PCNONLIN[29] with a modified Gauss–Newton method[31] for fitting nonlinear regression functions by least squares[32,33]. High values for k indicated rapid passage and vice versa. The period necessary after intragastric administration for marker recovery in the cecal compartment to reach 10% was calculated by interpolation and defined as gastrocecal transit time.

Results

Mathematical description of gastrointestinal transit. The total recovery of phenol red in 324 experiments was 84% ± 7%. A schematic drawing, figure 1, represents the gastrointestinal segments investigated.

Assuming linear kinetics, the system could be expressed by linear, first order differential equations[34]:

$$dx_n = k_n \cdot x_n - k_{n+1} \cdot x_{n+1}$$

n representing the number of the compartment in question (fig. 1), $k_n \cdot x_n$ describing the filling (not considered in the case of the stomach) and $- k_{n+1} \cdot x_{n+1}$ the evacuation of a compartment.

In the table, k_1 to k_5 are specified for the different experimental series.

The transport rates of phenol red between the gastrointestinal segments were dependent on the substance and dosage administered. As seen for the negative control ('blank' in the table), the passage rates from the stomach to the first half of the small intestine (k_1, see also fig. 1) and from the latter to the third quarter (k_2) were relatively high. In contrast, in the distal parts of the intestinal tract, the speed of marker transport fell drastically: low values were found for k_3 (transport from the third quarter to the fourth quarter of the small gut) and for k_4 (from the fourth quarter to the cecum). Since in all test series the amounts of marker recovered in the colon within the experimental time were low or undetectable, k_5 resulted in negative or very low data (table).

Generally the transport constant k_1 of the test substances had values comparable to the blank series. The values were reduced with high doses of maltitol or sorbitol only (600 and 1200 mg/kg b.wt), indicating delayed stomach emptying (table). With a small dose of sucrose, k_2 corre-

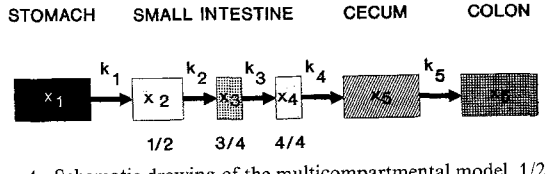

Figure 1. Schematic drawing of the multicompartmental model. 1/2, 3/4 and 4/4 symbolize the first half, the third quarter and the fourth quarter of the small intestine. See text for further description.

Transport rate constants (k_1 to k_5) and gastrocecal transit times characterizing the gastrointestinal marker flux between different compartments (stomach, first half, third quarter and distal quarter of small intestine, cecum and colon) after intragastric administration of different doses of sugar or sugar substitutes to rats.

Substance	Initial [mg/kg b.wt]	k_1	k_2	k_3	k_4	k_5	Gastrocecal transit time
Blank	0	3.731	5.921	0.715	0.240	−0.139	1h 40min
Maltitol	150	3.786	4.934	0.550	0.200	−0.306	1h 55min
	300	3.433	4.436	0.900	0.302	−0.227	1h 28min
	600	2.653	2.383	0.870	0.516	−0.152	1h 27min
	1200	2.516	1.929	1.072	0.678	0.040	1h 21min
Sucrose	150	3.473	5.928	0.923	0.296	−0.088	1h 27min
	300	3.053	3.631	0.965	0.190	0.112	1h 58min
	600	3.480	4.000	0.830	0.300	0.261	1h 42min
	1200	3.121	3.953	1.000	0.297	0.007	1h 33min
Sorbitol	150	3.818	3.655	0.893	0.857	0.090	1h 04min
	300	3.337	3.748	0.964	0.876	0.122	1h 04min
	600	2.818	3.031	1.514	0.971	0.086	0h 58min
	1200	2.384	2.473	1.465	1.186	0.253	1h 01min

sponded to that of the blank. However, with higher sucrose concentrations k_2 was reduced, indicating a delayed passage of dye into the third quarter of the small intestine. The reduction of k_2 was more pronounced when maltitol and sorbitol were administered and was in proportion to the increase in the initital dose. On the other hand, k_3 as well as k_4 for maltitol and sorbitol indicated an acceleration of transport in proportion to increases in the initial concentrations. The k_4 for 600 and 1200 mg/kg b.wt maltitol, and for all sorbitol concentrations, illustrated high rates of marker transport into the cecal compartment. In experiments with sucrose, such effects were not observed (table).

The gastrocecal transit time after maltitol administration (table) was in the range 1 h 21 min to 1 h 55 min and apparently shortened with increased initial concentrations. Transit times with sucrose were of the same order of magnitude but without any comparable relation to the dosage administered. The shortest stomach-to-cecum passages (about 1 h, table) were observed with all doses of sorbitol.

Figure 2 shows the observed results and the corresponding fitted kinetics of marker distribution in the gastrointestinal tract for the lowest and highest dose of maltitol applied (i.e., 150 and 1200 mg/kg b.wt). Because the colon lacked substantial marker recovery during the test period, this compartment was not considered.

For both maltitol concentrations, by 0.5 h after ingestion about 80% of the marker had already left the stomach and had entered the small intestine; most was found in its proximal half ('first half' in fig. 2). With the small dose of maltitol, the peak level was lower and emptying of the compartment was already completed after 2 h instead of 3 h as seen with 1200 mg/kg b.wt of maltitol.

With both concentrations, on average about 90% of the marker had left the stomach 1 h after administration. Most of the dye was then recovered in the distal parts of the small intestine. The 1-h marker peak occurred in the third quarter of the small gut ('third quarter' in fig. 2). However, peak values differed significantly and a lower level was noted for the high maltitol admixture. No termination of compartment evacuation was noted within the experimental time for both series.

In the distal segment of the small intestine ('fourth quarter' in fig. 2) no marker peak occurred within the 3 h of experimental time with 150 mg/kg b.wt maltitol but was noted after only 2 h with 1200 mg/kg b.wt.

In agreement with the data on the shortened gastrocecal transit time and the relatively high k_4 value for 1200 mg maltitol/kg b.wt in the table, the fitted curve described an enhanced cecal filling with this high dose, starting about 1 h after administration of the bolus ('cecum' in fig. 2). Experiments with sorbitol yielded similar results for the transit of the highest and the lowest doses. However, compared to maltitol, emptying of the distal small intestine and cecal filling were substantially enhanced in all

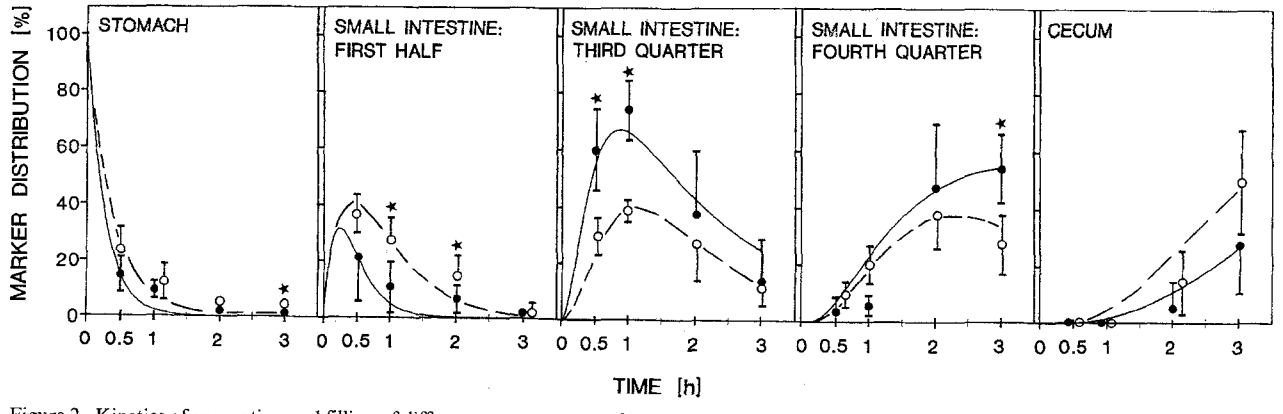

Figure 2. Kinetics of evacuation and filling of different compartments of the gastrointestinal tract of rats over 3 h after intragastric administration of different doses of maltitol. Points and solid lines mark the 150 mg/kg b.wt, open symbols and dashed lines the 1200 mg/kg b.wt series, respectively. Symbols indicate measured data; the lines are the fitted curves. * indicates significant differences between the measured marker distribution. Calculating the intervals of confidence for the parameters of the model (see table) revealed significant differences ($p < 0.05$) between the kinetics of transit of the two dosages in the small intestinal compartments.

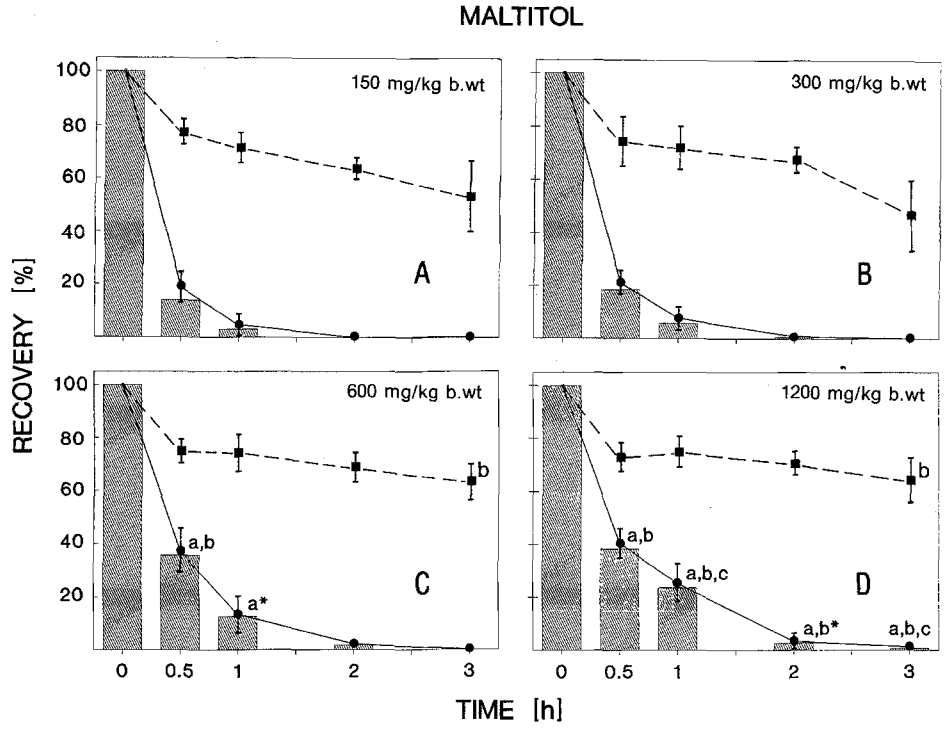

Figure 3 A–D. Relative recovery of maltitol components in the gastrointestinal tract of rats 0.5, 1, 2 and 3 h after intragastric administration of different doses. Columns correspond to recovery of disaccharide sugar alcohols and the initial amount is represented at t = 0. Solid lines indicate the recovery of the glucose fraction of maltitol (free and bound), dotted lines that of the sorbitol part. Standard deviations for columns were in the same range as marked for the glucose recovery. Significant differences are indicated: a = versus the 150, b = versus the 300 and c = versus the 600 mg/kg b.wt series. Marked at the columns, they are equally valid for maltitol and glucose recovery; * indicates a significance for maltitol only.

series (compare k_4 and 'gastrocecal transit time' in the table).

With sucrose, however, no significant differences in gastrointestinal marker distribution over time were revealed when the different doses administered were compared.

Gastrointestinal recovery of maltitol, sucrose and sorbitol. The relative recovery of uncleaved substance was significantly higher in the experiments with 600 and 1200 mg maltitol/kg b.wt compared to the tests with 150 and 300 mg/kg b.wt (compare columns A and B with C and D in fig. 3). With low doses, the percentage of maltitol recovered was already minor after 1 h and zero 2 h after ingestion. However, with 1200 mg maltitol/kg b.wt, uncleaved residues of the initial dose were still recovered (mainly in the distal quarter of the small intestine and in the rat ceca) as much as 3 h after ingestion (fig. 3 D).

Only traces of free glucose were analysed in the gastrointestinal samples; therefore, the relative glucose recovery indicated in figure 3 A–D was based mainly on the calculated amount of bound glucose in maltitol and consequently followed the same kinetics over time as indicated for the recovery of intact sugar substitute (compare columns and solid lines in fig. 3 A–D).

By contrast, the relative recovery of sorbitol in free form was substantial in all maltitol experiments (equal to the difference of the values indicated by the columns and the dotted lines in fig. 3 A–D). Two out of six rats receiving 300 mg maltitol/kg b.wt had an extremely shortened intestinal transit time and marker was detected even in their large intestines; an early onset of intracecal degradation may be responsible for the lowest mean recovery of sorbitol 3 h after ingestion in this series (fig. 3 B).

With the lowest dose of maltitol the 1-h recovery of the initial glucose content was only 5% and the degree of hydrolysis was as high as 97% (compare fig. 3 A). The availability of the glucose moiety liberated would already correspond to about 1.9 kcal/g. Additionally, the energetic contribution of 29% of the sorbitol part of maltitol (since 71% was its mean recovery after 1 h, fig. 3 A) would increase the small intestinal caloric availability to about 2.5 kcal/g. In contrast, the same calculation performed with values derived from the experiments with the high dose of maltitol (1200 mg/kg b.wt) would result in only 2 kcal/g furnished by the small intestine since 25% of the glucose and 75% of the sorbitol part were still recovered after 1 h (fig. 3 C).

Irrespective of the dose administered, the gastrointestinal fate of sucrose was characterised by a drastic decrease in detectable substance within 0.5 h of ingestion (fig. 4, A–D). With the low initial doses (150 and 300 mg/kg b.wt) only minimal amounts of free glucose and fructose were measured in the gastrointestinal tract and the cleavage of sucrose was immediately followed by the disappearance of the cleavage products from the gut lumen (fig. 4 A and B). On the other hand, in experiments with

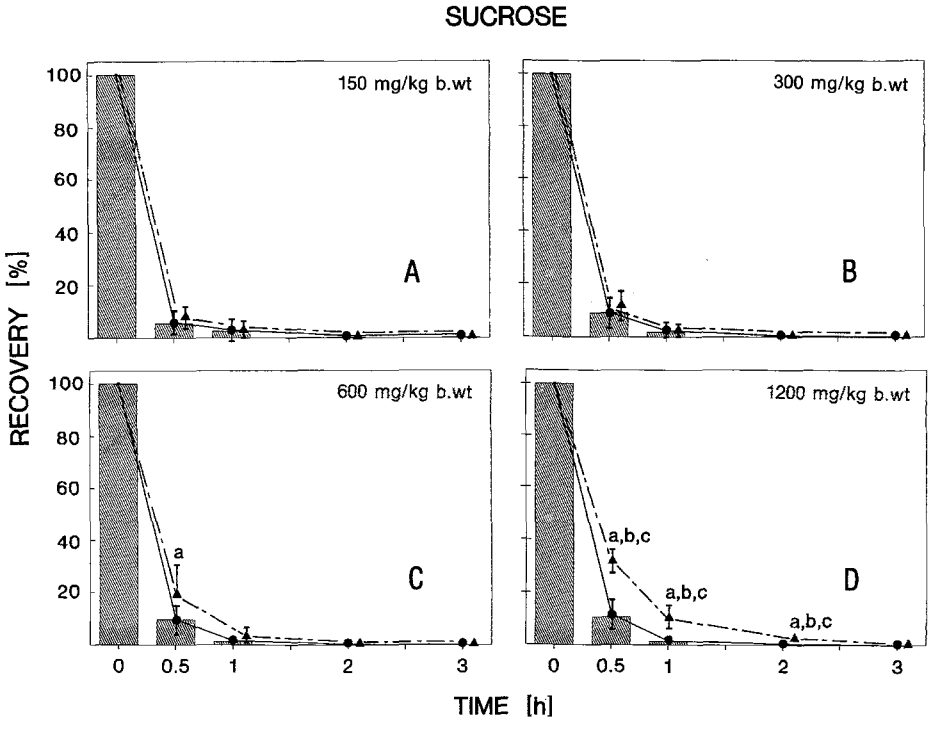

Figure 4A–D. Relative recovery of sucrose components in the gastrointestinal tract of rats 0.5, 1, 2 and 3 h after intragastric administration of different doses. Columns correspond to recovery of disaccharides and the initial amount is represented at t = 0. Solid lines indicate the recovery of the glucose fraction of sucrose (free and bound), dotted lines that of the fructose part. Standard deviations for columns were in the same range as marked for the glucose recovery. Significant differences for fructose recovery are indicated: a = versus the 150, b = versus the 300 and c = versus the 600 mg/kg b.wt series.

600 and 1200 mg/kg b.wt of sucrose, some free fructose was found mainly in the distal small intestinal contents, still 2 h after administration of the highest dose (fig. 4C and D). Since almost total splitting and disappearance was observed in the small intestine within the first hour after ingestion (fig. 4, A–D) total caloric availability (i.e. 4 kcal/g) can be expected even with high doses. The low residual amounts of liberated fructose recovered up to the second hour of experiments in rats fed the non-physiological high dose (fig. 4D) would lead to a reduction of only approximately 4% of caloric availability in the small intestine if calculated for the 1-h time point as above.

When comparing the different initial dosages, no significant differences in gastrointestinal disappearance were revealed when pure sorbitol was administered to the rats. The relative recovery was about 80% of the initial doses after 0.5 h and decreased continuously to an average of 60% after 3 h of experimental time (fig. 5). The 1-h recovery of an average of 75% of initial sorbitol enabled us to calculate a small intestinal absorption of 1 kcal/g.

Net water movement in the upper gastrointestinal tract. In order to obtain more information on small intestinal absorption capacity under the different experimental conditions, the gastrointestinal net water movement was calculated after the 1-h digestion time and plotted as a function of the different doses of sugar or sugar substitutes administered (fig. 6).

No significant correlation between intestinal waterflux and dose administered was revealed in the sucrose experiments. On the contrary, a linear regression between increasing doses of the applied sugar substitutes and the reduction of intestinal water absorption capacity was clearly demonstrated (fig. 6).

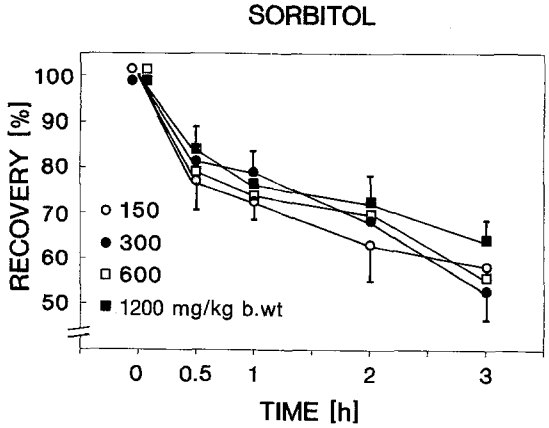

Figure 5. Relative recovery of sorbitol in the gastrointestinal tract of rats 0.5, 1, 2 and 3 h after intragastric administration of different doses. At each time point, the standard deviations are indicated for the lowest or highest recovery only; they were of the same order of magnitude for the intermediate values.

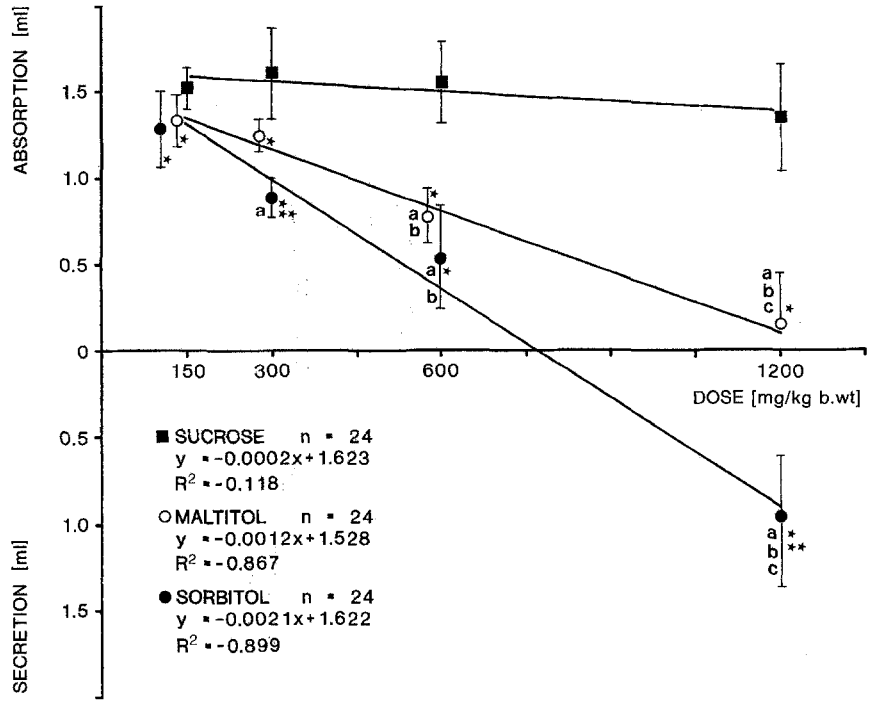

Figure 6. Net water movement in the upper gastrointestinal tract of rats 1 h after intragastric administration of different doses of sugar or sugar substitutes. Significant differences are indicated as in figure 3. *marks significant differences versus sucrose and ** versus maltitol experiments. Statistical comparison of the slopes of the regression lines by analysis of covariance revealed significant differences ($p < 0.05$) between the three series.

Discussion

Mathematical models of the complex interactions occurring during the digestive process aim to evaluate physiological concepts and to facilitate understanding of the system[35]. In an earlier investigation[6], the gastrointestinal transit in rats was characterized by calculating the percentage of a marker which had left an intestinal segment in relation to the total amount which had entered it. However, this method does not seem to be so well suited to revealing the dynamics of intestinal propulsion. As proposed more recently for ruminants[7,34], we considered the digestive flow as a multicompartmental process which can be approximated by a series of exponential components, i.e. the filling and evacuation of the different compartments in question. Based on either the defined model for the marker distribution or on the experimental data, the transport constants were estimated. This involves fitting the model to the experimental results. As shown in figure 2, the observed values and predicted curves correlated well. Such a fitting provides an opportunity to use only a few of the experimental results to calculate the time until onset of filling of a compartment (as done for the cecum, table) or to estimate the time necessary for the complete evacuation of parts of the small intestine (fig. 2).

Although limitations to the use of phenol red as a 'non-absorbable' marker in digestion studies have been discussed[6,26], previous work had proved its usefulness in following the gastrointestinal transit and in determining the site of absorption of a test substance in the rat[13,25]. With both doses of maltitol (compared in fig. 2) only slight differences concerning the exponential course of stomach evacuation were revealed. Despite this, the kinetics of intestinal transit differed clearly (table and fig. 2). This ties in with results on human subjects indicating that small bowel transit and gastric emptying may each be controlled independently[9]. Indeed, small intestinal transit was shown to be dependent on the ratio of intestinal volume and flow rate[2].

In our experiments with high maltitol doses and at 0.5 h after gavage, the volume in the first half of the rats' small intestines had increased dramatically by up to 4-fold compared with the low-dosed series or to the blanks (data not shown). This enormous increase in luminal content due to water influx reflected the osmotic activity of the high dose of maltitol and of its cleavage product sorbitol[14]; the latter was readily liberated but poorly absorbed (compare fig. 3D). Nevertheless, after 1 h, the data on net water movement indicated little reabsorption ('1200 mg/kg b.wt' in fig. 6).

Obviously, the volume increase was not compensated for by an adequate acceleration of the luminal flow rate. Consequently, the transit to the next compartment was somewhat delayed with the large dose of maltitol (k_2 in table and 'first half' in fig. 2). However, in the distal parts of the small gut (corresponding essentially to the ileum[36]) the flow rate was apparently enhanced as compared to the tests with the low doses or to the blank series (table and fig. 2).

Moreover, with the onset of water reabsorption (fig. 6), a reduction in the volume of the intestinal content may be expected. This would have the potential to diminish the intestinal transit time since the volume/flow relationship is one of its determinants[2]. The drastic acceleration of transport in the lower small intestine led to a reduction of the total gastrocecal transit time (table and fig. 2).

One may conclude that with disaccharide sugar alcohols, effective cleavage in the small intestine and the absorption of liberated sugar (as it occurs with low doses of maltitol, fig. 3 A and B) of a large osmotically active load may be impeded by two main mechanisms: a) in the upper part of the small intestine, dilution in a larger volume would limit the contact of the substance with the digestive surface and b) as an additional step, digestibility in the lower parts of the small intestine may be reduced by an enormous acceleration of luminal flow with subsequent prevention of the normal ileal stasis[6,9]; a certain fraction of uncleaved material and the barely absorbable cleavage products (hexitols) would escape small bowel digestion and enter the cecum. In a previous study by other authors, evidence for a combination of these effects leading to malabsorption in the small intestine was also demonstrated with humans[37].

As indicated by the gastrocecal transit times in the table, the carbohydrate recovery data on the 1-h time point were suitable for judging the digestive capacity of the small intestine alone. However, 2 h after ingestion of the test substances some marker had entered the cecal compartment (fig. 2), hindering clear discrimination between small gut digestion and cecal fermentation. After 1 h the marker peak occurred in the third quarter of the small intestine under all experimental conditions (fig. 2); the peak had thus passed the jejunum where the disaccharidase activities for maltitol and sucrose are highest[38]. A similar distribution was also observed in rats, 1 h after feeding a fluid-associated chromium chloride marker with the diet[39].

Unfortunately, for this time point it was almost impossible for us to detect the presence of absorbed sugar or sugar substitute in the portal vein, especially when the low doses were given. It is likely that the detection limits of the analytical methods were exceeded; portal blood flow occurs at a high rate (about 8 ml/min in the rat[40]), and only minor changes in the portal venous carbohydrate concentration may be expected under these conditions. Moreover, with sucrose, the peak of glucose entry in the portal blood was shown to occur only 15 min after gavage in rats[14]. Thus, calculations on the small intestinal digestibility of the substances investigated could only be done by measuring their disappearance from the luminal side of the gut. Nevertheless, 1 h after administration of the highest dose of maltitol or sucrose (1200 mg/kg b.wt) to the animals, we observed a significant increase in portal blood glucose, indicating 30% more than the controls (6.4 $\pm$ 1.4 mM) for maltitol and 40% for sucrose (unpublished results).

The caloric availability of maltitol is underestimated when extrapolating experimental results obtained with non-physiological high doses to the level of realistic intake (see above). In an earlier paper[13], we demonstrated this effect in a even more pronounced form with a mixture of disaccharide sugar alcohols, Palatinit®.

However, one has also to consider the possibility that the values for digestible energy presented here are underestimated when the substance undergoes exclusive small intestinal digestion for more than 1 h. In fact, after a 2-h digestion time and an initial application of 150 mg/kg b.wt maltitol, in three out of six animals tested no marker had entered the ceca. In these rats, neither maltitol nor glucose were detected in the intestinal contents and on average only 65% of the sorbitol part was recovered. In this case, one can assume an increased small intestinal availability of maltitol.

Moreover, as indicated by the marker (fig. 2), the passage from the small intestine to the cecum was not a sudden event. Even 3 h after maltitol administration, the emptying of the distal small gut was not completed (fig. 2) and the possibility of continuous small intestinal absorption of maltitol residues must be taken into account. Therefore, the present calculations on the small intestinal caloric availability of maltitol must be considered as a minimum estimate.

Moreover, a certain fraction of the energy content of residual amounts of the substance or cleavage products which escape the small intestine and enter the cecum would be available from fermentation products after microbial degradation[41,42] and increase even more the total physiological availability of maltitol.

Recent results obtained with humans using a perfusion technique and postprandial administration of the substance, indicated a total physiological caloric value of 3.5 kcal/g maltitol, mainly digested in the small intestine[43].

The results on sucrose digestion were supported by the data on intestinal net water movement, indicating efficient absorption of all dosages tested (fig. 6). In previous experiments with rats, even 3000 mg sucrose/kg b.wt was shown to correlate with the absorption of water after a 1-h digestion time[14]. Absorption of fructose in the upper gut occurs at a slower rate than glucose[44,45]. Thus, after increased administration of sucrose, a substantial part of its fructose moiety can be expected to be malabsorbed and enter the cecum[46,47]. Changes in metabolism of cecal flora, and species selection seem to occur after high-dose sucrose adaptation[48,49] would therefore mainly reflect the influence of the fructose part of sucrose.

The digestion of sorbitol lacked dose dependence although the physiological response, in the form of intestinal water movement, strongly correlated with the dose administered (figs 5 and 6). This finding is in agreement with the time-dependent absorption of equal fractions of sorbitol within a broad range of concentrations (20–200 mM), shown using a duodenojejunal loop technique

in rats [50]. In our test animals, the intraluminal concentrations of sorbitol in the different small intestinal compartments investigated were of this order of magnitude (data not shown). Substantially lower intraluminal concentrations (0.001–2 mM) were reported to be absorbed to a higher degree [50].

With sorbitol, as indicated for maltitol (fig. 2), small intestinal emptying was not completed even 3 h after administration. Therefore, as discussed above, the calculation on small intestinal availability must be regarded as a minimum estimate, too.

In conclusion, the calorie-saving effect of the new sugar substitute maltitol must be considered as minor. Ingested in a realistically low amount, its capacity to reduce energy availability compared to sucrose may lie in the slow small intestinal absorption rate of its cleavage product sorbitol; this would partially escape the small intestine and undergo, with a certain energy loss, fermentation in the large gut. The suggestion that a postulated incompetence of the small intestinal disaccharidases to digest maltitol is the main reason for a reduced physiologic caloric yield cannot be supported by the results from our study.

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