Zeitschrift für
Ernährungswissenschaft Z. Ernährungswiss. 22, 185–194 (1983)

Bioavailability of glucose from Palatinit®[1])

S. C. Ziesenitz

Division of Experimental Dentistry, School of Dentistry, University of Würzburg

Summary

For the sake of metabolic insight into the fate of the sugar substitute Palatinit®, its two components D-glucosyl-α(1→1)-D-mannitol and D-glucosyl-α(1→6)-D-glucitol [D-glucosyl-α-(1→6)-D-sorbitol] were assayed for glucose bioavailability by the procedure of Karimzadegan et al. using ketotic rats. With conversion rates into glucose of 6 and 20 %, respectively, for free mannitol and glucitol (sorbitol), 39 % for glucosylmannitol and 42 % for glucosylglucitol, the metabolic glucose pool of the rat does not receive the full carbohydrate complement of these compounds. The preformed glucose moiety of the glucosylhexitols is bioavailable by 36 and 32 %, respectively, from glucosylmannitol and glucosylglucitol, with 50 % as theoretical maximum.

Less than theoretical bioavailability of glucose from Palatinit® is ascribed to microbial attack in the hindgut. The data on rats are held valid also for other species demonstrating carbohydrate fermentation in the caecum and/or colon. Differences between D-glucosyl-α(1→1)-D-mannitol and D-glucosyl-α(1→6)-D-glucitol are caused by a differential delay of glucose absorption in the small intestine, also exerted by D-glucitol.

The deep metabolic insight offered by the glucose bioavailability assay into the fate of carbohydrates includes the mammal-microbial symbiosis in the large bowel. Since a rather complete survey of the metabolic consequences after their intake can be obtained, the assay system should be generally applied in assessments of food safety also of other sugar substitutes.

Zusammenfassung

Zur Vertiefung des Verständnisses vom Stoffwechsel des Zuckeraustauschstoffes Palatinit® wurden seine zwei Bestandteile D-Glucosyl-α(1→1)-D-mannit und D-Glucosyl-α(1→6)-D-glucit [D-Glucosyl-α(1→6)-D-sorbit] nach dem Verfahren von Karimzadegan et al. auf ihre Glucose-Bioverfügbarkeit an ketotischen Ratten untersucht. Bei Umwandlungsraten in Glucose von 6 bzw. 20 % für Mannit und Glucit (Sorbit) sowie von 39 bzw. 42 % für Glucosylmannit und Glucosylglucit erhält demnach der metabolische Glucose-Pool nicht das volle Glucose-Äquivalent aus diesen Verbindungen. Von dem Anteil an präformierter Glucose in den Glucosylhexiten – theoretisches Maximum 50 % – sind nur 36 % aus Glucosylmannit bzw. 32 % aus Glucosylglucit bioverfügbar.

Die im Vergleich zur Theorie verminderte Bioverfügbarkeit von Glucose aus Palatinit® wird auf partiellen mikrobiellen Abbau in unteren Darmabschnitten zurückgeführt. Die an Ratten erhaltenen Ergebnisse werden auch für alle anderen Spezies gelten, welche in Caecum und/oder Colon Kohlenhydrate vergären. Die

[1]) Palatinit® is a registered trademark by Süddeutsche Zucker-A.G., Mannheim; generic name: Isomalt

Unterschiede zwischen D-Glucosyl-α(1→1)-D-mannit und D-Glucosyl-α(1→6)-D-glucit werden durch unterschiedliche Verzögerung der Glucoseresorption im Dünndarm, wo auch D-Glucit angreift, bedingt.

Die Ermittlung der Glucose-Bioverfügbarkeit gewährt weitgehende Einblicke in das Schicksal von Kohlenhydraten einschließlich der Symbiose zwischen Säugetier und Mikroorganismen im Dickdarm. Da ein ziemlich vollständiger Überblick über die metabolischen Konsequenzen nach ihrer Zufuhr erhalten wird, sollte das Verfahren zur Messung der Bioverfügbarkeit von Glucose daher bei Abschätzungen der Lebensmittelsicherheit anderer Zuckeraustauschstoffe ebenfalls angewandt werden.

Key words: sugar substitutes, D-glucose, bioavailability, D-glucitol (D-sorbitol), D-mannitol, Palatinit®, D-glucosyl-α(1→1)-D-mannitol, D-glucosyl-α(1→6)-D-glucitol

Introduction

Assay procedures for sugar substitutes (nutritive sweeteners) include screening methods according to the intended use pattern, e.g. of non-cariogenicity, as well as a general survey of their metabolic behaviour for an assessment of food safety.

Sugar substitutes whose structure is derived from common dietary carbohydrates will in most cases enter intermediary metabolism via carbohydrate pathways. Since glucose degradation, mostly by glycolysis, constitutes a major catabolic route in the body, contributions by smaller amounts of nutritive sweeteners of carbohydrate nature are usually difficult to recognize within total body carbohydrate turnover.

Yet, an important criterium of the metabolic fate of sugar substitutes is held to be the amount of glucose bioavailable from such compounds in mammals. The magnitude of glucose supply by nutritive sweeteners for the carbohydrate pool may be experimentally determined if a perturbation of metabolism toward ketogenesis is established, and carbohydrates thus constitute the sole growth-limiting nutrient (1).

In view of the rather complex nature of bioavailability assays, Palatinit® was chosen as model of sugar substitutes, consisting of equimolar amounts of D-glucosyl-α(1→1)-D-mannitol and D-glucosyl-α(1→6)-D-glucitol, respectively[2]). We wish to demonstrate in this paper the kind of information obtainable by an investigation of glucose bioavailabilities from these two D-glucosylhexitols.

Materials and methods

Animals. Weanling male Han: SPF-Sprague-Dawley rats of 3 weeks of age (Zentralinstitut für Versuchstiere, Hannover) were kept individually in plastic cages ("Macrolon") and supplied with tapwater and test diet ad libitum. The animals were

[2]) The formerly used designations (9) α-D-glucopyranosido-1,6-mannitol and α-D-glucopyranosido-1,6-sorbitol have been replaced in this paper, according to correct nomenclature, by D-glucosyl-α(1→1)-D-mannitol and D-glucosyl-α(1→6)-D-glucitol, respectively (cf. Lichtenthaler (2, 3)).

alloted to the experimental groups under strict randomization. The rats were housed for the test periods of 28 days under standardized conditions (22 ± 2 °C room temperature, 40–60 % relative humidity, 12/12 hours light/dark intervals) and were weighed daily.

Table 1. Comparison of the effects of glucose, hexitols and glucosylhexitols on the weight increase of ketotic rats (Mean values ± standard error of the mean for n rats).

Differences within and between subgroups are highly significant, unless listed below:
Glucosylmannitol series:
significant: H → A, I → A, G → B, E → G;
weakly significant: G → C, D → G;
not significant: I → H, D → B, D → F, E → C, F → B.

Glucosylglucitol series:
significant: S → K, O → M, P → O;
weakly significant: Q → M, S → N;
not significant: R → K, R → S, P → N, Q → O, P → L, N → L.

At the 2% as well as at the 4% level of added glucosylhexitols, the difference between D-glucosyl-α(1 → 1)-D-mannitol and D-glucosyl-α(1 → 6)-D-glucitol is weakly significant ($0.0135 \leqq p \leqq 0.05$ at 2%, and $0.0264 \leqq p \leqq 0.05$ at 4% glucosylhexitol, respectively).

Carbohydrate	% in diet	group	n	28-day weight gain (g)
Initial weight		52.8 ± 0.8	71	
Glucose	0	A	8	43.1 ± 3.6
	1	B	7	78.4 ± 4.5
	2	C	8	118.6 ± 4.8
Glucose + mannitol	1 + 1	D	8	87.0 ± 6.2
	2 + 2	E	8	121.1 ± 3.8
D-glucosyl-α(1 → 1)-	2	F	8	77.2 ± 3.7
D-mannitol	4	G	8	102.6 ± 5.2
Mannitol	3	H	8	55.7 ± 2.6
	6	I	8	56.0 ± 2.5
Initial weight		46.2 ± 0.5	89	
Glucose	0	K	10	46.0 ± 2.5
	1	L	10	91.7 ± 3.3
	2	M	10	126.7 ± 4.8
Glucose + glucitol	1 + 1	N	9	80.5 ± 6.0
	2 + 2	O	10	109.8 ± 3.3
D-glucosyl-α(1 → 6)-	2	P	10	92.3 ± 4.7
D-glucitol	4	Q	10	115.7 ± 3.8
Glucitol	1	R	10	54.0 ± 4.3
	2	S	10	64.2 ± 5.5

Diets. The basal diet, free of absorbable carbohydrates (1), consisted of 35 % oleic acid, 7.5 % soybean oil, 13.3 % casein (corresponding to 12 % protein), 1 % sodium hydrogencarbonate, 4.55 % mineral mixture, 1 % vitamin mixture, 0.02 % antioxidants (each 0.01 % of butylated hydroxytoluene and Ethoxyquin) and 37.13 % cellulose.

The experimental diets were designed to be isocaloric as well as isonitrogenous. They contained, for standardization, 0 or 1.11 % (w/w) D-glucose monohydrate (Merck, Darmstadt) equivalent to 1.0 % D-glucose, or 2.22 % (w/w) D-glucose monohydrate corresponding to 2.0 % D-glucose, which were exchanged in the basal diet by equal weights of cellulose. D-Glucosyl-α(1→1)-D-mannitol and D-glucosyl-α(1→6)-D-glucitol, as substances under assay (obtained from Dr. Schiweck, Obrigheim), as well as D-mannitol and D-glucitol (Merck, Darmstadt) and their mixtures with D-glucose were – in regard of their respective moisture contents – added to the basal diet in exchange for equal weights of cellulose.

The following experimental groups were set up: for calibration (3 groups; fig. 1) 0, 1.0 and 2.0 % glucose; for the assay of glucosylmannitol (6 groups), 3.0 and 6.0 % mannitol, 2.0 and 4.0 % D-glucosyl-α(1→1)-D-mannitol, as well as 1.0 % glucose plus 1.0 % mannitol and 2.0 % glucose plus 2.0 % mannitol; for the assay of glucosylglucitol (6 groups), 1.0 and 2.0 % glucitol, 2 % and 4 % D-glucosyl-α(1→6)-D-glucitol, as well as 1 % glucose plus 1 % glucitol and 2 % glucose plus 2 % glucitol. The theoretical metabolizable energy of the 9 diets comprising each experiment was calculated to be 18.4 MJ/kg ± 0.9 %.

Due to the high content of oleic acid and other polyunsaturated lipids in the rations, basal and experimental diets were obtained (Ssniff Co., Soest/Westfalen) as separate "oil" and "flour" premixes which were (as needed, usually once a week) finally mixed mechanically in 1-kg batches. 5 grams each of every batch of the final diets were, at the end of the experiment, combined and assayed for their combustion heats (Ssniff Co., Soest/Westfalen) and found to contain 25.8 MJ/kg ± 0.4 % in the 9 diets used in the glucosylmannitol assay, and 25.8 MJ/kg ± 0.1 % in the 9 diets for the glucosylglucitol assay; thus, there was no statistically significant difference between the combustion heats of the diets used in the two series of feeding experiments (see also table 1).

Analytical procedures. The calculated concentrations in experimental diets of added carbohydrates (see above) were confirmed analytically in the "flour" premixes (see above). They were extracted with methanol or iso-propanol (4); the extracts were cleared (5), separated from the precipitate by centrifugation and assayed for glucose according to Luff-Schoorl (5), for sorbitol enzymatically (6), and for D-glucosyl-α(1→1)-D-mannitol, D-glucosyl-α(1→6)-D-glucitol, and glucose with anthrone (7).

Statistical analysis. The data were treated statistically using the analysis of variance and the method of linear regression. The relative bioavailability of glucose was calculated according to the "common-zero, 5-point-slope-ratio assay" (8) from the weight gain in 28 days of the following five groups: two glucose calibration groups, two groups with the substance under assay, and the group on the basal diet, devoid of any absorbable carbohydrate.

Results

The calibration of the assay system with glucose as sole growth-limiting nutrient of the diet is shown in figure 1. The weight increase of ketotic rats is strictly proportional to the glucose concentration of the respective diet.

The growth rate of ketotic rats in all experimental groups was strictly linear during the 28-day test periods. Correlation coefficients of growth versus time were in all groups near r = 0.988 to 0.999. The final weight gains

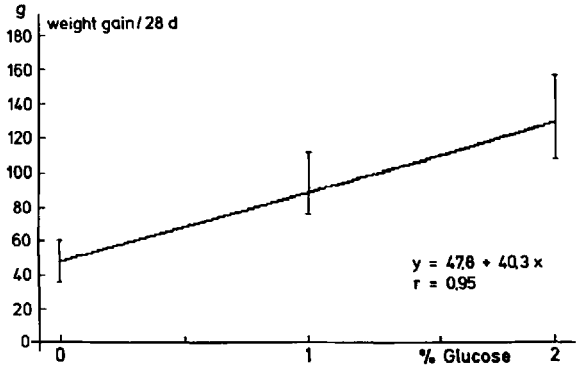

Fig. 1. Calibration of the bioavailability assay of glucose (numbers of animals as in table 1, groups "K to M"). Not shown are data for groups "A to C" (cf. table 1) with $y = 42.3 \pm 37.8\ x$, $r = 0.94$.

are shown in table 1. From these data, mean values and 95 % fiducial limits of the bioavailabilities of glucose were calculated following the "common-zero, 5-point-slope-ratio assay" (8) and are given in figure 2.

Only a very small amount of glucose (6 %) is available from D-mannitol for the rat. From a 1:1 mixture of D-glucose plus D-mannitol, 54 % become bioavailable as glucose, thus indicating an additive situation regardless if the single components or their mixtures are fed. If these two components are fed as D-glucosyl-α(1→1)-D-mannitol, however, they are significantly less efficient (39 %) than their mixture (54 %). Subtracting the contribution by D-mannitol, preformed glucose is bioavailable only by 36 % (95 %

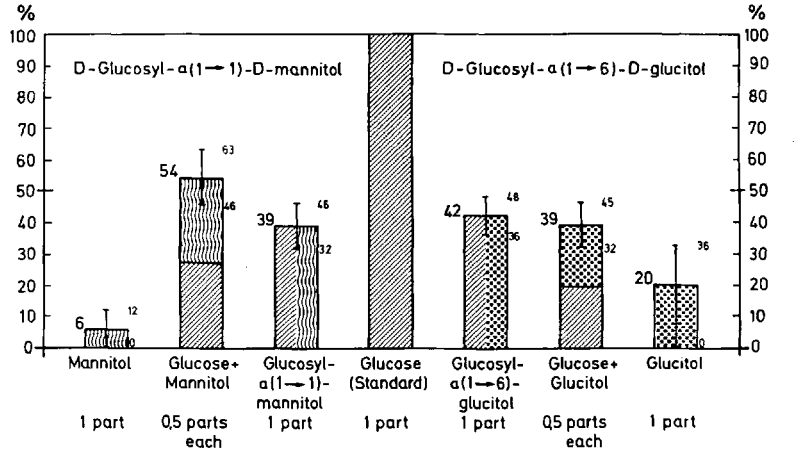

Fig. 2. Relative bioavailability of glucose from two hexitols and glucosylhexitols. The height of the columns derives from theoretical 100 % for one part (e.g. of glucose) and, respectively, from theoretical 50 % for those diet components which consist by one half of glucose (e.g. glucose plus hexitols and glucosyl-hexitols).

fiducial limits in the range of 33–39 %); thus, only 72 % of the glucose bound in D-glucosyl-α(1→1)-D-mannitol is made available as such in the rat.

The hexitol D-glucitol yields, in contrast to D-mannitol, 20 % as glucose. Glucose bioavailability from D-glucosyl-α(1→6)-D-glucitol amounts to 42 %, seemingly resembling the 39 % reported above for D-glucosyl-α(1→1)-D-mannitol. When the contribution by D-glucitol is subtracted, however, preformed glucose in this glucosylglucitol is bioavailable only at 32 %, thus demonstrating more than one third "loss" (64 % of the theoretical value) for glucose, linked to D-glucitol by an α(1→6)-bond.

Surprisingly enough, glucose plus glucitol mixtures 1:1 demonstrate a bioavailability of glucose of 39 %, whereas the theoretical value would be 60 % if additive behaviour were to be expected as in the case of the glucose plus mannitol mixtures. The "loss" of one third of the calculated value, though no glycoside bonds need to be cleaved, may be caused, in intact animals, by interference of the absorption of D-glucose with either D-glucitol or Palatinit®, the mixture of the two D-glucosylhexitols which then was studied[3]) in narcotized, intestine-perfused animals.

The active absorption of D-glucose in the small intestine of SPF--Sprague-Dawley rats was investigated with the in-situ perfusion technique of Förster and Menzel (9) in the concentration range of 0 to 50 mM glucose. As shown in figure 3, D-glucitol and Palatinit® inhibit, under the

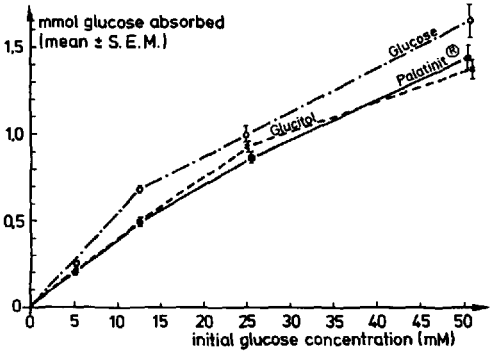

Fig. 3. Influence on glucose absorption in the small intestine by Palatinit® and by D-glucitol (according to Förster*)).
Significance: Control versus Palatinit® and glucitol highly significant at 5 and 12.6 mM glucose; weakly significant at 25.2 mM glucose with Palatinit® and at 50.5 mM glucose with glucitol; not significant at 25.2 mM glucose with glucitol and at 50.5 mM glucose with Palatinit®.

*) Prof. Dr. H. Förster (Frankfurt) kindly provided the data on the absorption of glucose as advance information; a detailed publication of these investigations is planned for the near future.

[3]) Prof. Dr. H. Förster (Frankfurt) kindly provided the data on the absorption of glucose as advance information; a detailed publication of these investigations is planned for the near future.

experimental conditions chosen, the active absorption of D-glucose in the small intestine of rats. At most of the values taken, the inhibition was highly significantly different from polyol-free controls; formally, the inhibition demonstrated a competitive character. The extent of inhibition was about the same at 275 mM D-glucitol and 207 mM Palatinit®, respectively.

Discussion

The experiments have demonstrated that the preformed glucose from both components of Palatinit® enters mammalian metabolism to an incomplete extent only, the remainder being used in different pathways.

The calculation of the data of figure 2 arrives for Palatinit® (50 parts of glucose and 50 parts of hexitols) at a bioavailability of glucose of 40 %, for D-glucosyl-α(1→1)-D-mannitol at 39 %, and for D-glucosyl-α(1→6)-D-glucitol at 42 %, respectively. These values include the conversion rates of the two hexitols, 6 % for Palatinit®, 3 % for D-mannitol, and 10 % for D-glucitol, respectively. Since one half of the glucosylhexitols is represented by preformed glucose, its bioavailability is found at 34 % for Palatinit®, 36 % for D-glucosyl-α(1→1)-D-mannitol, and 32 % for D-glucosyl-α(1→6)-D-glucitol, respectively. In the average, one third of preformed glucose escapes the carbohydrate pool of the ketotic rat. The use of a "5-point-slope-ratio assay" (as mentioned under Methods) leads to practically the same result.

Energetically, D-glucosyl-α(1→1)-D-mannitol and D-glucosyl-α(1→6)-D-glucitol have been found in a growth experiment with rats to be of equal value (10), but this aspect should not be stressed here since an assay of glucose bioavailability centers around the metabolic fate of a compound and is, if at all, but indirectly connected with caloric aspects. Furthermore, it has been repeatedly described (10, 11) that Palatinit® is not excreted in urine or feces at any appreciable rate.

It has been shown in experiments with rats (10–12) and man (13) that microorganisms of colonic and/or caecal origin participate in vivo in the degradation of Palatinit®. We explain, on this basis, the diminished glucose bioavailabilities observed in the present experiments as being caused by microbial fermentations of part of the test substances in the hindgut, because man and rat do not excrete, after a short period of adaption, any sizable amount of glucosylhexitols in feces or urine (10, 12, and footnote[4]). In the case of D-glucitol, partial microbial degradation has been demonstrated earlier (14) in man and with rats. It is assumed that different microbial patterns prevail in the large bowel and are responsible for the fact that the glucose bioavailability from D-glucitol has been found to be higher (1) in Davis (California) than we could demonstrate (fig. 2) in the present experiment.

Microbial fermentation products of Palatinit® are essentially volatile fatty acids, as already shown for many other large-intestinal microbial fermentations of carbohydrates (15–19). In consequence, a fraction of

[4]) S. C. Ziesenitz, C. Benning, E. J. Karle, R. Vallon, G. Siebert, unpublished data.

ingested carbohydrate enters mammalian metabolism as volatile fatty acids; this fact explains why the diminished bioavailability of glucose (this paper) cannot be directly related to the reduced caloric utilization of Palatinit® shown in a number of experiments (10–12).

The inhibition of glucose absorption by Palatinit® (fig. 3) leads to the question which of the two components of this sugar substitute might be responsible for the effect. The data of the present paper speak against glucosylmannitol, whereas the results of figure 2 are an argument for glucosylglucitol; in addition, the glucitol moiety of D-glucosyl-α(1→6)-D-glucitol adopts (3), like D-glucitol itself (20), a non-linear, bent chain conformation.

Besides these conformational aspects of glucosylhexitols, their route of administration deserves a comment: If perfused small intestines of anesthetized animals are compared with the feeding experiments of this paper, the data on glucose plus glucitol (fig. 2 and table 1) point toward a practical relevance of the effect of glucitol on glucose absorption; according to figure 3, we have to assume significance also for the inhibition of glucose absorption by D-glucosyl-α(1→6)-D-glucitol, especially under the condition of low glucose levels in small intestinal contents, as they prevail under Palatinit® feeding of the present experiments.

Very low concentrations of glucose are reported for the contents of different anatomical parts of the gastrointestinal tract of rats (10)[5]), whereas glucosylhexitols and free hexitols are found at substantial concentrations. Accordingly, there is even in the presence of glucosylglucitol and/or glucitol sufficient capacity to absorb glucose effectively and thus to allow the formation of very low ratios of glucose:glucosylglucitol and glucose:glucitol. The experiments mentioned above (10) were performed on rats with at least 34.5 % corn starch, besides Palatinit®, in the diet; therefore, a large amount of bound glucose was present in the gut, while both D-glucosyl-α(1→1)-D-mannitol and D-glucosyl-α(1→6)-D-glucitol could exert their kinetically differentiated inhibitory effects on the cleavage of maltose (10). In the present experiments, starch was not included in the diet. The observed inhibition of glucose absorption (fig. 3) can therefore be interpreted as a delay of the absorption process. Under such circumstances, more carbohydrates will be accessable for microbial degradation in the large intestine; we thus explain the non-additive behaviour of glucose plus glucitol mixtures (fig. 2) and the diminished bioavailability of preformed glucose from D-glucosyl-α(1→6)-D-glucitol in comparison with D-glucosyl-α(1→1)-D-mannitol.

Hexitols may be of relevance for glucose metabolism, as shown in the present experiments for free hexitols as well as for certain types of glucosidic bonds between glucose and hexitols; the contributions of small-intestinal processes (carbohydrates; glucose absorption) and of the large-intestinal symbiosis between mammals and microorganisms serve as explanations for the observations reported in this paper. The comprehensive and deep insight, thus offered by an investigation of glucose bioavailability, into the metabolic fate of Palatinit® becomes evident from the above considerations. It would be highly desirable – if not mandatory –

[5]) S. C. Ziesenitz, C. Benning, E. J. Karle, R. Vallon, G. Siebert, unpublished data.

to extend such studies also to other promising sugar substitutes as one of the criteria of food safety assessments. The observations of this paper on the rat are in principle applicable to all other species which demonstrate microbial attack on carbohydrates in the hindgut.

Acknowledgements

The author is grateful to Dr. H. Schiweck for supplying Palatinit® and to Dr. I. Haubitz for advice with the slope ratio assay. The technical assistance of Miss A. Heidloff is gratefully acknowledged. My sincere thanks are due to Prof. G. Siebert for advice and discussions.

References

1. Karimzadegan, E., A. J. Clifford, F. W. Hill: A Rat Bioassay for Measuring the Comparative Availability of Carbohydrates and Its Application to Legume Foods, Pure Carbohydrates and Polyols. J. Nutr. **109,** 2247–2259 (1979).
2. Lindner, H. J., F. W. Lichtenthaler: Extended Zigzag Conformation of 1-0-D-α-glucopyranosyl-D-mannitol. Carbohydr. Res. **93,** 135–140 (1981).
3. Lichtenthaler, F. W., H. J. Lindner: The Preferred Conformations of Glucosylalditols. Liebigs Ann. Chem. 2372–2383 (1981).
4. Official Methods of Analysis, 10th ed., Assoc. Offic. Agr. Chemists, Washington, D.C., 1965, Sections 6.002 & 6.074.
5. Acker, L.: Nachweis und Bestimmung der Mono- und Oligosaccharide. pp. 349, in: Handbuch der Lebensmittelchemie, Hrsg.: J. Schormüller, Band II/2. Teil, Springer Verlag (Berlin 1967).
6. Bergmeyer, H. U., W. Gruber, J. Gutmann: D-Sorbit, in: Methoden der Enzymatischen Analyse (Hrsg.: H. U. Bergmeyer), 2. Aufl., S. 1292–1295, Verlag Chemie (Weinheim/Bergstr. 1970).
7. Roe, J. H.: The Determination of Sugar in Blood and Spinal Fluid with Anthrone Reagent. J. Biol. Chem. **212,** 335–343 (1955).
8. Finney, D. J.: Statistical Method in Biological Assay. Third Edition. pp. 159–162, Charles Griffin & Co. Ltd. (London and High Wycombe 1978).
9. Förster, H., B. Menzel: Untersuchung der intestinalen Glucoseresorption bei künstlich erhöhter Blutglucosekonzentration. Z. Ernährungswiss. **11,** 10–23 (1972).
10. Grupp, U., G. Siebert: Metabolism of Hydrogenated Palatinose, an Equimolar Mixture of α-D-Glucopyranosido-1,6-sorbitol and α-D-Glucopyranosido-1,6-mannitol. Res. Exp. Med. (Berl.) **173,** 261–278 (1978).
11. Zinner, P. M., M. Kirchgeßner: Zur energetischen Verwertung von Palatinit®. Z. Ernährungswiss. **21,** 272–278 (1982).
12. Kirchgeßner, M., P. M. Zinner, H. P. Roth: Energiestoffwechsel und Insulinaktivität bei Ratten nach Palatinitfütterung. Internat. J. Vit. Nutr. Res. **53,** 86–93 (1983).
13. Siebert, G., R. Vallon: Nutzung von Nahrungskohlenhydraten durch Darmbakterien. Dtsch. zahnärztl. Z. **37,** S42–S43 (1982).
14. Schnell-Dompert, E., G. Siebert: Metabolism of Sorbitol in the Intact Organism. Hoppe-Seyler's Z. Physiol. Chem. **361,** 1069–1075 (1980).
15. Cummings, J. H.: Short Chain Fatty Acids in the Human Colon. Gut **22,** 763–779 (1981).
16. Saunders, D. R., H. S. Wiggins: Conservation of Mannitol, Lactulose, and Raffinose by the Human Colon. Amer. J. Physiol. **241** (Gastrointest. Liver Physiol. 4). G 397–G 402 (1981).

17. Soergel, K. H.: Absorption of Fermentation Products from the Colon. Colon and Nutrition (Falk Symposium: 32), edited by H. Kasper and H. Goebell, pp. 27–34, MTP Press Limited, Falcon House, Lancaster (England 1982).
18. Roediger, W. E. W.: Role of Anaerobic Bacteria in the Metabolic Welfare of the Colonic Mucosa in Man. Gut **21**, 793–798 (1980).
19. Roediger, W. E. W.: Utilization of Nutrients by Isolated Epithelial Cells of the Rat Colon. Gastroenterology **83**, 424–429 (1982).
20. Jeffrey, G. A., H. S. Kim: Conformations of the Alditols. Carbohydr. Res. **14**, 207–216 (1970).

Received July 8, 1983

Author's address:

Dr. Susanne C. Ziesenitz, Division of Experimental Dentistry, School of Dentistry, University of Würzburg, Pleicherwall 2, D-8700 Würzburg