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INTESTINAL DIGESTION OF MALTOTRIOSE IN MAN

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SUMMARY

The digestion of maltotriose by human duodenal juice and small intestinal mucosa has been investigated.

Maltotriose represented approx. 25% of the end products of the action of duodenal juice on starch *in vitro*. It was largely resistant to the action of the juice but was readily hydrolysed to glucose by homogenates of small intestinal mucosa.

The rate of hydrolysis of maltotriose by intestinal mucosa was of the same order as that of the disaccharides maltose, isomaltose, sucrose, lactose and trehalose. The Michaelis constant for maltotriose was similar to that for maltose.

Mixed substrate incubation experiments suggested that the intestinal maltotriase activity is at least partially due to enzymes which also act on maltose, isomaltose and sucrose.

By means of heat inactivation, the intestinal maltotriase activity could be separated into three distinct fractions, all of which had maltase and isomaltase activity. The pattern of heat inactivation suggested that the maltotriase activity is chiefly exerted by the α-glucosidases designated (DAHLQVIST[11]) maltases Ia, Ib and III.

Maltotriase activity was considerably below normal in duodenal mucosal specimens obtained from patients with congenital absence of maltases Ia and Ib. This provides further evidence that these enzymes are involved in the hydrolysis of maltotriose.

INTRODUCTION

Maltotriose is a trisaccharide formed during the action of α-amylase (EC 3.2.1.1) on starch or glycogen[1]. Although it constitutes a large portion of the end products of the α-amylolysis of starch[2], little is known regarding its gastrointestinal hydrolysis to glucose. RUTLOFF, FRIESE AND TÄUFEL[3,4] recently showed that maltotriose is largely resistant to the action of purified pig pancreatic α-amylase, but is readily hydrolysed by pig and rat intestinal mucosa.

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The object of the present investigation was to obtain information about the digestion of maltotriose in the human intestine, in particular about the identity of the enzyme or enzymes responsible for its hydrolysis.

MATERIALS AND METHODS

Materials

Maltotriose (*O*-α-D-glucopyranosyl-(1→4)-*O*-α-D-glucopyranosyl-(1→4)-D-glucose) was prepared essentially by the method of WHELAN, BAILEY AND ROBERTS[5], which consists of partial acid hydrolysis of potato amylose, followed by isolation of the maltotriose contained in the hydrolysate by elution from a charcoal column. The main modification was that introduced by THOMPSON AND WOLFROM[6], *viz.* the use of granular charcoal ("Nuchar C Unground", a product of the West Virginia Pulp and Paper Co., Chicago, Ill., U.S.A.) instead of a mixture of finely ground charcoal and Celite. The maltotriose obtained after the first elution was found, by means of paper chromatography, to be contaminated with traces of maltose and maltotetraose; these were removed by replacing the maltotriose on the column and repeating the elution.

Isomaltose (6-*O*-α-D-glucopyranosyl-D-glucose) was kindly supplied by Dr. R. WEIDENHAGEN, Neuoffstein, Germany.

Isomaltotriose (*O*-α-D-glucopyranosyl-(1→6)-*O*-α-D-glucopyranosyl-(1→6)-D-glucose was a gift from Dr. A. DAHLQVIST, Lund, Sweden.

Intestinal mucosa

Specimens of duodenal mucosa were obtained from children or infants by peroral biopsy with the paediatric CROSBY capsule[7]. They were homogenised in 0.9% NaCl to give a final concentration of 2% (wet wt./vol.). Ileal mucosa was obtained from a macroscopically normal piece of lower ileum resected during surgical operation. The mucosa was scraped off with a glass slide and homogenised in 10 mM sodium maleate buffer (pH 6.0) to give a final concentration of 10% (wet wt./vol.). Homogenates were stored at −20° with little loss of activity over several weeks.

Assay of intestinal oligosaccharidase activity

Maltotriase, maltase, isomaltase, sucrase, lactase and trehalase activities were assayed by the following modification of the method of DAHLQVIST[8].

A 25-μl sample of suitably diluted intestinal mucosa homogenate was added to 25 μl 56 mM oligosaccharide substrate (in 0.1 M sodium maleate buffer, pH 6.0), contained in a small tube. The mixture was incubated at 37° and the reaction stopped after 60 min by heating at 100° for 1 min.

After cooling, 300 μl of a modified Tris–glucose oxidase reagent were added. This reagent was prepared by mixing 100 μl 1% *o*-dianisidine (in 95% ethanol) and 500 μl of a mixture of 0.1% horse-radish peroxidase (Sigma Chemical Co., Type 1) and 0.2% highly purified glucose oxidase ("Glucose oxidase 130 000", from Fermco Labs., Inc., Chicago, Ill., U.S.A.) with 10 ml 0.5 M Tris buffer (pH 7.0). The reagent differed from that of DAHLQVIST[8] chiefly in the use of highly pure glucose oxidase and the omission of detergent.

The mixture was incubated at 37° for 30 min, which was sufficient for complete oxidation of the glucose. 150 μl 50% sulphuric acid[9] were then added and the ab-

sorbance of the purple solution read at 530 mμ against a boiled enzyme blank. An Unicam SP 600 spectrophotometer and microcell of 10 mm light path was used.

The amount of glucose produced was calculated from a standard graph in which an absorbance of 1.0 corresponded approximately to 5.0 μg glucose. In no case did the amount of glucose exceed 5.0 μg; this value corresponds to a degree of oligosaccharide hydrolysis of 0.67–2%, depending on the substrate used. Rates of oligosaccharide hydrolysis were expressed as μmoles hydrolysed per min per g wet wt. mucosa. In calculating the rate of hydrolysis of maltotriose it was assumed that the trisaccharide was completely hydrolysed to glucose (see RESULTS).

Amylase, maltotriase and maltase activities of duodenal juice

The amylase activity of human duodenal juice was assayed by measuring the liberation of reducing groups from starch using dinitrosalicylic acid. The method used was identical with that described by DAHLQVIST[10], except that the enzyme incubation was done at 37° instead of 25°. Maltotriase and maltase activities were assayed by incubating 25 μl diluted juice with 25 μl 56 mM substrate in 50 mM phosphate buffer (pH 6.9) containing 10 mM NaCl, at 37° for 1 h; these conditions are the same as those for the amylase assay. The glucose produced was then measured using glucose oxidase as described above. In calculating the maltotriase activity it was assumed that the trisaccharide was hydrolysed to glucose and maltose, with negligible further hydrolysis of maltose, during the initial reaction (see RESULTS).

Heat inactivation

Heat inactivation of intestinal enzyme activity was done in 10 mM sodium phosphate buffer (pH 7.0) as described by DAHLQVIST[11]. For the inactivation of duodenal mucosa, homogenates of mucosa obtained from several individuals were pooled to provide sufficient material.

Paper chromatography

This was done by the descending technique with Whatman No. 1 paper. Isopropanol–water (3:1, v/v) was used as the solvent system in an overnight run. The papers were developed by the silver nitrate procedure of SMITH[12].

RESULTS

Action of human duodenal juice on starch; formation of maltotriose

When diluted duodenal juice was allowed to act on soluble starch, a very rapid production of glucose, maltose and maltotriose was observed by means of paper chromatography (Fig. 1a). No isomaltose could be detected, but there was evidence of the presence of poorly staining and slow moving reducing substances which were presumably larger branched oligosaccharides containing one or more 1:6 links (α-limit dextrins)[2].

Comparison of the maltotriose spots on the chromatogram with a series of standard spots showed that the maltotriose represented approx. 25% by weight of the end products of starch hydrolysis.

Action of human duodenal juice on maltotriose and maltose

When maltotriose was incubated with diluted duodenal juice under the same

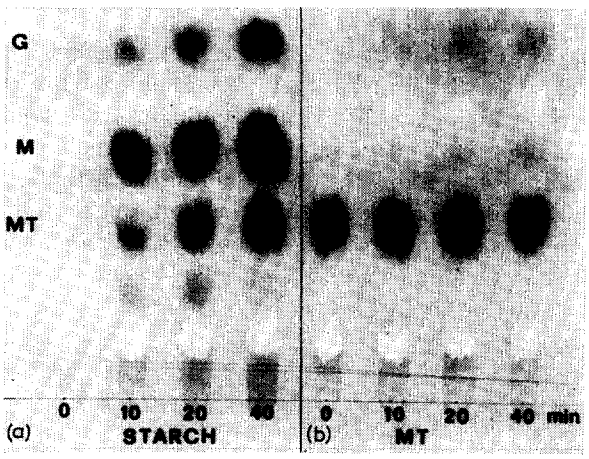

Fig. 1. Effects of human duodenal juice on starch (a) and maltotriose (b). Duodenal juice (Sample 1, Table I) diluted 40 times, 100 μl, was incubated at 37° with 100 μl 2% soluble starch solution (a) or 0.5% maltotriose solution (b); both substrates were made up in 10 mM sodium phosphate buffer (pH 6.9) containing 10 mM NaCl. Samples of 10 μl were removed for paper chromatography at the times indicated. G, glucose; M, maltose; MT, maltotriose.

conditions as those of the previous experiment, only slight hydrolysis could be detected chromatographically (Fig. 1b). The results of experiments in which the rates of hydrolysis of starch, maltotriose and maltose by samples of duodenal juice from two normal individuals were estimated quantitatively, are given in Table I. The mean rate of maltotriose hydrolysis was 0.45 μmole per min per ml juice; this was several hundred times less than the mean rate of formation of reducing compounds (mainly maltose and maltotriose) from starch. The maltase activity of the juice was very low.

All the amylase activity and a major part of the maltotriase activity of the juice was abolished by 1 mM EDTA. This suggests that most but not necessarily all of the maltotriase activity of the juice was due to the action of pancreatic α-amylase. The maltase activity was largely unaffected by EDTA (Table I).

TABLE I

AMYLASE, MALTOTRIASE AND MALTASE ACTIVITIES OF HUMAN DUODENAL JUICE

Amylase activity expressed as μmoles maltose equivalents per min per ml undiluted juice; Maltotriase and maltase activity as μmoles oligosaccharide hydrolysed per min per ml undiluted juice.

Juice (sample No.)	*Amylase*	*Maltotriase*	*Maltase*
1	240	0.22	0.038
1a*	670	0.96	0.051
2	80	0.18	0.035
2 + 1 mM EDTA	0	0.045	0.029

* After pancreozymin stimulation.

Action of human intestinal mucosa on maltotriose

When maltotriose was incubated with a homogenate of human intestinal mucosa, it was hydrolysed to glucose with very little intermediate formation of maltose (Fig. 2). Table IV permits a comparison of the rates of hydrolysis of maltotriose and of five disaccharides by specimens of duodenal mucosa from twelve normal individuals. The mean rate of maltotriose hydrolysis is seen to be of the same order as that of the hydrolysis of disaccharides. This rate was calculated on the assumption that all the maltose produced from maltotriose was hydrolysed to glucose. Since some maltose, may, however, accumulate (Fig. 2), the rate of hydrolysis (Table IV) must be regarded as a minimum value; if none of the maltose produced from maltotriose were hydrolysed, the rate of maltotriose hydrolysis would be three times as high. In all mucosa specimens, the rate of glucose formation from maltose was 2–3 times greater than that from maltotriose; this suggests that under the conditions of the quantitative assay the rate-limiting step in the hydrolysis of maltotriose was the splitting of the first bond, and not the further hydrolysis of maltose.

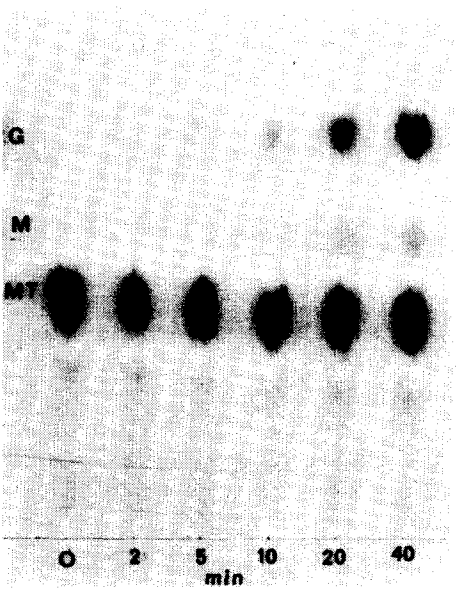

Fig. 2. Digestion of maltotriose by human intestinal mucosa. A 25-μl sample of homogenate of duodenal mucosa (1%, wet wt./vol.), was incubated with 25 μl 56 mM maltotriose in 0.1 M sodium maleate buffer (pH 6.0) at 37°; these conditions are identical with those for the quantitative estimation of intestinal maltotriase activity. Samples of 4 μl were removed for paper chromatography at the times indicated. G, glucose; M, maltose; MT, maltotriose.

The Michaelis constant for maltotriose, estimated from a double reciprocal plot of velocity against substrate concentration[13], at maltotriose concentrations between 3.5 and 28 mM was found to be 3.0 mM. This value is similar to the Michaelis constant for maltose (3.2 mM)[9].

Unlike the maltotriase activity of duodenal juice, that of intestinal mucosa was not abolished by 1 mM EDTA.

TABLE II

MIXED SUBSTRATE INCUBATIONS

The hydrolysis of maltotriose and disaccharides separately and in mixture. All substrates were used at a final concentration of 14 mM, and were incubated in the presence of duodenal mucosa from a normal individual. The amount of glucose produced from maltotriose varied between experiments because different concentrations of mucosa homogenate were used in each case.

Expt. No.	*Substrate*	*Glucose (μg per tube)*		*Found × 100 / Expected*
		Found	*Expected**	
1	Trehalose	1.50	—	—
	Maltotriose	4.43	—	—
	Trehalose + maltotriose	6.00	5.93	101
2	Lactose	0.82	—	—
	Maltotriose	3.67	—	—
	Lactose + maltotriose	4.35	4.49	97
3	Maltose	4.90	—	—
	Maltotriose	1.90	—	—
	Maltose + maltotriose	3.62	6.80	53
4	Isomaltose	0.93	—	—
	Maltotriose	2.13	—	—
	Isomaltose + maltotriose	1.57	3.06	51
5	Sucrose	0.70	—	—
	Maltotriose	2.60	—	—
	Sucrose + maltotriose	2.71	3.30	82

* Sum of glucose produced from disaccharide and maltotriose when incubated separately.

TABLE III

DISTRIBUTION OF HUMAN INTESTINAL MALTASE AND MALTOTRIASE ACTIVITIES BETWEEN THE FOUR MALTASES

All figures represent the mean values from at least three heat-inactivation experiments, similar to those illustrated in Fig. 3.

Enzyme	*Substrate*	
	Maltose	*Maltotriose*
	Duodenum	
Maltase Ia	52	58
Maltase Ib	36	36
Maltases II and III*	12	6
	Ileum	
Maltase Ia	45	48
Maltase Ib	20	28
Maltase II	22	4
Maltase III	13	20

* By difference.

Mixed substrate incubations

To obtain information on the identity of the enzyme(s) responsible for the intestinal hydrolysis of maltotriose, the trisaccharide was incubated with mucosal homogenate in the presence of various disaccharides; the amount of glucose produced was compared with the sum of that produced when maltotriose and disaccharide were incubated separately with the homogenate.

With maltotriose and trehalose, and maltotriose and lactose, the amount of glucose produced by mucosa in the presence of both substrates was approximately equal to the expected value, *i.e.* to the sum of that produced when the substrates were incubated with mucosa separately (Table II). Hence maltotriose and trehalose, and maltotriose and lactose, had no effect on each other's hydrolysis. With maltotriose and maltose, however, the amount of glucose produced was only 53% of the expected value; with maltotriose and isomaltose 51%, and with maltotriose and sucrose 82% (Table II).

Heat inactivation

It has been shown that the maltase activity of human small intestinal mucosa can be separated into four fractions by heat inactivation[10,14,15]. These fractions were considered to represent four different enzymes and have been called[11] maltases Ia, Ib, II and III, in order of increasing heat stability*. In the experiments of DAHLQVIST[11], maltase Ia was inactivated at 45°, maltase Ib at 50°, maltase II at 60° and maltase III at 75°, complete inactivation being achieved in each case by heating at pH 7.0 for 1 h. Maltase Ia had both maltase and isomaltase activity, maltase Ib had maltase and sucrase activity, and maltases II and III had only maltase activity.

In order to obtain information on the relationship of the human intestinal maltotriase activity to the above four maltases, the heat-inactivation experiments of DAHLQVIST[11] were repeated, using maltose, isomaltotriose**, sucrose and maltotriose as substrates.

The heat inactivation curves for maltases Ia and Ib obtained with duodenal mucosa were similar to those obtained by DAHLQVIST[11] with jejunal mucosa, the chief difference being that it was necessary to use slightly higher temperatures (46° and 55°, respectively) to achieve complete inactivation. Maltase Ia (isomaltase) contributed approx. 52% of the total maltase activity, and maltase Ib (sucrase) 36%. No attempt was made to inactivate maltases II and III, since these enzymes together contributed only about 12% of the total duodenal maltase activity. The heat inactivation curves for maltotriase activity closely followed those for maltase; this suggested that the major part of the duodenal maltotriase activity is due to the action of maltases Ia and Ib, maltase Ia contributing about 58% of the total maltotriase activity, and maltase Ib 36%.

To obtain information on the possible contribution of maltases II and III to the intestinal maltotriase activity, the heat inactivation experiments were repeated

* SEMENZA, AURICCHIO AND RUBINO[16] have numbered the human intestinal maltases according to a system which is the reverse of that of DAHLQVIST[11]. This system is based on the order of emergence of the papain-solubilised enzymes from a gel filtration column. In the present work the numbering system of DAHLQVIST is used throughout, since all experiments were done with unsolubilised and unfractionated material.

** Isomaltotriose was used as substrate for isomaltase in these experiments because no isomaltose was available at the time.

using ileal mucosa, since it has been shown[11,15] that the lower part of the small intestine contains a greater proportion of these heat-stable maltases than the upper. This observation was confirmed in the present work, since approx. 35% of the total ileal maltase activity remained after heating at 46° and 55° (Fig. 3). As with duodenal mucosa, the heat inactivation curves for maltotriase were very similar to those for maltase at these two temperatures. When the ileal mucosa was then heated at 63°, the maltase activity fell by 22% of the original activity (Fig. 3), but there was very little reduction in the maltotriase activity; this suggested that maltase II, which is inactivated at this temperature, has very little maltotriase activity. When heating was continued at 75°, the remaining maltase as well as maltotriase activities were

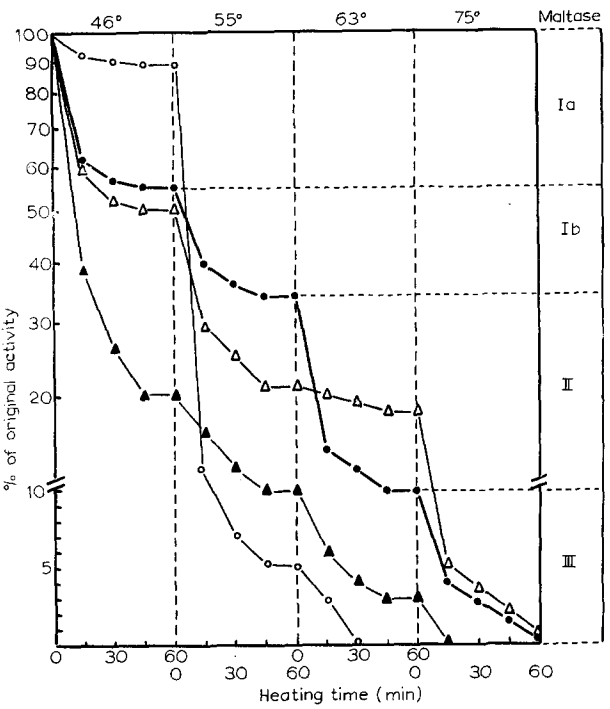

Fig. 3. Step-wise heat inactivation of the maltase, maltotriase, isomaltotriase and sucrase activity of human ileal mucosa. The figure is a composite from four separate experiments, one for each heat-inactivation temperature. Mucosa which was heated at one of the higher temperatures had been pre-heated at all the lower ones. Note that part of scale of ordinate (below 10) is not logarithmic. ●—●, maltase; △—△, maltotriase; ▲—▲, isomaltotriase; ○—○, sucrase.

inactivated at identical rates; this indicated that the maltotriase activity which remains after heating at 63°, amounting to approx. 20% of the original activity, is due to maltase III. Since the rate of glucose formation from maltose by mucosa which had been heated at 63° was approximately twice as great as that from maltotriose, it is unlikely that the parallel nature of the maltotriase and maltase curves during heating at 75° was an artifact, *i.e.* that the rate-limiting step in maltotriose hydrolysis by heated mucosa was the rate of hydrolysis of maltose produced from maltotriose.

Table III summarises the contributions presumed to be made by each of the

four maltases to the total maltase and maltotriase activities of duodenal and ileal mucosa. It is based on the observation that in each case the curves flattened out, suggesting that a complete separation of the enzyme activities was therefore achieved. The results for maltase activity are similar to those of DAHLQVIST[11].

Maltotriase activity of duodenal mucosa from patients with congenital intestinal sucrase and isomaltase deficiency

Recent work has shown that patients with congenital intolerance towards sucrose are deficient in intestinal isomaltase and sucrase (maltases Ia and Ib)[17-24]. The above results suggest that these enzymes are involved in the hydrolysis of maltotriose; these patients could therefore be expected to have reduced intestinal maltotriase activity.

TABLE IV

OLIGOSACCHARIDASE ACTIVITY OF HUMAN DUODENAL MUCOSA

Normal subjects and patients with congenital sucrose intolerance. All activities are expressed as μmoles oligosaccharide hydrolysed per min per g wet wt. of mucosa. The mean activities for normal subjects are average values from 12 individuals.

	Lactase	*Trehalase*	*Sucrase*	*Isomaltase*	*Maltase*	*Maltotriase*
Normal subjects						
Mean	2.8	1.7	6.6	6.4	16	3.8
Range	1.0–6.2	0.6–2.8	2.6–13	2.2–13	9.5–28	2.4–4.7
Sucrose-intolerant patients						
P.O.	2.9	1.0	0.03	0.25	3.0	0.35
A.S.	4.7	2.4	0.02	0.19	3.7	0.35
J.S.	4.0	2.7	0.01	0.30	7.5	0.72
F.N.	1.3	0.64	0.02	0.11	1.6	0.19

Table IV presents a comparison of the duodenal oligosaccharidase activities of twelve normal individuals with those of four patients with congenital sucrose intolerance. It is seen that in the patients, the trehalase and lactase activities were within the normal range, but there was only a trace of sucrase activity and the isomaltase activity was very low. The maltase activity was well below the normal range. These results are similar to those previously obtained in this and other laboratories[19-24]. It is of considerable interest that in all four patients the maltotriase activity was greatly reduced.

When duodenal mucosa from two of the sucrose-intolerant patients (A.S. and F.N.) was subjected to heating at 55° for 1 h, a mean reduction of 26% in the isomaltase, and of 20% in the maltotriase activity was observed. Since heating at 55° inactivates maltases Ia and Ib (see above), most of the isomaltase and maltotriase activities of the intestinal mucosa of these patients must be due to the heat-stable maltases II and/or III.

DISCUSSION

Formation of maltotriose from starch

The digestion of starch begins with the action of salivary α-amylase in the mouth and stomach, and is continued by pancreatic α-amylase in the small intestine. The extensive investigations of WHELAN and co-workers[2,25,26], have shown that the main products of the action of human salivary α-amylase on starch are maltose, maltotriose and α-limit dextrins (branched oligosaccharides containing one or more 1:6 linkages). The maltotriose constitutes 28% of the products from amylopectin[2], and 37% of those from amylose[25]. Similar quantitative data are not available for human pancreatic α-amylase, but in the present investigation approx. 25% of the products of the action of human duodenal juice on starch were estimated, semi-quantitatively, to be maltotriose. Thus, in regard to the formation of maltotriose, the action of human pancreatic α-amylase appears to resemble that of salivary α-amylase, a conclusion which is consistent with the close similarity between other properties of the two enzymes[27].

The above experiments were done *in vitro*; during digestion of starch within the human intestine, the final yield of maltotriose can be expected to be greater than the above figures suggest, since the α-limit dextrins are known to be a further source of maltotriose[2].

ROBERTS AND WHELAN[2] have shown that the smallest α-limit dextrin formed from amylopectin by human salivary α-amylase is a pentasaccharide. Some authors (*e.g.* AURICCHIO *et al.*[28]) have stated, however, that isomaltose, a disaccharide, is formed during α-amylolysis of starch. In the present investigation no isomaltose could be detected among the products of the action of duodenal juice on starch.

Intestinal digestion of maltotriose

Human salivary α-amylase can catalyse the hydrolysis of maltotriose, but only at enzyme concentrations which are much greater than those required for the digestion of starch[26]. Our observations on the effect of human duodenal juice on maltotriose suggest that in this regard, too, human pancreatic α-amylase resembles the salivary enzyme, and confirm those of RUTLOFF, FRIESE AND TÄUFEL[3] on purified pig pancreatic α-amylase. These authors have concluded that the action of the pig enzyme on maltotriose is too small to be of digestive significance. This conclusion appears to apply equally to human pancreatic α-amylase, since the rate of hydrolysis of maltotriose by duodenal juice was over 100 times less than the rate of formation of maltose and maltotriose from starch.

RUTLOFF, FRIESE AND TÄUFEL[3] showed that pig and rat intestinal mucosa homogenates catalyse the hydrolysis of maltotriose at rates which are similar to those for the hydrolysis of maltose. The present findings on human tissue permit a comparison of the rates of hydrolysis of maltotriose and of five disaccharides, and of the Michaelis constants for maltotriose and maltose. The results show that the intestinal mucosa is capable of digesting the trisaccharide with an efficiency which is equal to that for the digestion of disaccharides. These results make it apparent that the site of digestion of maltotriose is the small intestinal mucosa, as it is for the digestion of disaccharides.

Maltotriase activity of the human intestinal maltases

The mixed substrate incubation experiments showed that maltotriose and maltose, maltotriose and isomaltose, and maltotriose and sucrose inhibited each other's hydrolysis; this suggests competition for the same enzyme(s) by members of each pair of substrates. The possibility that the observed inhibition was due to the maltose produced from maltotriose (Fig. 2) is made unlikely by the fact that the degree of hydrolysis of maltotriose in these experiments was no more than 1.3–4%; the maximum concentration of maltose that could have been present is therefore 0.56 mM, as against 14 mM for the other substrates.

Differential heat inactivation of the mucosa homogenates showed that the maltotriase activity is inactivated at the same temperatures, and at the same rates, as are maltases Ia, Ib and III. This suggests that the maltotriase activity of human intestinal mucosa is due to these three enzymes, and not to a specific maltotriase.

Further evidence for the role of maltases Ia and Ib in the hydrolysis of maltotriose was obtained by examining the maltotriase activities of duodenal biopsy specimens from children with congenital sucrose intolerance, a condition in which there is a deficiency in these two maltases[18–24,29]. The four children examined all had intestinal maltotriase activities considerably below normal.

Implications for congenital sucrose intolerance

Some patients with congenital sucrose intolerance also show mild intolerance towards starch[17]. It has been suggested that this effect is due to oligosaccharides containing 1:6 links, which remain unsplit in the small intestine because of the deficiency in intestinal isomaltase of these patients[28]. The present work shows that the patients also have reduced intestinal maltotriase activity, suggesting that they may be deficient in their ability to deal with maltotriose. Since maltotriose forms a substantial part of the products of the α-amylolysis of starch, it probably contributes to the starch intolerance of these patients.

The isomaltotriase activity of maltases II and III observed in the present experiments (Fig. 3) provides a possible explanation for the residual isomaltase activity which is always present in the intestinal mucosa of sucrose-intolerant patients[18–24]. It would seem that this activity may be due mainly to the heat-stable maltases II and III, since the isomaltase activity was only partially abolished by heating at 55°. The observation that some activity was, however, lost suggests that a small amount of maltase Ia (and/or Ib) activity is present in these patients.

The trace of sucrase activity also observed in duodenal mucosa specimens from these patients can be ascribed, partially or wholly, to the slight sucrase activity of maltase II (Fig. 3).

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