Elevated Intestinal Disaccharidase Activity in the Streptozotocin-Diabetic Rat Is Independent of Enteral Feeding

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SUMMARY

Specific and total activities of the disaccharidases, sucrase, maltase, and lactase are increased in mucosa of the small intestine of the streptozotocin diabetic rat. Because disaccharidases are essential for terminal digestion of carbohydrate, and disaccharidase deficiency is a common clinical problem, understanding the mechanisms regulating disaccharidase activity is important. In normal animals, disaccharidase activities are determined by route of feeding and are decreased by parenteral feeding. The indirect exocrine, endocrine, neurocrine, and paracrine functions of the gastrointestinal tract that are dependent on feeding via the gut are greatly decreased in parenteral as compared with enteral feeding. Hormone secretion by the gut and the pattern of response after feeding may be abnormal in diabetes and might be regulatory for disaccharidases. We tested the hypothesis that the elevated intestinal disaccharidases in diabetes are dependent on enteral feeding. Streptozotocin-injected rats (diabetics) and vehicle-injected rats (controls) were fed rat chow ad libitum for 4 days. A subset of control and diabetic animals was then killed to determine disaccharidase activity of the jejunum at the start of pair-feeding the elemental diet. The remaining animals were fed 60 cal/day of glucose, amino acid (Travasol), and electrolyte solution either intragastrically or intravenously for 4 days. Specific and total activities of disaccharidases were greater in diabetics than in controls under all feeding conditions. In controls, the pattern of activity of disaccharidase specific activity was initial > intragastric > intravenous. In diabetics, disaccharidase specific activities did not differ among groups. In both controls and diabetics, mean mucosal mass was highest initially; intermediate with intragastric feeding; and lowest with intravenous feeding. In both controls and diabetics, total disaccharidases decreased from initial to intragastric to intravenous. We conclude: (1) disaccharidase specific activity in controls is sensitive to feeding route and nature of diet, but is nearly independent of these factors in diabetics; (2) total disaccharidase activities respond to feeding stimuli in parallel with changes in mucosal mass in both controls and diabetics; and (3) the lack of feeding effect on the elevated specific activities of disaccharidases in diabetes suggests that this elevation is a response to the diabetic state and is independent of enteral factors such as luminal nutrition and gastrointestinal hormones.

DIABETES *32*:265–270, March 1983.

The disaccharidases of the brush border membrane carry out the terminal steps in carbohydrate digestion: their deficiency leads to osmotic diarrhea.[1] Disaccharidase deficiency may be primary or acquired secondary to mucosal disease or intestinal resection. Adult lactase deficiency with intolerance to dairy products is one of the most common causes of intestinal distress. Because of our limited knowledge of regulation of mucosal disaccharidases, no specific treatment for increasing disaccharidase activity is available. In the streptozotocin-diabetic rat, specific and total activities of the disaccharidases sucrase, maltase, and lactase are increased.[2–6] Elucidation of the mechanism of this response is potentially of great importance in treatment of disaccharidase deficiency, as well as in understanding regulation of mucosal function.

In all prior studies in diabetics, animals were fed enterally. Hormone secretion by the gut and the pattern of hormone release after feeding may be abnormal in diabetes. Serum gastrin is increased both fasting[7] and postprandially in human diabetics.[8,9] Gastrin is also increased in the genetically diabetic mouse[10] and in the streptozotocin-diabetic rat.[11] Hormonal, neurocrine, or paracrine factors released from the gut by feeding might be regulatory for the increased di-

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Received for publication 17 August 1982.

saccharidases in diabetes. Parenteral feeding produces structural and hormonal alterations in the gastrointestinal tract[12] presumably secondary to loss of enteral stimuli. To test the hypothesis that the elevated disaccharidases in the diabetic rat are dependent on enteral feeding, we pair-fed control and diabetic rats identical diets enterally and parenterally, and present our results here.

MATERIALS AND METHODS

Animals. Male albino rats of the Sprague-Dawley strain (King Laboratories, Oregon, Wisconsin) were purchased at the 150-g weight range. Animals were fed Purina Chow (No. 5012) ad lib on arrival. Two days after receipt, animals were weighed, marked, and ad lib feeding was continued. Animals were weighed regularly to establish rate of weight gain, and on reaching the 200–220-g weight range they were divided randomly into a control group and a group to be made diabetic. The diabetic group was injected intraperitoneally with a solution of streptozotocin, 80 mg/kg body wt (The Upjohn Co., Kalamazoo, Michigan), in aqueous citrate buffer (pH 4.5) within 2 min of preparation. Approximately 0.5 ml was injected, and animals in the control group received 0.5 ml of citrate buffer only. Diabetes was verified by polyuria, glucosuria (Tes-Tape, Eli Lilly and Company, Indianapolis, Indiana), and weight loss. Four days after injection, part of the controls and diabetics were killed and surgical procedures for enteral and parenteral feeding were performed in remaining control and diabetic groups.

Parenteral feeding. The technique for parenteral feeding was a modification of that of Steiger et al.[13] Rats were anesthetized with chloral hydrate injected intraperitoneally. The neck was shaved and prepared, and the animals were restrained and placed in the supine position on an operating board. With sterile instruments and aseptic technique, a longitudinal incision was made lateral and to the right of the midline, overlying the right jugular vein. The vein was isolated and ligated 0.5–1 cm above the clavicle with 5–0 silk suture. Another tie was placed around the vein distally, to be tied later around the catheter. The tip of a 50-cm long silicone rubber catheter (Silastic Brand Medical Grade Tubing 0.020 in IDX 0.037 in OD) was inserted through a phlebotomy in the jugular vein in between the sutures, and advanced so the tip lay in the superior vena cava or right atrium. The catheter was then secured with both ties, with attention being made not to occlude the lumen. The free end of the catheter was then tunneled underneath the skin, using a metal stylet, to exit through the skin in the midscapular area.

The catheter was passed through a specially constructed harness and a stainless steel spring stock and connected to a sterile swivel infusion apparatus (Model 375/22, Instech Laboratories). The swivel was fixed to a support assembly over the metabolic cage, and was further connected to the pump and bottle containing the parenteral nutrition solution by polyvinyl infusion tubing (No. 3002 No. 24 clear transflex tubing, Minnesota Mining and Manufacturing Co.). The harness was secured to the animal by adhesive tape. This technique facilitates the infusion of the desired solutions parenterally, with the rat being unrestrained and actively mobile in the metabolic cage.

The tubing was attached to a non-syringe-type infusion pump (Harvard Apparatus Co., Millis, Massachusetts), and the rats were infused with a solution containing the following: 100 ml of 5% dextrose monohydrate in 0.9% saline (Travenol Laboratories, Deerfield, Illinois), 30 ml of 8.5% Travesol (amino acids) injection with electrolytes (Travenol Laboratories), 20 ml of 50% dextrose monohydrate, and 0.1 ml of multivitamin infusion (MVI; USV Laboratories, Tuckahoe, New York). The infusion rate was 3 ml/h for the first day and was increased to 6 ml/h the day after surgery and was continued at this rate.

Enteral feeding. The same harness and swivel infusion apparatus was used to administer the nutrient solution into the stomach. For this purpose the catheter was passed from the midscapular area subcutaneously to the anterior abdominal wall. At this site it was inserted through an abdominal incision into the fundus of the stomach via a gastrotomy and held in place by sutures in the stomach wall.[14] The abdomen was closed by clips. The same infusion solution and infusion rate were used as for parenteral feeding. Both enterally and parenterally fed rats received water ad lib.

Sacrifice procedure. The rats were infused until immediately before death and then were anesthetized with Nembutal, 40 mg/kg body wt. A midline abdominal incision was made and blood was drawn from the bifurcation of the aorta. The blood was clotted on ice, centrifuged, and serum separated for analysis of glucose[15] and phosphorus.[16] Calcium was measured by atomic absorption spectrophotometry (Perkin Elmer 303, Perkin Elmer Corp., Norwalk, Connecticut) and osmolality by freezing point depression (Osmometer 31 LA, Advanced Instruments Inc., Highlands, Michigan).

The small intestine was transected at the pylorus and ileocecal valve, removed, and rinsed intraluminally with cold saline. Contents were expressed by finger pressure. The small intestine was divided by length into three equal proximal, mid, and distal segments and their lengths and weights were measured. The mid segment was placed on a glass plate over ice, slit longitudinally, and opened flat with the mucosal surface up. The mucosal surface was scraped with the edge of a microscope slide, and the mucosal scrapings and underlying tissue were weighed and the mucosa was mixed to homogeneity. One-third of the mucosa was frozen in acetone and dry ice and stored at −60°C for assay of disaccharidases,[17] protein,[18] and DNA.[19] At the time of assay, the frozen mucosal sample was thawed and homogenized in 10 vol of distilled water using a Potter-Elvehjem homogenizer with a Teflon pestle. The weight of the homogenate was obtained, and aliquots were taken and diluted appropriately. The proximal and distal segments were treated as above, except samples were not taken for assay. All remaining tissue was dried in a vacuum oven for 24 h at 90°C and $\frac{1}{3}$ atm pressure. Dry weights were measured after removal from the oven.

Statistical analysis between controls and diabetics was by the unpaired Student's *t* test. When comparisons were made between feeding modes for control or diabetic groups, *i.e.*, chow-fed versus enteral versus parenteral, data were analyzed by one-way analysis of variance with Tukey's multiple comparison test: P values of <0.05 were considered significant.

RESULTS

Body weight and serum analysis. All groups had similar body weights at the time of injection of buffer or streptozotocin (Table 1, injected). During the first 4 days postinjection,

TABLE 1
Body weight and serum data for control (C) and diabetic (D) groups, mean ± SE

Groups	Chow-fed		Enteral		Parenteral	
	C	D	C	D	C	D
Number in group	5	5	11	9	7	14
Body weight (g)						
Injected (Day 0)	208 ± 5	211 ± 5	216 ± 3	219 ± 3	216 ± 5	224 ± 5
Initial (Day 4)	231 ± 7	204 ± 4	243 ± 4	211 ± 6	240 ± 5	218 ± 5
Final* (Day 8)			232 ± 2	192 ± 5	224 ± 6	185 ± 4
Serum concentrations (mg/dl)						
Glucose	186 ± 7	576 ± 44†	203 ± 10	503 ± 38†	181 ± 14	576 ± 62†
Calcium	10.4 ± 0.2	10.6 ± 0.2	9.4 ± 0.1	8.6 ± 0.2†	10.0 ± 0.1	9.0 ± 0.2†
Magnesium	1.54 ± 0.03	1.53 ± 0.05	1.44 ± 0.03	1.60 ± 0.08	1.61 ± 0.11	1.77 ± 0.17
Phosphorus	8.1 ± 0.2	7.9 ± 0.2	7.4 ± 0.2	6.4 ± 0.3†	8.2 ± 0.2	9.0 ± 0.7
Osmolality (mosmol/kg)‡						
Total	304 ± 5	331 ± 4	300 ± 1	310 ± 4	300 ± 2	317 ± 3
Glucose	10 ± 1	32 ± 5	12 ± 2	28 ± 6	10 ± 2	30 ± 3
Non-glucose	294 ± 5	299 ± 3	289 ± 1	282 ± 3	291 ± 2	289 ± 3

*Final weight for the enterally and parenterally fed groups is adjusted for weight of contents of gastrointestinal tract present at implantation of catheters. Data are from chow-fed initial set of groups at death. For controls: 9 g is added to weight. For diabetics: 11 g is added to weight.
†Diabetic differs from control, $P < 0.02$ or less, unpaired Student's t test.
‡Comparing diabetics, chow-fed > enteral and parenteral for total osmolality and non-glucose osmolality, $P < 0.05$.

controls gained weight and diabetics lost weight (injected weight versus initial weight). Both groups lost weight during the 3- or 4-day period of enteral or parenteral feeding (initial weight versus final weight). Although all groups received identical intakes, diabetics lost more weight than controls. Weight losses of corresponding groups were comparable with enteral and parenteral feeding. Serum glucose concentrations and serum osmolalities were elevated in diabetics (Table 1). The higher serum glucose concentrations in diabetics accounted for their increased serum osmolality. Serum calcium was lower in enterally and parenterally fed diabetics than in corresponding control groups (Table 1). Serum magnesium was the same in diabetic and control groups regardless of route of feeding. Serum phosphorus was lower in enterally fed diabetics than in controls.

Intestinal mucosal weight, protein, and DNA. Mean mucosal dry weights (mg/cm) of proximal, mid, and distal segments at the start of enteral and parenteral feeding (chow-fed) were higher than at the end of feeding the elemental diet (enteral and parenteral) and higher in diabetics than controls, but differences were significant only in the proximal segment (Table 2). Mucosal weights (mg/cm of proximal, mid, and distal segments) were similar for controls and diabetics in both enteral and parenteral groups. Mucosal weight was greater in enterally than in parenterally fed groups in the proximal and mid segments and showed a gradient from proximal to distal. This gradient was lacking in the parenterally fed group. Mean mucosal protein and DNA data for the mid segment were higher in enterally than in parenterally fed groups, and higher in controls than diabetics, but differences were not significant.

Mid-segment disaccharidases. Specific activities of disaccharidases are shown in Figure 1. The initial data from animals taking Purina Chow at the start of pair-feeding elemental diets enterally and parenterally are shown in the upper left panel. At 4 days postinjection activities, U/g protein, are increased in diabetics as compared with controls for sucrase, maltase, and lactase. Activities after 4 days of

TABLE 2
Intestinal mucosal data (mg/cm, mean ± SE)

Groups	Status	Mucosa of intestinal segments† (dry wt)			Mid-intestinal segment mucosa‡	
		Proximal	Mid	Distal	Protein	DNA
Chow-fed	Control	9.4 ± 0.5	9.7 ± 0.5	7.3 ± 0.6	7.3 ± 0.5	0.33 ± 0.03
	Diabetic	11.9 ± 0.7*	10.9 ± 0.6	8.6 ± 0.5	8.9 ± 0.7	0.36 ± 0.03
Enteral	Control	8.7 ± 0.3	8.5 ± 0.4	5.9 ± 0.4	6.3 ± 0.4	0.30 ± 0.02
	Diabetic	7.6 ± 0.3*	7.9 ± 0.5	5.2 ± 0.4	5.5 ± 0.2	0.28 ± 0.01
Parenteral	Control	5.3 ± 0.2	6.2 ± 0.4	5.5 ± 0.4	5.1 ± 0.4	0.26 ± 0.04
	Diabetic	6.0 ± 0.3	6.8 ± 0.3	5.1 ± 0.2	4.9 ± 0.2	0.24 ± 0.02

*Diabetic differs from control, $P < 0.05$ or less, unpaired Student's t test.
†Comparing mucosal dry wt data among groups, analysis of variance and Tukey's multiple comparison test, $P < 0.05$ or less: (1) In proximal segment, for controls, chow-fed, enteral > parenteral; for diabetics, chow-fed > enteral > parenteral. (2) In mid segment, for controls, chow-fed, enteral > parenteral; for diabetics, chow-fed > enteral, parenteral. (3) In distal segment, for controls, chow-fed > parenteral; for diabetics, chow-fed > enteral, parenteral.
‡Comparing mid-segment data among groups, analysis of variance and Tukey's multiple comparison test, $P < 0.05$ or less: (1) Protein, chow-fed > parenteral for controls; chow-fed > enteral and parenteral for diabetics. (2) DNA, chow-fed, enteral and parenteral for diabetics.

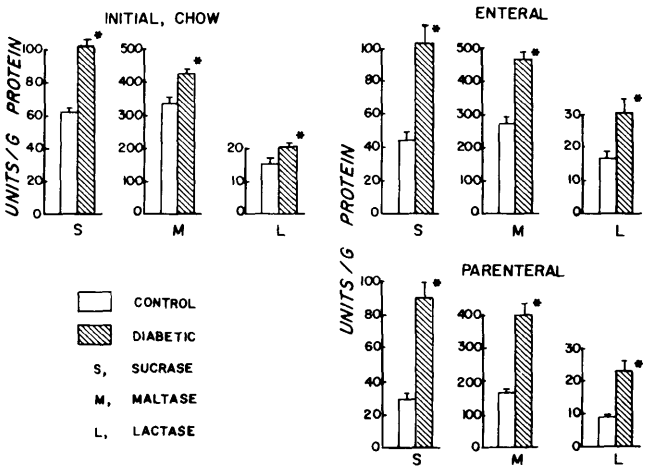

FIGURE 1. Mid-segment disaccharidase specific activities (U/g protein) of small intestine of control and diabetic rats at the start (initial, chow-fed) and after 4 days of pair-feeding elemental diets enterally or parenterally (mean ± SE). At 4 days after injection of buffer in controls and streptozotocin in animals to be made diabetic, sucrase (S), maltase (M), and lactase (L) are increased in diabetics (hatched bar) as compared with controls (clear bar), $P < 0.05$, upper left-hand panel. On the right are shown disaccharidase specific activities after 4 days of subsequent enteral (top) or parenteral (bottom) feeding of animals. Activities remain increased in diabetics as compared with controls after feeding the elemental diet enterally or parenterally. *Diabetic greater than control, $P < 0.05$.

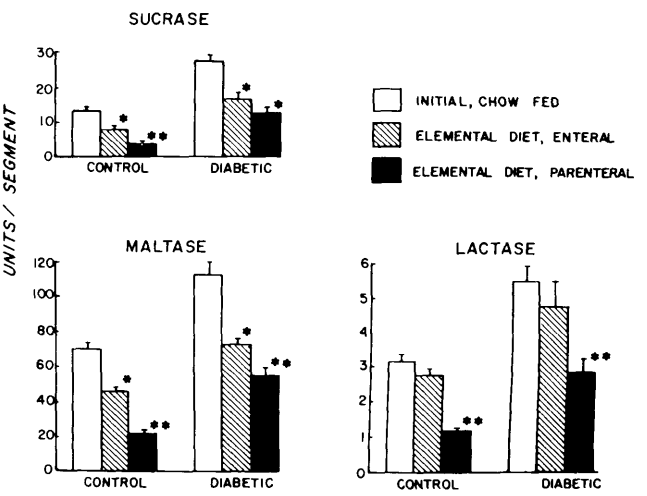

FIGURE 3. Total mid-segment disaccharidase activities (U/segment) in control and diabetic rats (mean ± SE). Total activities are shown for sucrase, maltase, and lactase in chow-fed animals at the start of pair-feeding the elemental diet (initial, chow-fed) and after 4 days of enteral or parenteral feeding. Total activities declined in both enterally and parenterally fed controls and diabetics in parallel with the decreased mucosal mass (Table 2), but relationships between controls and diabetics for total activities are the same as for specific activities: disaccharidase activities are greater in diabetics than controls, independently of feeding route. *Differs from corresponding initial, chow-fed group, $P < 0.05$. **Differs from both corresponding initial, chow-fed group and enteral group, $P < 0.05$.

enteral (top) or parenteral (bottom) feeding (8 days postinjection of streptozotocin or vehicle) are shown on the right side of Figure 1. Activities of all three enzymes remained increased in diabetics as compared with controls after feeding the elemental diet enterally or parenterally.

To demonstrate the pattern of disaccharidase specific activity from the start of feeding (chow: initial) and after enteral or parenteral pair-feeding of control and diabetic rats, data of Figure 1 are replotted in Figure 2. In controls, disaccharidase activities decreased with feeding the elemental diet, even when administered enterally, except for lactase, where enteral feeding had no effect. In enterally fed diabetics, mean sucrase did not change with the elemental diet, mean maltase specific activity increased slightly, and the increase with lactase was significant. When comparing enteral with parenteral feeding we find that in controls all three disaccharidases decreased significantly in specific activity ($P < 0.05$) with parenteral feeding; in diabetics, the small decrease with parenteral feeding was not significant.

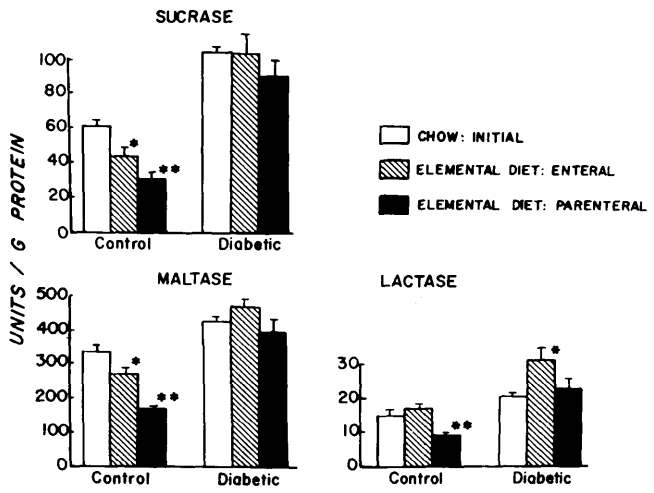

FIGURE 2. Data of Figure 1 replotted to show pattern of disaccharidase specific activities from the start (chow, initial) in relation to type of feeding, enteral or parenteral (mean ± SE). For each disaccharidase, activities are shown on the left for controls and on the right for diabetics. In controls, mean disaccharidase activities tended to decrease with feeding the elemental diet. In diabetics, disaccharidase activity was unaffected by feeding route, except for lactase, where the enteral group was higher than the chow, initial group. *Differs from corresponding chow-fed, initial group; **differs from both corresponding chow-fed, initial and enteral groups. Data analysis was by one-way analysis of variance and Tukey's multiple comparison test.

Total disaccharidase activity (U/segment) is the activity of the middle one-third of the small intestine (Figure 3). Mid segments were closely matched among groups: (1) on the basis of length, the mid segments ranged from 32.8 ± 0.4% of total length in the parenteral control group to 33.6 ± 0.3% in the parenteral diabetic group; (2) on the basis of wet weight, mid segments ranged from 36.4 ± 4.9% of total intestinal wet weight in the enteral control group to 38.2 ± 5.6% in the parenteral diabetic group. All other groups were within these ranges. In comparison with initial activities in chow-fed animals, total segment sucrase and maltase decreased significantly in both controls and diabetics given the elemental diet administered enterally or parenterally ($P < 0.05$). Enteral feeding did not significantly decrease total lactase activity in either controls or diabetics, in contrast to parenterally fed controls and diabetics, where lactase was greatly reduced. The decrease for all enzymes was greater in parenterally than enterally fed control and diabetic groups. Mid-segment mucosal weights of controls and diabetics were the same for corresponding enterally and parenterally fed groups (Table 2). Therefore, with respect to the comparison of controls and diabetics fed enterally or parenterally, the same relationship holds for total as for specific activities.

Total disaccharidase activity is greater in diabetics than controls, whether fed enterally or parenterally. The decrease in total segment activities as compared with initial values in chow-fed animals is in part the result of the decrease in mucosal weight, protein, and DNA in animals fed the elemental diet (Table 2).

DISCUSSION

These studies demonstrate that both specific and total activities of sucrase, maltase, and lactase are increased in small intestinal mucosa of diabetics as compared with controls as early as 4 days of diabetes in rats fed natural food. This increased disaccharidase activity in diabetics is maintained during 4 days of pair-feeding an elemental diet enterally or parenterally. Parenteral feeding is as effective as enteral feeding in maintaining the increased specific and total activities of disaccharidases in diabetics as compared with controls. Feeding an elemental diet subsequent to chow-feeding causes sucrase and maltase specific activities to decline in controls but not in diabetics. Similarly, specific activities of disaccharidases decrease in controls, but not diabetics, with parenteral as compared with enteral feeding. Thus, specific activities of disaccharidases in diabetics are independent of feeding route and intake of elemental diet as compared with chow. In contrast, total segment disaccharidases showed the same behavior in controls and diabetics: feeding an elemental diet after chow-feeding decreased activities because of decreased mucosal mass; parenteral feeding caused a further decline, but with a lesser decrease in diabetics than controls. Based on these findings, we conclude that the increased disaccharidases in diabetes are independent of enteral feeding, ruling out dependence on direct effects of luminal nutrients and indirect effects of enteral feeding, such as release of hormonal or neural factors. The possibility that parenteral as well as enteral feeding releases a hormone that mediates the disaccharidase response cannot be ruled out, but is unlikely, because of the lesser effect of parenteral feeding on hormone release.

The specific activity of disaccharidases in control animals is highly dependent on enteral feeding, unlike the diabetic, where activity appears to be relatively independent of this mechanism (Figure 2). Thus, the increased disaccharidase activity in diabetes appears to be secondary to stimuli related to the diabetic state, possibly hyperglycemia. Intracellular glucose and glucose secretion into the gut lumen is expected to be increased in diabetics because of hyperglycemia. Loading of mucosal cells with glucose from the blood is probably similar in both enterally and parenterally fed animals, since serum glucose values are similar in corresponding groups (Table 1). Higher blood glucose in diabetics, however, would increase glucose secretion in diabetics as compared with controls, so that luminal loading of mucosa with glucose would also be increased in diabetics. The findings with disaccharidases are similar to the increased intestinal mucosal growth response in diabetes,[20,21] which has no primary dependence on gastrin.[11] Mucosal cell turnover in the rat is 24–36 h and $t_{\frac{1}{2}}$ for disaccharidase is of the order of 5 h. Although these may be prolonged with parenteral as compared with enteral feeding, it is apparent that the increased disaccharidase activities of diabetics are maintained through cycles of cell renewal and disaccharidase resynthesis in parenterally fed animals.

Prior studies have examined regulatory factors for disaccharidases. In the newborn rat pup, disaccharidases are induced by carbohydrate feeding and by adrenal steroids.[22,23] In the adult rat, fasting decreases specific and total disaccharidase activities,[24,25] which are restored by carbohydrate feeding. Adrenalectomy of adult rats has little effect on disaccharidase activities, and there is no response to cortisone treatment of these animals.[25] Hence, it is unlikely that the elevated adrenal cortical activity in the rat with experimental diabetes[26] is responsible for the increased disaccharidases.

The effects of parenteral feeding on intestinal mucosal disaccharidase activities have been examined in normal rats.[27–29] Experimental designs differed from our protocol in that enterally fed control animals receiving nutrient solution were not sham operated, harnessed, and attached to the infusion assembly;[27,29] if operated, harnessed, and attached to infusion assemblies, "controls" received a stock diet and parenterally infused animals were not directly compared in the same experiment with animals receiving the nutrient solution by mouth.[28] Findings were generally similar to our study, with decreases in disaccharidase specific activities, mucosal weight, protein, and DNA of similar or greater magnitude to our findings in enterally and parenterally fed groups.

Previous studies of disaccharidases in the orally fed diabetic rat have shown increased specific activities in diabetics at 5, 7, and 15 days after induction of alloxan diabetes,[2,3,5] and at 5 and 12 days of streptozotocin diabetes.[4,6] The reversal or depression of the increased disaccharidases in diabetes by insulin treatment,[4,30,31] is consistent with the disaccharidase response being the result of the diabetic state. The role of intraluminal as compared with extraluminal factors in the increased disaccharidase activity has been studied at 5 days in the streptozotocin-diabetic rat.[32] Thiry-Vella fistulas in diabetic rats showed increased sucrase specific activity in mucosa of fistula as well as mucosa of small intestine in continuity. It was concluded that the enhancement of sucrase activity in experimental diabetes is independent of intraluminal factors. However, the circulation and innervation of the fistula is intact. Hence, interactions of intraluminal factors with the mucosa could mediate the disaccharidase response in the fistula if transmitted in the blood or by the nerve supply. Thus, secretin doubles sucrase and maltase specific activity, whereas cholecystokinin has no effect.[33] Since both controls and diabetics were fed enterally, any differences in release of hormones, neurocrine, or paracrine factors would act on the fistula mucosa if they differed in controls and diabetics. Experiments described in this paper rule out a role for such factors.

Data regarding disaccharidase responses to diabetes mellitus in man are conflicting: increased activity was found in two studies,[34,35] but no response in a third study.[36] The differing findings may be related to severity of diabetes and effects of insulin treatment, since insulin depresses disaccharidases toward normal in man[35] and rat.[4,30,31] Thus, present knowledge of disaccharidase activity in human diabetics suggests considerable variability. α-Glycosidase inhibitors have the potential for reducing postprandial hyperglycemia after carbohydrate ingestion. Effectiveness and dose of α-glycosidase inhibitors in treatment of diabetes mellitus cannot be predicted from available data on disaccharidase activity in man.

ACKNOWLEDGMENTS

We thank Fran Chapman, Duane Miller, and Dave Robison for technical assistance. Cari Malone and Kathleen Funk provided secretarial assistance. Drs. Barry Ginsberg and Robert Thompson of the Diabetes Center of the University of Iowa reviewed the manuscript.

This study was supported in part by the Kroc Foundation and the Research Service of the Veterans Administration.

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