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Changes in Mucosal α-Glucosidase Activities along the Jejunal−Ileal Axis by an Hm-HACS Diet Intake Are Associated with Decreased Lipogenic Enzyme Activity in Epididymal Adipose Tissue

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Heat−moisture (hm)-high-amylose corn starch (HACS), which includes a larger amount of resistant starch than HACS or regular cornstarch (CS), is more indigestible in the small intestine than HACS or CS. An hm-HACS diet was also shown to ameliorate glucose intolerance and lipid abnormalities. This study examined the effects of feeding rats an hm-HACS diet for 14 days on the activities of mucosal α-glucosidase along the jejunal−ileal axis and lipogenic enzymes in epididymal adipose tissue. The contents in the lumen of the cecum were increased by feeding rats the HACS and hm-HACS diets, and the cecal weight was increased by the hm-HACS diet. The HACS diet reduced the activity of α-glucosidase in the upper jejunal mucosa, induced its activity in the upper ileal mucosa, reduced lipogenic enzyme activity in epididymal adipose tissue, and reduced serum triglyceride levels. These effects were more pronounced with the hm-HACS than with the HACS diet. These results suggest that feeding rats the hm-HACS diet reduced the activities of lipogenic enzymes in adipose tissue and α-glucosidase in the jejunal mucosa and induced the activity of α-glucosidase in the ileal mucosa compared with the HACS diet.

KEYWORDS: Hm-HACS; α-glucosidase; lipogenesis; small intestine; epididymal adipose

INTRODUCTION

Studies have demonstrated that delaying the digestion/absorption of carbohydrates in the small intestine has various beneficial effects for preventing/improving lifestyle-related diseases such as obesity and diabetes and their complications (*1*, *2*). This is partially because of decreased postprandial hyperglycemia and an improved bacterial environment in the intestine. In addition, these changes lead to an improvement in lipid abnormalities. High-amylose starches, which are more indigestible in the small intestine than regular high-amylopectin starch, are thought to mediate these beneficial effects (*1*). Indeed, many studies have demonstrated that the intake of high-amylose starch reduced fasting and postprandial levels of glucose, cholesterol, triglycerides, and insulin in the blood of model animals and humans (*3*−*6*). An earlier study of ours has demonstrated that the intake of high-amylose cornstarch (HACS) for 14 days decreased the activities of lipogenic enzymes such as fatty acid synthase (FAS), malic enzyme (ME), and glucose-6-phosphate dehydrogenase (G6PDH) in the liver and epididymal adipose tissue, as well as serum triglycerides levels (*7*). These findings are attributed to decreased blood glucose and insulin levels after meals containing high-amylose starch because this compound is indigestible in the small intestine, unlike high-amylopectin starch (*6*). In addition, the activity of α-glucosidase was decreased in the upper jejunal mucosa and increased in the upper ileal mucosa by HACS (*7*). This is associated with delaying carbohydrate digestion along the jejunal−ileal axis. Similar effects are elicited by acarbose, a drug that reduces postprandial hyperglycemia by inhibiting pancreatic α-amylase and mucosal α-glucosidase activities along the jejunal−ileal axis in normal and diabetic rats (*7*−*9*). These results suggest that changes in the activity of mucosal α-glucosidase along the jejunal−ileal axis caused by drugs or food components that are resistant to carbohydrate digestion/absorption can delay carbohydrate digestion in the small intestine.

Several recent studies have shown that heat−moisture (hm) treatment of high-amylose starch increased the amount of resistant starch, which is defined as the undigested starch along the jejunal−ileal axis (*10*, *11*). Indeed, it was reported that hm treatment decreased the digestion of high-amylose starch by pancreatic α-amylase in vitro (*12*, *13*). A study has demonstrated that the intake of hm-treated HACS (hm-HACS) reduced triglyceride content in the blood and epididymal fat pad more than regular high-amylose cornstarch (HACS) (*10*). Another study showed that the intake of hm-HACS reduced the blood cholesterol level and liver triglyceride content (*11*). Hm-HACS may decrease the activities of α-glucosidase in the jejunal mucosa and lipogenic enzymes in adipose tissue and increase the activity of α-glucosidase

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Table 1. Diet Composition

	CS	HACS	hm-HACS
α-Cornstarch (g)	55		
α-High-amylose cornstarch		55	
Heat−moisture α-high amylose cornstarch			55
Lard (g)	13	13	13
Corn oil (g)	2	2	2
Casein (g)	20	20	20
AIN^{76}-M mix (g)	3.5	3.5	3.5
AIN^{76}-V mix (g)	1	1	1
Choline bitartrate (g)	0.2	0.2	0.2
DL-methionine (g)	0.3	0.3	0.3
Cellulose (g)	5.0	5.0	5.0
Total (g)	100	100	100

in the ileal mucosa compared with HACS. This is because the substrate structures of hm-HACS are indigestible compared with regular starch and HACS, which ultimately affects the digestion rate of mucosal enzymes.

In this study, we examined whether feeding rats an hm-HACS diet altered blood parameters, the activities of mucosal α-glucosidases along the jejunal−ileal axis, or the activities of lipogenic enzymes in the liver and epididymal adipose tissue, as compared with regular CS and HACS diets.

MATERIALS AND METHODS

Materials. CS (α-type), an A-type crystalline starch containing approximately 25% amylose and approximately 75% amylopectin, was purchased from Oriental Yeast (Tokyo, Japan). HACS (α-type, A-type crystalline starch) (amylose, approximately 70%; amylopectin, approximately 30%) and hm-HACS (α-type, A-type crystalline starch), which was autoclaved at 130 °C for 2 h, were provided from Nihon Shokuhin Kako Co., Ltd. (Tokyo, Japan).

Single-Dose Starch Loading. Wistar male rats, at 7 weeks of age, were purchased from Japan SLC Inc. (Hamamatsu, Japan) and fed a diet of 55% CS (CS diet, **Table 1**) from 10:00 a.m. to 2:00 p.m. for 3 days in a temperature- and humidity-controlled room (23 °C, 53%) under a 12-h light/dark cycle (dark phase, from 6:00 p.m. to 6:00 a.m.). On day 4, the rats were given 3 g of the diets including 55% CS, HACS, and hm-HACS (**Table 1**), respectively. All rats consumed the diets within 5 min. Plasma was collected at 0, 20, 40, 70, and 120 min after feeding from the tail vein by a capillary containing heparin and lithium. The blood glucose concentrations were determined by the WAKO glucose test (Wako Pure Chemical Industries).

Fourteen-Day Dietary Protocol. Wistar male rats at 7 weeks of age received a diet containing 55% CS, HACS, or hm-HACS (6 rats/group). The compositions of each diet are shown in **Table 1**. The animals were allowed free access to food and water for 14 days. At the end of the feeding period, the animals were killed by decapitation between 10:00 a.m. and 11:00 a.m. Serum, the entire small intestine, liver, epididymal adipose tissue, and mesenteric adipose tissue were immediately collected. The experimental procedures used in the present study met the guidelines for animal usage of the committee of the University of Shizuoka.

Intestinal Mucosal α-Glucosidase Assays. The duodenum was discarded, and the jejunoileum was divided into four segments of equal length, namely, the upper jejunum, lower jejunum, upper ileum, and lower ileum. Each segment was flushed with ice-cold 0.9% sodium chloride, and the mucosa was scraped from each segment with a glass microscope slide. The intestinal mucosa was homogenized in 10 volumes (v/w) of ice-cold 10 mmol/L potassium phosphate buffer (pH 7.0). Sucrase, isomaltase, and maltase activities were assayed as described by Dahlqvist (*14*) using 28 mmol/L sucrose, palatinose, and maltose, respectively, as the substrates. The protein concentration was measured according to the method by Lowry et al. (*15*).

Lipogenic Enzyme Assays. Fresh liver and epididymal adipose tissues were subjected to the lipogenic enzyme assays. All tissue preparations were performed at 0−4 °C. Two grams of liver or 1 g of epididymal adipose tissue was homogenized in 2 volumes (v/w) of 0.1 M potassium phosphate buffer (pH 7.4) containing 0.25 M sucrose, 0.07 M $KHCO_3$, 1 mM EDTA, and 1 mM dithiothreitol. The homogenate was centrifuged at 8000*g* for 20 min at 4 °C, and the resulting postmitochondrial supernatant was centrifuged at 105000*g* for 60 min. The clear supernatant was removed without disturbing the pellet or the floating fat layer. Aliquots of the soluble supernatant (cytosol) were used to determine FAS activity. The remaining supernatant was kept at −20 °C until use. The activities of ME and G6PDH were evaluated within 2 days. Enzyme activity was assayed spectrophotometrically, as previously described (*7*), and expressed as nanomoles of NADPH produced or used per minute per milligram of cytosol protein.

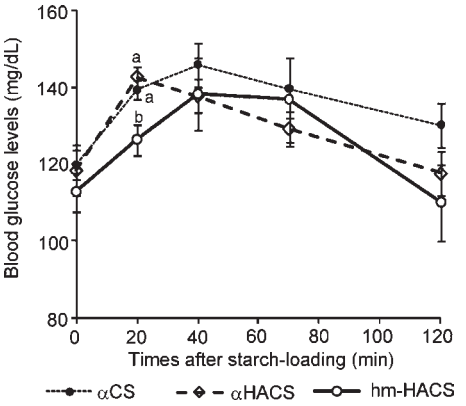

Figure 1. Effects of oral starch loading on plasma glucose. Values are means ± SEM for six animals. Values not sharing a common letter are significantly different ($P < 0.05$) from one another by Tukey's multiple-range test.

Other Assays. The serum triglyceride level was measured using an assay kit containing lipoprotein lipase, glycerol-3-phosphate oxidase, and glycerokinase (Triglyceride E-test, Wako Pure Chemical Industries). The serum total cholesterol level was measured using an assay kit containing cholesterol esterase and cholesterol oxidase (Cholesterol E-test, Wako Pure Chemical Industries).

Statistics. Results are expressed as means ± SEM. The significance of differences among groups was determined by Tukey's multiple-range test based on one-way ANOVA. A P value of < 0.05 was considered to indicate statistical significance.

RESULTS

Effects of a Single Dose of Starch on Normal Rats. To estimate the effects of hm-HACS on postprandial hyperglycemia, the plasma glucose concentrations were measured at 0, 20, 40, 70, and 120 min after starch loading. As shown in **Figure 1**, the plasma glucose level at 20 min after starch loading was lower in rats fed the hm-HACS diet than in those fed the HACS or CS diet ($P < 0.05$).

Effect of CS, HACS, and Hm-HACS on the Weight of the Liver and Adipose Tissue and Serum Triglyceride Levels. The average daily food intake over 14 days did not differ between the three groups of rats. No diarrhea was observed in any group. Similarly, body weight at the end of feeding period and the weight of the liver and adipose tissues did not differ between the three groups. However, fecal weight was significantly higher in the rats fed the hm-HACS diet than in the rats fed CS or HACS diets. Cecal content was higher in rats fed the hm-HACS group than in rats fed the CS or HACS diets and was higher in the group fed the HACS diet than in the group fed the CS diet. The serum triglyceride levels were significantly lower in the rats fed the HACS and hm-HACS diets than in the rats fed the CS diet (**Table 2**).

Effects of the HACS and Hm-HACS Diets on Mucosal α-Glucosidase Activity along the Jejunal−Ileal Axis. Next, we examined the effects of HACS or hm-HACS diet intake for 14 days on mucosal α-glucosidase activity along the jejunal−ileal axis in normal Wistar rats. As shown in **Figure 2**, feeding rats the HACS or hm-HACS

Table 2. Effects of Various Starches on Basic Parameters on Rats

	CS	HACS	hm-HACS
Body weight (g)			
Initial	144 ± 3	144 ± 3	143 ± 2
Final	191 ± 5	199 ± 3	197 ± 4
Weight gain (g/14 days)	47 ± 4	55 ± 3	53 ± 3
Food intake (g/day)	12.2 ± 0.53	11.8 ± 0.47	12.4 ± 0.35
Cecal weight (g/5 days)	5.8 ± 0.2 a	6.9 ± 0.7 a	22.9 ± 1.3 b
Cecal content (g)	2.97 ± 0.33 a	5.50 ± 1.06 b	9.92 ± 0.73 c
Liver	7.64 ± 0.30	7.54 ± 0.24	7.11 ± 0.24
Epididymal adipose tissue (g)	2.84 ± 0.19	2.44 ± 0.20	2.57 ± 0.08
Mesentery adipose tissue (g)	2.18 ± 0.14	2.04 ± 0.08	1.94 ± 0.11
Glucose (mg/100 mL)	146 ± 7	117 ± 7	115 ± 3
Triglycerides (mg/100 mL)	165 ± 15a	102 ± 10 b	84 ± 7 b
Cholesterol (mg/100 mL)	57.5 ± 3.1	54.5 ± 3.7	54.4 ± 2.6

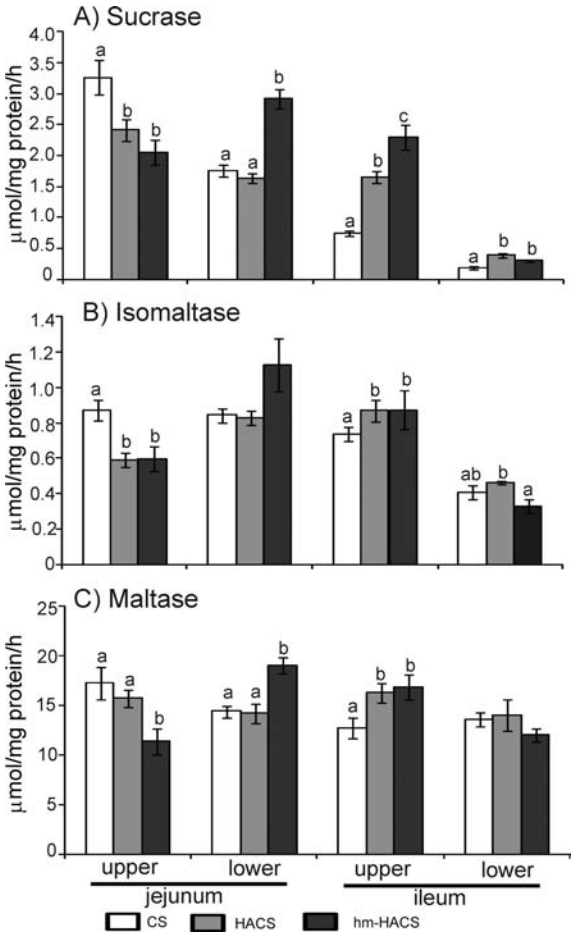

Figure 2. Activities of mucosal α-glucosidases along the jejunal−ileal axis in rats fed diets containing CS, HACS, or hm-HACS: (**A**) sucrase; (**B**) isomaltase; (**C**) maltase. Values are means ± SEM for six animals per group. Values not sharing a common letter are significantly different ($P < 0.05$) from one another by Tukey's multiple-range test.

diet for 14 days reduced sucrase, isomaltase, and maltase activities in the upper jejunal mucosa. By contrast, the activities of these mucosal α-glucosidases in the lower jejunum were enhanced by the hm-HACS diet. Similarly, the activity of these α-glucosidases in the upper ileal mucosa was induced by the HACS and hm-HACS diets. Overall, the hm-HACS diet had a more pronounced effect than the HACS diet on mucosal α-glucosidase activities along the jejunal−ileal axis, as compared with the CS diet.

Effects of the HACS and Hm-HACS Diets on the Activity of Lipogenic Enzymes in the Liver and Adipose Tissue. To determine whether delayed carbohydrate digestion associated with the HACS and hm-HACS diets affects lipid metabolism, we assessed the activity of lipogenic enzymes in the liver and epididymal adipose tissue. The rats fed the HACS and hm-HACS diets showed significantly reduced activities of ME and G6PDH in the liver (**Figure 3A**). Similarly, the rats fed the HACS and hm-HACS diets showed significantly reduced activities of ME and G6PDH in epididymal adipose tissue (**Figure 3B**). Taken together, the reduced activity of lipogenic enzymes in the liver and epididymal adipose tissue was greater in rats fed the hm-HACS diet than in rats fed the HACS or CS diets.

DISCUSSION

In this study, we have shown that feeding normal rats a HACS diet reduced the serum triglyceride level, but not the cholesterol level (**Table 2**), which is consistent with our previous study (*7*). One explanation for the lack of an effect of the HACS diet on serum cholesterol is that the diet used in this study did not include cholesterol. Indeed, it has been reported that feeding rats a HACS diet containing cholesterol, but not an HACS diet without cholesterol, reduced cholesterol levels in the blood (*3*, *7*). In addition, we have demonstrated that the activities of lipogenic enzymes in the liver and epididymal adipose tissue were reduced by feeding rats an HACS diet. We have also shown that the activities of mucosal α-glucosidases were reduced in the upper jejunum and increased in the upper ileum by the HACS diet, which is consistent with our previous results (*7*). The results of this and previous studies suggest that delaying carbohydrate digestion by feeding rats a HACS diet influences the activity of lipogenic enzymes in the liver and epididymal adipose tissue and the activities of mucosal α-glucosidases along the jejunal−ileal axis.

Recent studies have shown that hm treatment of HACS increased the content of resistant starch, which is defined as undigested starch along the jejunal−ileal axis (*10*, *11*). We found that the hm-HACS diet intake decreased the activity of α-glucosidase in the upper jejunal mucosa and lipogenic enzymes in the liver and adipose tissue and increased the activity of α-glucosidase in the upper ileal mucosa compared with regular HACS. One reason for this is that hm-HACS is more indigestible than HACS. Thus, we examined whether the hm-HACS diet intake affects the activities of lipogenic enzymes in the liver and epididymal adipose tissue or the activity of mucosal α-glucosidase along the jejunal−ileal axis, as well as blood parameters. As shown in **Table 2**, the serum triglyceride level tended to be lower in rats fed the hm-HACS diet for 14 days than in those fed the HACS diet. These results indicate that hm treatment of HACS reduced the digestion of carbohydrates and inhibited postprandial hyperglycemia. Also, intake of an hm-HACS diet for 14 days led to a reduction of triglycerides in the blood. Furthermore, the activities of lipogenic enzymes in the liver and epididymal adipose tissue were reduced by feeding rats the hm-HACS diet for 14 days rather than a HACS diet (**Figure 3**). It is known that the inhibition of postprandial hyperglycemia and hyperinsulinemia can reduce the activity of lipogenic enzymes in the liver and adipose tissue (*16*, *17*). Thus, the reduction in serum triglyceride level as a result of the hm-HACS diet may be due to the inhibition of postprandial hyperglycemia associated with the delay of carbohydrate digestion. In addition, in this study, we showed that the reduced activity of α-glucosidase in the upper jejunal mucosa and the enhanced activity of α-glucosidase in the upper ileal mucosa, relative to that in rats fed the CS diet, were more pronounced with the hm-HACS diet intake than with the HACS diet (**Figure 2**). Previous studies have demonstrated that treating diabetic rats with acarbose, a drug

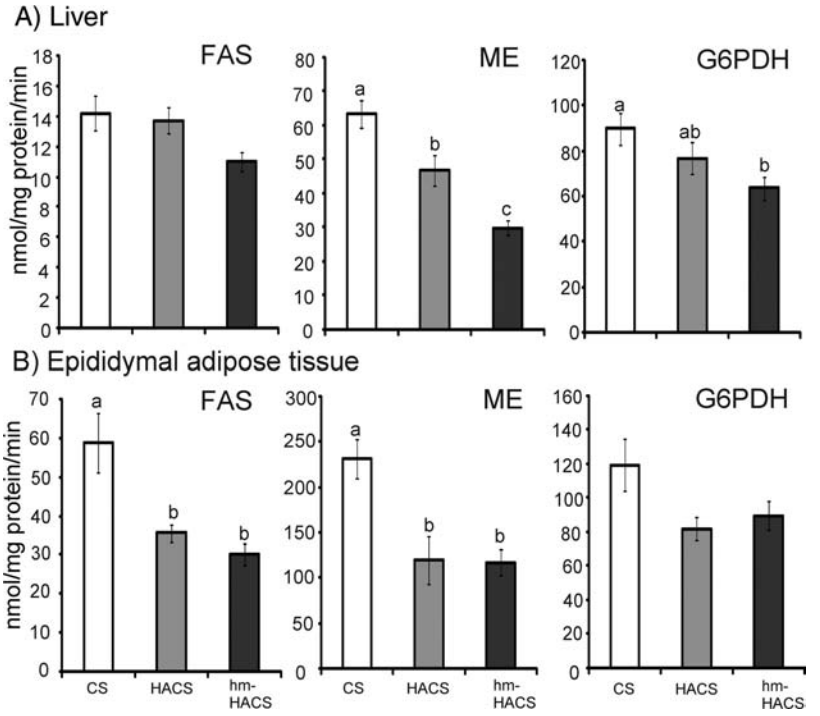

Figure 3. Effects of diets containing CS, HACS, or hm-HACS on the activity of lipogenic enzymes in rat liver and epididymal adipose tissue: (**A**) liver; (**B**) epididymal adipose tissue. Values are means ± SEM for six animals per group. Values not sharing a common letter are significantly different ($P < 0.05$) from one another by Tukey's multiple-range test.

that inhibits pancreatic α-amylase and mucosal α-glucosidase along the jejunal−ileal axis, for 84 days induced mucosal sucrase activity in the ileum, but not in the jejunum (*9*). Taking the results of our study together with those of the previous studies suggests that changes in α-glucosidase activity along the jejunal−ileal axis are dependent on the amount of carbohydrates that flows in the lumen of each segment to digest/absorb the carbohydrates and to avoid digestive symptoms. The shift in mucosal α-glucosidase activity from the upper jejunum to the ileum may lead to a decrease in postprandial hyperglycemia and a subsequent decrease in plasma triglyceride levels. Indeed, previous studies have demonstrated that dietary supplementation with acarbose reduced glucose incorporation in the upper jejunum (*18*). However, there are many possible reasons for the improvements associated with the hm-HACS and HACS diets. HACS and hm-HACS intake decreases postprandial hyperglycemia even if the activity of mucosal α-glucosidase along the jejunal−ileal axis remains unchanged because hm-HACS and HACS themselves are indigestible along the jejunal−ileal axis. The intake of hm-HACS and HACS also reduces total energy intake and improves the bacterial environment (*1*). Further studies should investigate whether the shift in activity of mucosal α-glucosidase from the upper jejunum to the ileum caused by hm-HACS and HACS reduces postprandial hyperglycemia in normal and diabetic animals.

It should be noted that cecal weight and cecal content were increased by the hm-HACS and HACS diets (**Table 2**). Thus, digestive symptoms such as abdominal fullness may be induced by the hm-HACS and HACS diets. Although the hm-HACS and HACS diets may have such adverse effects, carbohydrate flow to the cecum is reported to exert beneficial effects, such as improving the bacterial environment and increasing the secretion of glucagon-like peptide 1 (GLP1), which is a peptide hormone that reduces postprandial hyperglycemia by increasing insulin secretion and protects the pancreatic β-cells from apoptosis caused by excess insulin secretion (*1*, *2*, *19*). Thus, further studies should examine the dose-dependent effects of dietary hm-HACS and HACS on the activity of mucosal α-glucosidase along the jejunal−ileal axis and on lipogenic enzymes in adipose tissue, in addition to the effects on the bacterial environment, endogenous GLP1 secretion, and digestive symptoms.

In conclusion, this study has shown that feeding normal rats an hm-HACS diet reduces the activity of lipogenic enzymes in the liver and epididymal adipose tissue, reduces the activity of α-glucosidase in the jejunal mucosa, and increases the activity of α-glucosidase in the ileal mucosa along the jejunal−ileal axis to a greater extent than HACS.

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