Monostearoylglycerol-Starch Complex: Its Digestibility and Effects on Glycemic and Lipogenic Responses

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Summary We examined whether a modification of a starch into an α-amylase resistant form can lead to a reduction of postprandial glucose and insulin responses, and consequently to a change of lipid metabolism in liver and adipose tissue. For this purpose, a processed starch was prepared using a cornstarch (70% amylose and 30% amylpectin) and monoacylglycerol (monostearate; MS), forming monostearate-starch complex (MS-treated cornstarch). When we determined in vitro hydrolysis of MS-treated cornstarch using α-amylase and intestinal microvillar α-glucosidases, the glucose production rate of the MS-treated cornstarch was slower than the non-treated cornstarch. Measurement of a transmural potential difference (ΔPD) evoked by the MS-treated cornstarch in everted rat jejunum showed that the absorption rate of glucose released from the MS-treated cornstarch was also remarkably slower than that from the non-treated cornstarch. The postprandial plasma insulin response to the MS-treated cornstarch was reduced, although plasma glucose response was unchanged. In a feeding study, two groups of five or six male Wistar-strain rats were fed defined diets containing 61.1% MS-treated cornstarch or 58.2% non-treated cornstarch ad libitum for 14 days. Food intakes during the period were similar between the two groups. Feeding the MS-treated cornstarch resulted in a significantly lower maltase activity in upper jejunum than did the non-treated cornstarch feeding. The activities of lipogenic enzymes—fatty acid synthetase (FAS), malic enzyme (ME), and glucose-6-phosphate dehydrogenase (G-6-PDH)—significantly decreased in epididymal adipose tissue of rats fed the MS-treated cornstarch. In the liver, FAS activity was lower in the MS-treated cornstarch group. The results indicated that MS-treated cornstarch was digested less rapidly, and lowered blood insulin response, consequently leading to a declined lipogenesis of adipose tissue and liver. This study suggests that the rate of intestinal hydrolysis of starch is an important determinant of metabolic responses such as glycemic and
lipogenic responses to diets.

**Key Words** monostearate-treated cornstarch, lipid-starch complex, glucose, insulin, disaccharidase, lipogenic enzymes

Structure of dietary carbohydrate has been recognized as an important element in dietary therapy for diabetic and hyperlipidemic individuals. A number of studies have found the differences in postprandial glucose and insulin responses to different types of starch in foods (1–3). The study using baked potato, boiled white rice, kernel corn, and white bread revealed that the rice used had the flattened glycemic response pattern, but the potato showed a blood glucose response not significantly different from that of an equivalent amount of glucose (4). The differences in glycemic response to rice compared to potato were even greater in diabetics with impaired glucose tolerance (5, 6). Likewise, a study comparing rices with high and low amylose content found that the ingestion of rice containing amylose resulted in lower and flatter glycemic response patterns than rice containing no amylose (7). These observations suggest that the structure of dietary carbohydrate is important in determining postprandial glycemic responses.

The importance of food form to glycemic responses is also noted in a number of recent studies (8). The rice which was ground into flour showed a higher glycemic response than that caused by the whole rice. With white flour in the form of spaghetti, the blood glucose levels rose much less than when the same amount of white flour was given in the form of bread to human subjects (9). These findings suggest that the food form is a determinant of the rate at which the starch is hydrolyzed.

On the other hand, comprehensive studies have shown that high carbohydrate feeding elevated lipogenesis and that insulin was involved in the induction of lipogenic enzymes (10–13). The increased lipogenic enzyme activities with a high carbohydrate diet was mediated by hyperinsulinemia (11). Plasma insulin response to sucrose feeding is greater than to starch feeding (8), and sucrose-fed rats had greater lipogenic activity than starch-fed rats (13). Inhibition of carbohydrate absorption by $\alpha$-glucosidase inhibitor (acarbose) caused a delay in energy accumulation during refeeding of rats, both in glycogen stores, and more markedly, in adipose tissue triglyceride stores (14).

Taken together, it is clear that delaying glycemic response to dietary carbohydrates is important in attempt to prevent hyperlipidemia, obesity, diabetes, and disordered lipid metabolism. Factors affecting glycemic and insulin responses are considered to be food form, meal digestion rate, starch structure, and other factors. If a processed starch which is resistant to $\alpha$-amylase, is introduced as a dietary starch, it would provide the same benefits as that shown by the high-amylose diet and certain food forms like spaghetti (9). We designed to determine whether a modification of a starch into an $\alpha$-amylase resistant form can lead to a reduction of postprandial increases of plasma glucose and insulin, and consequently to a change.
of lipid metabolism in liver and adipose tissue. For this purpose, we prepared a processed cornstarch using monoacylglycerol (monostearate, MS) which is supposed to interact with amylose to form an monoacylglycerol-starch complex. The results in both from in vitro and in vivo studies provide an evidence that the processed cornstarch (MS-treated cornstarch) could delay the carbohydrate digestion and absorption, and reduce glycemic and insulin responses, leading to a decline of lipogenesis in epididymal adipose tissue as well as liver.

**MATERIALS AND METHODS**

**Experimental design.** In Experiment 1, digestion and absorption of the MS-treated cornstarch were evaluated in vitro by measuring the hydrolysis of the starches by α-amylase plus jejunal microvillar α-glucosidases and the transmural potential difference (ΔPD) evoked by starch in everted rat jejunum. In Experiment 2, the postprandial glucose and insulin responses to the MS-treated cornstarch were evaluated. In Experiment 3, we determined the effects of MS-treated cornstarch feeding on disaccharidase activities in the small intestines, and on lipogenic enzyme activities in the liver and epididymal adipose tissue in rats.

**Materials.** The processed starch was prepared by mixing a high-amylose cornstarch (70% amylose and 30% amylopectin, Honen Corporation, Tokyo) suspension in water with monostearate (Honen Corporation) at a weight ratio of 20:1, followed by heating and freeze-drying. Control starch was prepared by heating the high-amylose cornstarch suspension at the same condition as used for the MS-treated cornstarch and by freeze-drying. Porcine pancreas α-amylase (Type I-A) was purchased from Sigma Chemical Co., U.S.A.

**In vitro assay of digestibility of the starches.** Digestibility of MS-treated cornstarch and non-treated cornstarch were determined using α-amylase and microvillar membranes of rat small intestine. Microvillar membranes were prepared from the jejunum of rats according to the method of Kessler et al. (15). To prevent a possible attachment of pancreatic α-amylase in the microvillar membranes, these were subjected to pancreatico-biliary duct occlusion 18 h prior to killing rats according to the procedure described previously (16). One hundred microliters of 1 and 0.2% suspension in PBS of non-treated cornstarch or MS-treated cornstarch were incubated, at 37°C for 15, 30, 45, and 60 min with 50 μl of α-amylase (0, 0.05, and 0.5 μg/ml in PBS) and 50 μl of the brush border membranes, which contained maltase activity of 398 μmol-substrate hydrolyzed/h. The reaction mixture at the end of each incubation time was added by 200 μl of the Tris-glucose oxidase reagent (17) and again incubated at 37°C for 30 min, and the reactions were terminated with 400 μl of 66% H₂SO₄. The hydrolysis activity was expressed as nmol glucose produced from duplicate assays. This in vitro experiment was repeated.

**Measurement of transmural potential difference.** To compare the absorption rate of glucose released from the starches, transmural potential difference was
determined, as described by Himukai and Hoshi (18). Male rats of Wistar strain weighing about 280 g were used, and these animals were given a standard laboratory diet (MF, Oriental Yeast Co.) and water ad libitum. Under anesthesia by an intraperitoneal injection of sodium pentobarbital, a jejunal segment of 2 cm in length was obtained from a portion between 15 and 30 cm distal to the ligament of Treitz, before being everted and fixed over a small fenestrated polyethylene tube (diameter, 5 mm). The changes in transmural potential difference (ΔPD) induced by the co-transport of Na⁺ and glucose were recorded. The serosal and mucosal solutions consisted of 50 mM Na₂SO₄, 160 mM mannitol, 2.5 mM KHCO₃, 0.25 mM KH₂PO₄, 1.5 mM CaSO₄, and 1.0 mM MgSO₄ (pH 7.4). The solution on the serosal and mucosal sides were connected to a calomel electrode via 1 M KCl agar bridges and to a high sensitive direct-current potentiometer (Hitachi, 056-1001). First, we confirmed that the increment of transmural potential (ΔPD) was evoked by introducing 1.0 ml of a 200 mM glucose solution into 9 ml of mucosal-side medium (final concentration, 20 mM). Then, the mucosal-side medium was discarded by aspirating and newly added the same buffer. After the transmural potential had reached a steady state, α-amylase solution was added into medium to make 4 μg/ml of α-amylase (a porcine α-amylase, Type I-A), followed by adding the starch samples into the mucosal-side medium (final concentration, 0.1% of either starch). The samples of the MS-treated cornstarch or non-treated cornstarch were dispersed in the medium buffer. The changes in the transmural potential difference (ΔPD) were recorded on the same recording paper and compared between the two starches.

Measurement of postprandial glucose and insulin levels. Wistar-strain male rats weighing 110–130 g, which were fed ad libitum a laboratory chow diet (MF, Oriental Yeast Co.), were fasted for 3 h from 9:00 A.M. The animals were intubated into a stomach with 3.1 ml of 10% starch suspension per 100 g body weight; the non-treated cornstarch (control diet) and MS-treated cornstarch (experimental diet), respectively. These starches were the same as the starches used in the Experiment 3. Rats were lightly anesthetized by ether vapor and blood samples were collected from tail tip into the heparinized capillary tube (Terumo Co., Tokyo). Blood samples were withdrawn from each rat prior to starch loading and at 15, 30, 45, 60, and 90 min following the starch intubation. Plasma glucose was determined by the glucose oxidase method using an assay kit (Glucose B-test, Wako Pure Chemical Industries, Osaka). Plasma insulin was measured by the immunoassay using a kit (Insulin-EIA test, Sanyo-Kasei Industries, Tokyo).

Animals and diets. In Experiment 3, male wistar-strain rats weighing 170 g (Japan SLC Inc., Hamamatsu) were housed in individual wire cages in a temperature- and humidity-controlled room (23°C, 53%). The control group of 5 rats received a diet containing the non-treated cornstarch (control diet). The experimental group of 7 rats received a diet containing the MS-treated cornstarch (MS-treated cornstarch diet). The details of diet composition are shown in Table 1. The animals were allowed to receive free access to the diets and water for 14
Table 1. Composition of diets.

| Ingredient                      | Non-treated cornstarch (control) | MS-treated cornstarch |
|---------------------------------|----------------------------------|-----------------------|
| α-Cornstarch<sup>1</sup>        | 58.2                             | —                     |
| MS-treated cornstarch<sup>2</sup>| —                                | 61.1                  |
| Vitamin-free casein             | 24.9                             | 24.9                  |
| Corn oil                        | 5.0                              | 5.0                   |
| Cellulose<sup>3</sup>           | 7.9                              | 7.9                   |
| AIN<sup>76</sup> mineral mixture| 2.8                              | 2.8                   |
| AIN<sup>76</sup> vitamin mixture| 0.8                              | 0.8                   |
| DL-Methionine                   | 0.24                             | 0.24                  |
| Choline bitartrate              | 0.16                             | 0.16                  |

<sup>1</sup>α-Cornstarch was purchased from Honen Corporation, Tokyo. <sup>2</sup>A processed cornstarch with monostearate/cornstarch complex (see MATERIALS AND METHODS). The two diets contained identical amount of starch. <sup>3</sup>Cellulose powder “D” (Toyo Roshi Co., Tokyo).

days. At the end of feeding period, the animals were killed by decapitation and the tissue samples of intestine, liver, and epididymal fat pad (epididymal adipose tissue) were quickly collected. After these tissues were washed with ice-cold saline and blotted with a tissue paper, the tissues were weighed. Blood samples were collected and separated by centrifugation at 3,000 rpm for 15 min.

**Intestinal disaccharidase assay.** The duodenum segment of the small intestine was discarded and the rest of the intestine was divided into three segments of equal length, referred to as upper jejunum, lower jejunum, and ileum, respectively. After each segment was flushed with ice-cold saline, mucosa was scraped from each segment with a glass microscope slide. Intestinal mucosa was weighed and the mucosa was homogenized in 10 volumes (v/w) of ice-cold 10 mM potassium phosphate buffer (pH 7.0). The mucosa homogenate was kept at −20℃ until use for maltase and isomaltase assays. Maltase and isomaltase activities were determined according to the procedure described by Dahlqvist (17), with 28 mM-maltose and 28 mM-palatinose as substrate, respectively. Protein was measured by the method of Lowry et al. (19) using bovine serum albumin as a standard.

**Lipogenic enzyme assay.** The fresh tissues of liver and epididymal fat pad were prepared for the lipogenic enzyme assay. All tissue preparation procedures were done at 0–4℃. Liver (2 g) or epididymal fat pad (1 g) was homogenized in two volumes (v/w) of 0.1 M potassium phosphate buffer (pH 7.4) containing 0.25 M sucrose, 0.07 M KHCO<sub>3</sub>, 1 mM EDTA, and 1 mM dithiothreitol. After centrifugation of the homogenate at 8,000×g for 20 min, the resulting postmitochondrial supernatant was centrifuged at 105,000×g for 60 min. The clear supernatant was removed with care being taken not to disturb the pellet or the floating fat layer. Aliquot of the soluble supernatant was used to determine the fatty acid synthetase (FAS) activity and the remaining supernatant was kept at −20℃ until use. Determination of malic enzyme (ME) and glucose-6-phosphate dehydrogenase (G-
6-PDH) activities were carried out within 2 days. The FAS, ME, and G-6-PDH activities were determined spectrophotometrically as previously described (20). The activities were expressed as micromoles of NADPH produced or decreased per minute per total tissue per 100 g body weight.

**Other assays.** Serum samples were used for measuring the concentrations of glucose and insulin. Serum glucose was determined by the glucose oxidase method using an assay kit (Glucose B-test, Wako Pure Chemical Industries, Osaka). Serum insulin was measured by immunoassay using a kit (Insulin-EIA test, Sanyo-Kasei Industries, Tokyo).

**Statistics.** The results were subjected to ANOVA analysis. Furthermore, difference in mean values between the two groups was determined by *t*-test analysis at *p* < 0.05.

## RESULTS

**Degree of in vitro hydrolysis of MS-treated cornstarch**

In the absence of α-amylase, incubation of both starches, the MS-treated cornstarch and non-treated cornstarch, with rat brush border membranes produced little glucose during 60 min incubation (Fig. 1). In the presence of 0.0125 and 0.125% α-amylase, the MS-treated cornstarch was hydrolyzed to produce glucose, but the rate of glucose production was slower than non-treated cornstarch (Fig. 1), indicating that the MS-treated cornstarch was resistant to α-amylase tested.

**Transmural potential difference induced by MS-treated cornstarch**

The transmural potential differences (ΔPD) induced by the MS-treated cornstarch in the everted jejunal segment of rats were less than that induced by the non-treated cornstarch (control starch). The difference of the ΔPD values between the two starches became larger as the incubation time was longer (Fig. 2). Thus, it was evident that the hydrolysis/absorption rate of the MS-treated cornstarch in the jejunum was lower than that of non-treated cornstarch.

**Postprandial glucose and insulin levels of MS-treated cornstarch**

The overall serum glucose and insulin responses to the control and MS-treated starch were determined by calculating the areas under the curves of glucose and insulin for the entire test period (90 min) by the formula. The overall glucose response to the MS-treated cornstarch was not different from that of the control cornstarch. However, the overall insulin response to the MS-treated cornstarch was significantly lower (*p* < 0.05) than the response of the control starch (Fig. 3).

**Animals in the feeding study**

The weight gain of animals during the experimental period (Experiment 3) showed no difference between the two groups (Table 2). The total food intake during feeding period of 14 days was the same in the MS-treated cornstarch group.
Fig. 1. In vitro hydrolysis of the MS-treated cornstarch and the non-treated cornstarch. In vitro determinations of digestibility of the MS-treated cornstarch and the non-treated cornstarch were carried out using α-amylase (porcine α-amylase) and α-glucosidase (jejunal microvilli of rats) as described in MATERIALS AND METHODS. The open symbols of circles, triangles, and squares represent the MS-treated cornstarch and the closed symbols represent the non-treated cornstarch. The solid lines represent data of incubations with no α-amylase. The dot lines represent data of incubations with 0.0125 μg/ml of α-amylase. The broken lines represent data of incubations with 0.125 μg/ml of α-amylase. The data were expressed as average amounts of glucose produced per an incubation tube, as duplicate assays were done.

as in the control group (Table 2). The weight of epididymal adipose tissue was not different statistically between rats fed the MS-treated cornstarch diet and the control diet (data not shown).

The blood samples collected at the end of the experimental period showed that the serum glucose levels were not different between the two starch groups, but the serum insulin level in the MS-treated cornstarch group tended to be reduced relative to the control group, although the difference was not statistically significant (Table 2).

**Effect of feeding MS-treated cornstarch on small intestinal disaccharidase activities**

The maltase and isomaltase activities in the mucosal homogenates of the three small intestinal segments of rats in Expreiment 3 are shown in Table 3. In the all segments, mucosal weight and mucosal protein contents were unaffected by feeding the MS-treated cornstarch. The maltase activity in the upper jejunal segment of
Fig. 2. Transmural potential difference (ΔPD) evoked by the MS-treated cornstarch and the non-treated cornstarch. In vitro absorption rate of the MS-treated and non-treated cornstarches were measured as ΔPD (mV). The solid line represents the ΔPD evoked by the 0.1% non-treated cornstarch and the broken line represents the ΔPD evoked by the 0.1% MS-treated cornstarch.

Fig. 3. Summed levels of postprandial plasma glucose and insulin in response to the MS-treated cornstarch and the non-treated cornstarch. Each bar represents the M±SEM of five rats. *denotes significant difference from the control group (non-treated cornstarch) at p<0.05 according to t-test analysis.

Rats fed the MS-treated cornstarch diet was lower than that of rats fed the control diet in spite of similar food intake. In the lower jejunum and ileum, the MS-treated cornstarch group exhibited a higher maltase activity than that of the control group. These findings indicate an altered distribution of the maltase. The isomaltase activities in the three segments of small intestine of rats fed the MS-treated

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Table 2. Body weight gain, food intake, and serum concentrations of glucose and insulin of rats fed non-treated and MS-treated cornstarch.

| Group (No. of rats) | Non-treated cornstarch (5) | MS-treated cornstarch (7) |
|---------------------|----------------------------|--------------------------|
| Final body weight (g) | 151±3                      | 154±2                    |
| Body weight gain (g/14 days) | 44±3                      | 47±3                     |
| Food intake (g/14 days) | 163.0±9.4                  | 160.1±4.1                |
| Serum glucose (mg/dl) | 120±6                      | 126±5                    |
| Serum insulin (μU/ml) | 45.0±8.2                   | 34.8±6.6                 |

Data are expressed as M±SEM. Serum samples were collected around 11:00 A.M. when animals were killed at the end of feeding period.

Table 3. Effects of feeding MS-treated cornstarch on maltase and isomaltase activities in rat small intestine.

| Group (No. of rats) | Non-treated cornstarch (5) | MS-treated cornstarch (7) |
|---------------------|----------------------------|--------------------------|
| Upper jejunum       |                            |                          |
| Maltase             | 33.8±0.3                   | 31.2±0.7*                |
| Isomaltase          | 0.55±0.01                  | 0.52±0.01                |
| Lower jejunum       |                            |                          |
| Maltase             | 37.6±1.9                   | 40.5±0.8*                |
| Isomaltase          | 0.73±0.03                  | 0.84±0.01*               |
| Ileum               |                            |                          |
| Maltase             | 15.5±0.9                   | 18.9±0.9*                |
| Isomaltase          | 0.27±0.03                  | 0.30±0.02                |

Data are expressed as M±SEM. * denotes significant difference from the control group (non-treated cornstarch) at p<0.05 according to t-test analysis.

cornstarch diet also exhibited similar tendency showing significant higher activity in lower jejunum than that of rats fed the control diet.

Effect of feeding the MS-treated cornstarch on lipogenic enzyme activities in liver and adipose tissue

To examine whether the delay of carbohydrate digestion caused by the MS-treated cornstarch feeding would influence lipid metabolism, the lipogenic enzyme activities in liver and epididymal adipose tissue of rats in Experiment 3 were determined (Table 4). The MS-treated cornstarch feeding brought about low activities of FAS, ME, and G-6-PDH in the epididymal adipose tissue. In the livers of rats fed the MS-treated cornstarch diet, only the FAS activity significantly decreased, and G-6-PDH activity tended to decrease although its level was not
Table 4. Effects of feeding MS-treated cornstarch on lipogenic enzyme activities in liver and epididymal adipose tissue of rats.

|                      | Group (No. of rats) |                      |                      |
|----------------------|---------------------|----------------------|----------------------|
|                      | Non-treated cornstarch (5) | MS-treated cornstarch (7) |                      |
|                      | (μmol/min/total tissue/100 g body weight) |                      |                      |
| Liver                |                      |                      |                      |
| FAS                  | 4.2 ± 0.3           | 3.4 ± 0.1*           |
| ME                   | 16.2 ± 0.8          | 14.8 ± 0.1           |
| G-6-PDH              | 38.2 ± 4.2          | 29.4 ± 1.3           |
| Epididymal adipose tissue |                      |                      |                      |
| FAS                  | 0.25 ± 0.04         | 0.13 ± 0.02*         |
| ME                   | 2.61 ± 0.60         | 1.38 ± 0.09*         |
| G-6-PDH              | 0.90 ± 0.21         | 0.33 ± 0.03*         |

Data are expressed as M±SEM. * denotes significant difference from the control group (non-treated cornstarch) at p < 0.05 according to t-test analysis.

The hepatic ME activity was similar to that of rats fed the control diet.

DISCUSSION

The present studies demonstrated that the food form of lipid-starch complex could delay digestion and absorption of its starch constituents, and resulted in a decrease of lipogenesis in the epididymal adipose tissue and liver. Indeed, in vitro studies clearly demonstrated a significant delay of the hydrolysis of the MS-treated cornstarch and the consequent glucose absorption. The hydrolysis of the MS-treated cornstarch was slower than the non-treated cornstarch (control starch) (Fig. 1). The transmural potential difference (JPD) induced by the MS-treated cornstarch in the jejunal segment of rats was less than that induced by the non-treated cornstarch (Fig. 2). Feeding the MS-treated cornstarch diet led to a decreased maltase activity in upper jejunum and to elevated maltase activity in the lower jejunum and ileum (Table 3), suggesting that digestibility of the ingested MS-treated cornstarch in the upper jejunum segment was so low that more amounts of undigested MS-treated cornstarch were transferred to the lower jejunum and ileum parts compared to the control starch. These effects of processing of cornstarch with monostearate digestion and absorption of starch might indicate that the MS-treated cornstarch was possibly in an \( \alpha \)-amylase resistant form in the test diet. In this regard, it was reported that postprandial blood glucose response was reduced, when saturated fat (butter) was ingested with carbohydrate (21). Also, addition of protein to a carbohydrate was reported to decrease its glycemic response (22), possibly because of protein/carbohydrate interaction.

The findings in respect of the low enzymatic hydrolysis (Fig. 1) and the less
The ΔPD value (Fig. 2) of the MS-treated cornstarch may be related to a reduction in the postprandial serum glucose and insulin responses, when rats were given the MS-treated cornstarch. The overall insulin response to the MS-treated cornstarch was significantly lower (Fig. 3). Indeed, the serum insulin level in rats fed for 14 days the MS-treated cornstarch diet also tended to be lower than the control group, although the glucose levels were not different between the two starch groups (Table 2).

In intestinal lumen, amylose is converted into polymers up to 9 glucose units by the pancreatic α-amylase action. Amylopectin is broken down into branched segments of 5 to 9 glucose units. In turn, α-1,4 linkages of these polymers of both amylose and amylopectin are hydrolyzed by the action of α-glucosidases including maltase and isomaltase located in the mucosal brush border membranes (23). In addition, isomaltase attacks α-1,6 linkages of the branched segments (24). Namely, isomaltase attacks amylopectin fragments. In the present study, the control diet as well as the MS-treated cornstarch diet contained the same amount of amylopectin (30%). Besides, the isomaltase activity in the upper jejunum and ileum was similar between the two groups, although the maltase activity was remarkably influenced by feeding the MS-treated cornstarch diet. Taking together this information, we assumed that the MS-treated cornstarch may be resistant to α-amylase on its amylose fragment. However, the mechanism by which the interaction of monoaacylglycerol (monostearate) with amylose causes a decrease of susceptibility of starch to α-amylase is unclear at present.

In this study, feeding the MS-treated cornstarch effectively reduced the activities of FAS, ME, and G-6-PDH in adipose tissue, and reduced the FAS activity in liver (Table 4). Because of high amounts of starches used as a carbohydrate source in the test diets, these enzyme activities observed in this study were relatively low compared to the sucrose feeding (25). The reduction of the lipogenic enzyme activities caused by feeding the MS-treated cornstarch might be related with the delay of digestion and absorption of dietary carbohydrate and reduced postprandial insulin response to the diet (Fig. 3).

It is well known that high-carbohydrate feeding associates with remarkably elevated insulin-stimulated glucose uptake and lipogenic metabolism in the adipose tissue (26) and liver (10,27). The effects of insulin on lipogenesis in adipose tissues as well as liver are considered to be a function of the plasma insulin concentration and the insulin receptor concentration in the target cells. The enhancement in lipogenic metabolism after high-carbohydrate feeding is conceivably a consequence of elevated plasma concentrations of glucose and insulin. It is also known that high carbohydrate intake induces the lipogenic capacity of liver and adipose tissue by increasing lipogenic enzyme synthesis (28). The induction of the lipogenic enzymes involves direct carbohydrate control of gene transcription (29), mRNA processing (30), and mRNA stability (31). This induction of mRNA for lipogenic enzymes was paralleled in a rise in enzyme activities in adipose tissue and liver (32, 33).

The results obtained from the present study strongly suggest that the use of the
carbohydrate form with lipid/starch interaction such as MS-treated cornstarch might be effective in delaying digestion and absorption of carbohydrate, and in reducing postprandial blood insulin response, leading to a reduction of lipogenesis in adipose tissue and liver.

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