Effect of nutritional counseling and long term isomaltulose based liquid formula (MHN-01) intake on metabolic syndrome

Eiji Takeda,1,*, Hisami Yamanaka-Okumura,1 Yutaka Taketani,1 Nobuya Inagaki,2 Masaya Hosokawa,2 Kenichiro Shide,3 Hiroshi Maegawa,4 Keiko Kondo,4 Eiji Kawasaki,5 Shoko Shinozaki,5 Yuichi Fujinaka,7 Tsukasa Matsubara,8 Taka’fumi Katayama,9 Hajime Sasaki,10,* Akihiro Kawashima10 and Hiromitsu Aonuma10

1Department of Clinical Nutrition, Institute of Health Biosciences, University of Tokushima Graduate School, 3-18-15 Kuramoto-cho, Tokushima 770-8503, Japan
2Department of Diabetes, Endocrinology and Nutrition, Kyoto University Graduate School of Medicine and 3Department of Clinical Nutrition, School of Medicine, Kyoto University, Yoshida-Konoe-cho, Sakyo-ku, Kyoto 606-8501, Japan
3Division of Endocrinology and Metabolism, Department of Medicine, Shiga University of Medical Science, Seto Tsukinowa-cho, Otsu, Shiga 520-2192, Japan
4Department of Metabolism/Diabetes and Clinical Nutrition and 5Division of Dietary Service, Nagasaki University Hospital, 1-7-1 Sakamoto, Nagasaki 852-8501, Japan
6Endocrinology and Metabolism, Tokushima University Medical and Dental Hospital, 2-50-1 Kuramoto-cho, Tokushima 770-8503, Japan
7Director, Matsubara Mayflower Hospital, 944-55 Fujita, Kato, Hyogo 673-1462, Japan
8Faculty of Statistic and Computer Science, College of Nursing Art and Science, University of Hyogo, 13-71 Kitaioji-cho, Akashi, Hyogo 673-8588, Japan
9Food Science Research Labs., R&D Div., Meiji Co., Ltd., 540 Narita, Odawara, Kanagawa 250-0862, Japan

The isomaltulose based liquid formula (MHN-01), suppresses postprandial plasma glucose and insulin levels in healthy persons and patients with impaired glucose tolerance (IGT) or type 2 diabetes. MHN-01 intake as a part of breakfast also suppresses glucose and insulin levels after lunch, suggesting second meal effect. The objective of this study was to investigate the effects of nutritional counseling and long-term (24 weeks) MHN-01 ingestion on biomarkers of metabolic syndrome. Forty-one subjects with criteria of metabolic syndrome participated in this study composed with the control period (0–12 week) followed by nutritional counseling and the experimental period (12–36 week) followed by 200 kcal (837 kJ) of MHN-01 or dextrin-based standard balanced liquid formula (SBF) loading as a part of breakfast. In 16 of 41 subjects became to out of criteria for liquid formula loading study during control period (unqualified group). In the unqualified group, several biomarkers were improved. In experimental period, serum HbA1c levels significantly increased in SBF group (n = 12) but did not change in MHN-01 group (n = 10). Thus, intake of 837 kJ MHN-01 as a part of breakfast may be effective for suppression of deteriorating glucose metabolism in metabolic syndrome.

Key Words: postprandial hyperglycemia, insulin, diabetes, impaired glucose tolerance

Abdominal obesity is frequently associated with a collection of metabolic disorders that include elevated blood pressure, characteristic lipid abnormalities (low high-density lipoprotein cholesterol and high triglycerides) and increased fasting glucose, with an underlying situation of insulin resistance, which has been defined as metabolic syndrome, conferring a high cardiovascular risk profile to these subjects. A multidisciplinary approach, including lifestyle changes and pharmacological and surgical approaches is required for prevention and treatment of metabolic syndrome.

Anti-hyperglycemic therapy focused on control of postprandial glucose level has a greater impact on overall metabolic control, and thus improves long-term outcome compared with the more traditional approaches focused on fasting glucose level.10 Cohort studies have shown that postprandial hyperglycemia is an independent risk factor for cardiovascular disease.2-4 In the STOP-NIDDM study, correction of postprandial hyperglycemia reduced the onset of myocardial infarction.5 Dietary carbohydrates influence insulin secretion, postprandial plasma glucose, and plasma lipid profile.6 The glycemic index (GI) was proposed as a system for classifying carbohydrate-containing foods according to glycemic response.7 Another point concerning the deterioration of the glycemic profiles is the significant increase in glucose levels during the morning periods in type 2 diabetic patients due to an overproduction of glucose by the liver.8,9 These metabolic disturbances known as “dawn phenomenon”9 is observed at prebreakfast time, but have a prolonged deleterious effect on glucose levels over the entire postbreakfast period.

Ingestion of isomaltulose by type 2 diabetic humans and rats resulted in a reduction in their postprandial plasma glucose and insulin levels.10,11 In our previous studies, the isomaltulose based liquid formula (MHN-01) containing isomaltulose and oleic acid suppresses postprandial hyperglycemia and hyperinsulinemia in humans and Sprague-Dawley rats, reduces visceral fat accumulation, and improves insulin sensitivity in Sprague-Dawley rats.12-14 Consumption of MHN-01 at breakfast also appeared to improve glycemic control by reducing postprandial plasma glucose and insulin levels after lunch (second meal effect) in healthy men.15 However, it is not clear that the effect of continuous MHN-01 intake at breakfast improves glucose metabolism in metabolic syndrome. This prospective, multicenter, blind and randomized control study was designed to investigate the effects of long-term MHN-01 ingestion as a part of breakfast on glycemic control and body composition in metabolic syndrome.

Materials and Methods

Subjects. Individuals were eligible to participate if they met the following inclusion criteria. In the control and the experimental period of this study, they had more than 25 kg/m² of body mass index (BMI), 100–125 mg/dl of fasting plasma glucose (FPG), 5.2–6.5% of hemoglobin A1c (HbA1c) and were between

*To whom correspondence should be addressed.
E-mail: takeda.eiji@tokushima-u.ac.jp
†Present address: Department of Nutrition and Life Sciences, Kanagawa Institute of Technology, 1030 Shimo-ogino, Atsugi, Kanagawa 243-0292, Japan

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the ages of 20 and 70 years. This study was carried out at 5 hospitals (Kyoto University Hospital, Shiga University Hospital, Nagasaki University Hospital, Tokushima University Hospital and Matsubara Mayflower Hospital) and ethical approval was received from each hospital. Patients with diabetes treated with drugs, with pancreatic diseases, with endocrine diseases such as Cushing’s syndrome and thyroid diseases, hepatic diseases, gastric diseases, heavy alcohol drinkers, pregnant and lactating women were excluded from this study. Between February 2008 and December 2010, 41 residents who met the study entry criteria of metabolic syndrome were enrolled in this study.

**Study design.** The study was divided by the control period in that subjects obtained nutritional counseling for 12 weeks (0–12 week) by registered dietitians and 24 week experimental period (12–36 week) followed by 200 kcal (837 kJ) of MHN-01 or dextrin-based standard balanced liquid formula (SBF) loading as a part of breakfast that was a prospective, randomized, open, blinded-endpoint design and multiple center trial (Fig. 1).

In the control period, each subject was consulted on energy intake with 30 kcal/kg of ideal body weight/day every month by registered dietitians. Energy intake and dietary habits in each subject were calculated from daily dietary records. A dietician calculated the quantity of intake energy from each patient’s dietary records and the mean values for the 3 days leading up to the scheduled clinic visits were determined. The total energy of the breakfast in the experimental period was fixed to that of the control period based on each dietary record. The subjects were asked to maintain a constant lifestyle and kept a dietary record to be completed during the 3 days prior to each scheduled visit to each of the institutions.

In the experimental period, eligible subjects were randomized to receive low glycemic index (GI) MHN-01 and SBF. The subjects visited at 0, 4, 8, 12, 24 and 36 week for collection of body composition measurement, and provided 3-day’s dietary records. Fasting blood samples were taken by venipuncture on 0, 12, 24, 36 week for analysis of parameters of carbohydrate and lipid metabolism. Abdominal fat value was assessed by CT scan on 12 and 36 weeks.

The study was performed after obtaining written informed consent from all patients, and was approved by the Ethics Committee of each institution. The protocol conformed to the Helsinki Declaration. The aim and study design were registered to Umin (Unique ID issued by UMIN: UMIN000001301) before the start of this study.

**Liquid formulas.** MHN-01 was prepared by partially replacing dextrin in SBF with 55.7% isomaltulose of carbohydrate and 72.3% of oleic acid of fat. The protein, fat, and carbohydrate % of energy were 20.0%, 29.7%, and 50.3%, respectively. SBF was a dextrin-formula that also contained sucrose; the protein, fat, and carbohydrate % of energy were 16.0%, 25.0%, and 59.0%, respectively (Table 1).

**Methods for analysis.** Plasma samples were kept at −20°C until analyzed. Plasma glucose concentration was measured by using a glucose oxidase-based autoanalyzer. Serum insulin concentration was measured by a standard radioimmunoassay. The total incremental area under the curve (AUC) for plasma glucose and insulin were calculated for a 180-min period after ingestion of each liquid formula. The insulin resistance index was calculated using the homeostasis model assessment for insulin resistance (HOMA-IR = fasting insulin (µU/ml) × fasting glucose (mmol/L) / 22.5). HbA_1c concentration was determined by high-performance liquid chromatography; serum triglyceride, total cholesterol, and high density lipoprotein (HDL) cholesterol concentrations were determined by enzymatic techniques using a Hitachi Model 736 auto-analyzer (Mito, Japan). Serum adiponectin concentrations were measured by human adiponectin ELISA kit (Otsuka Pharmaceutical Co., Ltd., Tokyo, Japan).

**Table 1. Composition of MHN-01 and standard balanced formula**

|                     | MHN-01 | Standard balanced formula (SBF) |
|---------------------|--------|---------------------------------|
| **Energy**          | 1 kcal/ml | 1 kcal/ml                      |
| **Protein**         | 20.0%  | 16.0%                           |
| **Fat**             | 29.7%  | 25.0%                           |
| **SFA**             | 10.9%  | 24.0%                           |
| **MUFA**            | 72.3%  | 52.0%                           |
| **PUFA**            | 15.1%  | 21.0%                           |
| **Carbohydrate**    | 50.3%  | 59.0%                           |
| Branched dextrin    | 23.9%  | Sucrose                         |
| Xyitol              | 5.3%   | Dextrin                         |
| Isomaltulose        | 55.7%  | Dietary fiber                   |
| Other carbohydrate  | 15.1%  |                                 |

Other carbohydrate: mixed carbohydrate from raw material and dietary fiber.
Statistical analyses. Data are presented as mean ± SD. Changes chemical and clinical parameters were analyzed using Wilcoxon matched-pairs signed-rank test. In addition, to adjust for age and BMI, multiple linear regression analysis was used. Significance was determined at <0.05. All statistical analyses were performed with Stat View for Windows, ver. 5.0 (SAS Institute; Cary, NC).

Results

Changes of clinical parameters by nutritional counseling. The clinical characteristics of entry subjects and change of the anthropometric and laboratory data from the beginning to the end of the control period (0–12 week) are shown in Table 2. Daily total energy intake, blood pressure and serum HbA1c level significantly decreased and fasting immunoreactive insulin (FIRI) and HOMA-IR levels significantly increased during the control period possibly due to the nutritional counseling and subsequent dietary changes. No significant changes were observed in body weight, BMI, waist circumference or fasting plasma glucose level and any other parameters in this period.

Comparison of clinical parameters changed in two groups with or without improvement during the control period. Sixteen of 41 subjects could not proceed to long term liquid formula administration study because of becoming to be out of criteria. In that unqualified group, several biomarkers were improved. In the unqualified (improved) group (n = 16) during 12 weeks, daily total energy intake, HbA1c and blood pressure significantly decreased (Table 3). In qualified (non-improved) group (n = 25), diastolic blood pressure and HbA1c levels decreased and both FIRI and HOMA-IR levels increased for 12 weeks. Daily total energy intake in unqualified (improved) group was significantly higher than those in qualified (non-improved) group at 0 week of this study. HbA1c levels in unqualified (improved) group were also lower than those in qualified (non-improved) group at 0 week and 12 week.

Effect of MHN-01 intake on clinical parameters. During the experimental period, 25 subjects residing in two long-term care facilities were enrolled in the study, and two subjects withdrew due to conflicts of schedule and health problems unrelated to the study, and one subject was excluded from analysis due to poor compliance. Therefore, all long-term study data were analyzed and are presented 10 subjects in MHN-01 group and 12 in SBF group completed the entire 24-week study. Serious side effects such as anemia, renal, or hepatic disorders did not appear during this study.

In the group ingested 200 kcal (837 kJ) MHN-01 as a part of breakfast during the experimental period, no significant changes were observed in body weight, BMI, abdominal visceral fat, fasting plasma glucose, fasting insulin, triacylglycerol, total

| Table 2. Changes of clinical parameters by nutritional counseling |
|----------------------|----------------------|
|                      | 0 week               | 12 week               |
| N (M/F)              | 41 (16/25)           |                       |
| Body weight (kg)     | 76.5 ± 16.2          | 76.1 ± 16.1           |
| BMI (kg/m²)          | 29.4 ± 4.0           | 29.2 ± 4.7            |
| Waist circumference (cm) | 95.4 ± 10.4        | 96.5 ± 13.4           |
| Systolic blood pressure (mmHg) | 130.4 ± 16.8    | 124.3 ± 14.8*        |
| Diastolic blood pressure (mmHg) | 83.6 ± 11.8      | 76.7 ± 12.0*         |
| Energy intake (kcal/day) | 1,876 ± 459         | 1,728 ± 319*         |
| FPG (mg/dl)          | 108 ± 9              | 108 ± 13              |
| FIRI (µU/ml)         | 6.7 ± 3.7            | 9.0 ± 4.8*            |
| HOMA-IR              | 1.78 ± 1.01          | 2.44 ± 1.46*         |
| Hemoglobin A1c (%)   | 5.6 ± 0.4            | 5.4 ± 0.4*            |

Data are presented as mean ± SD. * vs 0 week (p<0.05). BMI, body mass index; FPG, fasting plasma glucose; FIRI, fasting immunoreactive insulin; HOMA-IR, the homeostasis model assessment for insulin resistance.

| Table 3. Changes of clinical parameters in the improved and the non-improved groups by nutritional counseling |
|----------------------|----------------------|
|                      | The qualified (non-improve) group | The unqualified (improve) group |
|                      | 0 week               | 12 week               | 0 week               | 12 week               |
| N (M/F)              | 25 (8/17)           |                       | 16 (8/8)             |                       |
| Body weight (kg)     | 76.0 ± 16.5          | 75.7 ± 16.6           | 77.3 ± 14.8          | 76.6 ± 15.5           |
| BMI (kg/m²)          | 29.8 ± 5.1           | 29.6 ± 5.1            | 28.7 ± 3.6           | 28.4 ± 4.1            |
| Waist circumference (cm) | 95.4 ± 10.6        | 95.9 ± 10.9           | 95.4 ± 10.5          | 94.2 ± 11.1           |
| Systolic blood pressure (mmHg) | 130.6 ± 20.4       | 126.5 ± 15.8          | 130.1 ± 11.1         | 120.9 ± 12.8*        |
| Diastolic blood pressure (mmHg) | 82.6 ± 13.4       | 77.2 ± 12.9*          | 85.7 ± 9.3           | 75.9 ± 10.7*         |
| Energy intake (kcal/day) | 1,776 ± 453         | 1,687 ± 309           | 2,174 ± 354*        | 1,850 ± 340*         |
| FPG (mg/dl)          | 109 ± 8             | 109 ± 6               | 106 ± 10             | 107 ± 19              |
| FIRI (µU/ml)         | 5.9 ± 2.8           | 8.4 ± 3.5*            | 8.0 ± 4.5            | 10.1 ± 6.3            |
| HOMA-IR              | 1.60 ± 0.80         | 2.24 ± 0.89*          | 2.08 ± 1.24          | 2.76 ± 2.07           |
| Hemoglobin A1c (%)   | 5.7 ± 0.4           | 5.6 ± 0.3*            | 5.4 ± 0.3*           | 5.3 ± 0.3*            |

Data are presented as mean ± SD. * vs 0 week (p<0.05). † vs The qualified (non-improved) group at 0 week (p<0.05). ‡ vs The qualified (non-improved) group at 12 week (p<0.05). BMI, body mass index; FPG, fasting plasma glucose; FIRI, fasting immunoreactive insulin; HOMA-IR, the homeostasis model assessment for insulin resistance.
IRI levels at 30 min after MHN-01 loading were lower than after E. Takeda

Data are presented as mean ± SD. *vs SBF group at 12 week (p<0.05). BMI, body mass index; FPG, fasting plasma glucose; FIRI, fasting immune-reactive insulin; HOMA-IR, the homeostasis model assessment for insulin resistance.

Table 4. Effect of Inslow intake for 24 weeks on clinical parameters

|                  | MHN-01 group | Standard balanced formula (SBF) group |
|------------------|--------------|--------------------------------------|
|                  | 12 week      | 24 week                              |
|                  | 36 week      |                                      |
|                  | 12 week      | 24 week                              |
|                  | 36 week      |                                      |
| N (M/F)          | 10 (2/8)     | 12 (4/8)                             |
| Body weight (kg) | 74.9 ± 21.0  | 74.2 ± 20.7                          |
|                  | 74.4 ± 21.1  | 74.9 ± 14.9                          |
|                  | 74.5 ± 15.2  | 74.7 ± 15.7                          |
| BMI (kg/m²)      | 30.9 ± 7.4   | 30.7 ± 7.3                           |
|                  | 30.7 ± 7.6   | 28.8 ± 3.0                           |
|                  | 28.7 ± 3.1   | 28.7 ± 3.3                           |
| Waist circumference (cm) | 102.6 ± 21.2 | 102.8 ± 20.9                     |
|                  | 101.1 ± 21.4 | 93.9 ± 7.9                           |
|                  | 92.1 ± 5.3   | 92.6 ± 5.5                           |
| Systolic blood pressure (mmHg) | 131.3 ± 20.8 | 124.1 ± 19.4                     |
|                  | 125.0 ± 18.6 | 126.3 ± 13.9                        |
|                  | 125.6 ± 9.4  | 132.0 ± 10.2                         |
| Diastolic blood pressure (mmHg) | 78.4 ± 15.7  | 78.4 ± 10.7                        |
|                  | 77.4 ± 10    | 80.9 ± 11.4                         |
|                  | 78.9 ± 11.1  | 82.6 ± 10.3                         |
| Energy intake (kcal/day) | 1,799 ± 330  | 1,758 ± 401                         |
|                  | 1,582 ± 263  | 1,552 ± 270                         |
|                  | 1,660 ± 276  | 1,657 ± 427                         |
| PPG (mg/dl)      | 108 ± 6      | 109 ± 10                            |
|                  | 107 ± 6      | 110 ± 270                           |
|                  | 111 ± 10     | 112 ± 12                            |
| FIRI (µU/ml)     | 9.3 ± 4.1    | 8.0 ± 2.7                           |
|                  | 7.9 ± 2.4    | 8.2 ± 2.9                           |
|                  | 9.0 ± 3.7    | 8.1 ± 2.9                           |
| HOMA-IR          | 2.47 ± 1.03  | 2.14 ± 0.67                         |
|                  | 2.08 ± 0.65  | 2.23 ± 0.75                         |
|                  | 2.40 ± 0.83  | 2.26 ± 0.86                         |
| Hemoglobin A1c (%) | 5.6 ± 0.3    | 5.6 ± 0.4                           |
|                  | 5.6 ± 0.4    | 5.6 ± 0.4                           |
|                  | 5.6 ± 0.4    | 5.7 ± 0.4                           |
|                  | 5.7 ± 0.4*   |                                      |
| Abdominal visceral fat (cm²) | 144.1 ± 8.1  | —                                   |
|                  | 146.0 ± 77.2 | 137.7 ± 39.6                        |
|                  | 135.2 ± 38.1 |                                      |
| OGTT AUCpg (mg·h/dl) | 350.5 ± 40.0 | 361.9 ± 50.2                      |
|                  | 346.6 ± 51.7 | 365.6 ± 56.5                        |
| OGTT AUCiri (µU·h/ml) | 79.9 ± 37.6 | —                                  |
|                  | 66.0 ± 35.1  | 80.7 ± 45.3                         |
|                  | 64.2 ± 31.9  |                                      |

Discussion

The lifestyle modifications that combine energy restriction and healthy eating (increased intake of fruits, vegetables, fish and water and reduced consumption of sugar, fat, sodium, and fried foods) would appear to be a preferred treatment strategy for metabolic syndrome. In this study, 16 of 41 subjects became to be out of criteria by nutritional counseling for 12 weeks. Improved group was characterized by higher energy intake and lower serum HbA1c level at 0 week, suggesting poor lifestyle and short period of glucose metabolic impairment. Several studies have revealed the importance of nutritional education aiming at preventing metabolic syndrome. The present study confirmed this by observing a significant decline in energy intake and HbA1c in the improved group.

Glycated hemoglobin (HbA1c) levels are closely associated with postprandial glucose levels in type 2 diabetes, in comparison with fasting glucose levels. Since energy amounts during the experimental period were unchanged, it is conceivable that the maintaining serum HbA1c levels in MHN-01 group rather than increased concentration in SBF group is due to the effect of the long-term (24 weeks) ingestion of MHN-01 as a part of breakfast in these subjects with metabolic syndrome. The glycemic index of foods is now considered to be an important feature in the development of insulin resistance as determined by HOMA-IR. After adjustment for potential confounding variables, total but also fruit and cereal dietary fiber intakes were inversely associated with HOMA-IR in the Framingham Offspring Study.

A previous study demonstrated that peak plasma glucose and IRI levels at 30 min after MHN-01 loading were lower than after SBF loading in the human study. Postprandial fat oxidation rates in the MHN-01 group were higher than those in the SBF group. The free fatty acid (FFA) concentration in the MHN-01 group immediately after lunch was significantly lower than that in the SBF group. Plasma glucose and IRI levels in the MHN-01 group after the standard lunch were lower than those in the SBF group, though the peak levels in these groups were not different. Development of type 2 diabetes is associated with progressive histopathological changes in pancreatic islets, including selective loss of β-cells and fibrosis. The most obvious mechanism to explain pancreatic decompensation is a progressive loss of β-cell mass, which is hastened by islet fibrosis. Our data demonstrated that isomaltulose, relative to sucrose, had an antifibrotic effect on the islets of Zucker fatty rats. Sucrose-fed rats showed a much higher proportion of distinctly fibrotic islets than isomaltulose-fed rats. Furthermore, a recent systematic review of short-term randomized feeding trials in diabetic patients showed that low-GI diets were associated with improvements of glycemic control, insulin sensitivity, and other intermediate biomarkers.

There is also an indication that lean, physically active subjects can adjust to the postprandial glucose challenge following a high-GI meal by increasing insulin sensitivity, while obese, inactive subjects must increase their insulin secretion in order to reestablish glucose homeostasis.

In a recent study in healthy subjects, β-cell function and insulin sensitivity progressively improved in the postprandial state as the proportion of mono-unsaturated fatty acids (MUFAs) with respect to saturated fatty acids (SFAs) in fatty meals increased. Also, in a cross-sectional study of the general population in the Southeast of Spain, an oral glucose tolerance test was used in 538 subjects to calculate insulin resistance and β-cell function derived from HOMA indexes and results showed a favorable association between MUFAs intake and insulin secretion. Recent data from subjects of a Mediterranean country with high dietary MUFA intake in the form of olive oil show a significant inverse association between the serum phospholipid proportions of oleic acid, the main MUFA, and insulin resistance assessed by the HOMA method.

Findings from obese Zucker rats fed for 4 vs 8 weeks provide strong evidence that pancreatic islets may be target sites that translate the effects of different combinations of dietary carbohydrates and fats into metabolic changes.

Development of type 2 diabetes is associated with progressive histopathological changes in pancreatic islets, including selective loss of β-cells and fibrosis. The most obvious mechanism to explain pancreatic decompensation is a progressive loss of β-cell mass, which is hastened by islet fibrosis. Therefore, our previous findings indicate that sucrose and linoleic acid together may act to
induce subtle but striking changes in pancreatic islets long before manifestation of type 2 diabetes symptoms. A combination of isomaltulose and oleic acid, which would help preserve of β-cells and prevent deleterious changes in pancreatic islets, may reduce the risk of developing obesity, type 2 diabetes and metabolic syndrome.

Regarding the absence of disease or infirmity there is excellent evidence that low-GI diets reduce the risk for many diseases.(29) All this suggests that GI influences physiological functions in ways that are relevant to virtually everyone. This is particularly important for Asian because the glycemic response to the foods was higher in Asian Indian subjects compared with UK Caucasian subjects, although there were no significant differences in the GI values of the same foods between the two groups.(29) Because this study was carried out with a small number of subjects, further study with larger populations should be initiated to support our findings. In conclusion, we suggest that long-term ingestion of MHN-01 as a part of breakfast may be effective in keeping patients with metabolic syndrome under good glucose and lipid metabolism.

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Conflict of Interest

H.S., A.K., and H.A. are employees of Meiji Co., Ltd.