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Original article

Amelioration of postprandial hyperglycemia by exercise with ultra-soft rubber bands: A non-crossover test.

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Abstract

***Purpose*:** Postprandial hyperglycemia induces a chain reaction of aldehyde production, thus causing vascular endothelial cell damage which greatly increases the risk of cerebral cardiovascular disease. In this study, we verified the postprandial hyperglycemia improvement effect of a resistance exercise using a rubber band after eating.

***Methods*:** 66 men and women with no regular exercise habits were recruited, followed by selecting in the order of high fasting blood glucose, 12 subjects (7 males, 5 females, 55.3 ± 5.2 years old) were analyzed. After ingesting cooked rice (200 g), three types of resistance exercises using a super soft rubber band (large or small size) were performed, and blood glucose and insulin levels were measured. The test was non-cross-over, and every week, control (no exercise), GB10 (large, 10 min, 35 kcal), GS10 (small, 10 min, 35 kcal), GS6 (small, 16 min, 21 kcal) of each group was performed.

***Results*:** Compared with the control, GS10 showed significantly lower blood glucose levels at 30 min, while GS6 at 30 and 90 min ($p < 0.05$). There were no differences between groups in glucose AUC, iAUC, Cmax, Tmax, HOMA-IR. The insulin levels of GB10, GS10, and GS6 were significantly higher than those of the control at 60 min ($p < 0.05$). The low order of fasting blood glucose, Cmax and the high order of insulin AUC were GS6 group (4th), GS10 group (3rd), GB10 group (2nd), control (1st), and indicating accumulative effect of the exercise. There were no adverse events.

***Conclusion*:** Middle-aged people with insufficient physical activity performed resistance exercises with this band for 6 to 10 min after eating, leading to reduced postprandial hyperglycemia and an increase in glucose-responsive insulin secretion. In addition, an accumulation effect was observed, suggesting that glycative stress may be improved by continuing this exercise.

***KEY WORDS*:** postprandial hyperglycemia, insulin secretion, glucose tolerance, resistance training, glucose spikes, ultra-soft rubber band

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Introduction

The major causes of cerebral cardiovascular disease, which is the top cause of death in Japanese, are high glycative stress, *i.e.*, impaired glucose tolerance, dyslipidemia, and high blood pressure that causes mechanical damage to the vascular endothelium. These factors are accumulated in the metabolic syndrome triggered by visceral obesity and are also a major risk factor for type 2 diabetes mellitus (T2DM). Among the Japanese aged 40 to 70, 19.4 million are with metabolic syndrome or its high-risk group [1, 2]. Regarding the number of diabetic patients, in the Ministry of Health, Labor and Welfare "Overview of 2017 Patient Survey", it reported that it increased by 123,000 from the previous survey in 2014, reaching a record high of 3,289,000 [3]. The rapid increase in the number of metabolic syndrome and T2DM cases has become a major social problem.

As a measure against this problem, it is important to improve lifestyle habits from the earliest stage as much as possible, that is, to prevent progression from the stage of mild glucose intolerance to the next stage. Postprandial hyperglycemia is referred to as glucose spikes [4, 5] which induce production of various aldehydes by a chain reaction (aldehyde spark) [6], thus causing vascular endothelial cell damage, which is a risk factor of cerebral cardiovascular disease. In this study, we have chosen short-term muscle load exercise using a rubber band and verified the amelioration effect on postprandial hyperglycemia.

Methods

Subjects

The subjects were healthy men and women aged from 45 to 65 years without exercise habits and 66 participants were selected. Physical examination, tests of hematology, general blood chemistry, urinalysis, electrocardiography, and doctor interviews were performed as a prior examination (screening: SCR) on the recruiters. Finally, 14 patients were selected in descending order of fasting blood glucose (FBG) level from those who met the selection criteria and did not violate the exclusion criteria and were judged to be appropriate to participate in the study based on the investigator's judgment.

The selection criteria are shown below.

1) Men and women 40 to 60 years old at the time when the consent for participation in the study was obtained.
2) Healthy persons without chronic physical illness including skin diseases.
3) Persons with the ability to give consent after receiving an adequate explanation of the purpose and content of the study, and who volunteer to participate on their own accord after proper understanding and provide a written consent to participate in this study.
4) Persons who can come on the designated examination date to undergo examination.
5) Persons determined to be suitable as a subject of this study by the principal investigator.

The exclusion criteria are shown below.

1) Persons determined to be unsuitable as a subject of this study by the principal investigator.
2) Persons with a history of, or currently suffering from mental illness, sleep disorders, hypertension, diabetes mellitus, dyslipidemia, or a serious illness.
3) Persons who have been taking drugs for the treatment of a disease for the past one month (excluding those with a history of taking temporary-relief medication for headaches, menstrual pain, and colds)
4) Persons with a history of, or currently suffering from a severe disease of the liver, kidney, heart, lungs, digestive organs, or hematologic disease.
5) Persons with a history of, or currently suffering from a severe disease of the gastrointestinal tract.
6) Persons who have an abnormality on electrocardiography.
7) Persons with a body mass index (BMI) of less than 18.5 kg/m^2 or more than 30 kg/m^2.
8) Persons who have donated 200 ml of blood in the past one month, or over 400 ml within the past 3 months.
9) Persons with severe anemia.
10) Persons who have difficulty collecting blood as scheduled.
11) Persons with daily exercise habits.
12) Persons who have difficulty in exercising for 10 min.
13) Persons who consume more than 60 g/day of alcohol on average.
14) Persons who may change their lifestyle during the test, *i.e.*, long-term travel.
15) Currently and within the past 3 months, persons who have a habit of continuously taking functionally labeled foods/health foods that are associated with glucose metabolism, and those who plan to take them during the study period.
16) Women pregnant, lactating or possibly pregnant.
17) Persons who are currently participating in other human clinical trials, and those who have participated in other human clinical trials within the past 3 months.
18) Persons who have been determined by the principal investigator as not suitable to be a subject of this study.

Figure 1 shows the change in the number of test subjects. The 12 subjects (7 men, 5 women) analyzed were 55.3 ± 5.2 years (males: 55.0 ± 5.9, females; 55.6 ± 4.7 years).

Test design

As a non-intersecting type repeat test, this test performed postprandial loading of the following three types of exercise and compared the transion of glucose and insulin levels with control.

- GB10 group: Exercise full set using a large band (10 min).
- GS10 group: Exercise full set using a small band (10 min).
- GS6 group: Exercise half set using a small band (6 min).

The test schedule is shown in ***Fig. 2***. There was a one-week washout period between each visit.

The ultra-soft rubber band used is made by Kyowa Rubber Co., Ltd. (Hirakata, Osaka, Japan), and the silicon material is specially processed to be super-soft and intended to easily move the muscle without applying a large load. The large type had a thickness of 2 mm and the perimeter was about 1,600 mm, and the small type had a thickness of 2 mm and the perimeter was about 500 mm. The exercises were performed by passing a band through the arms and legs, with the trainer instructing the subject in the standing and sitting positions.

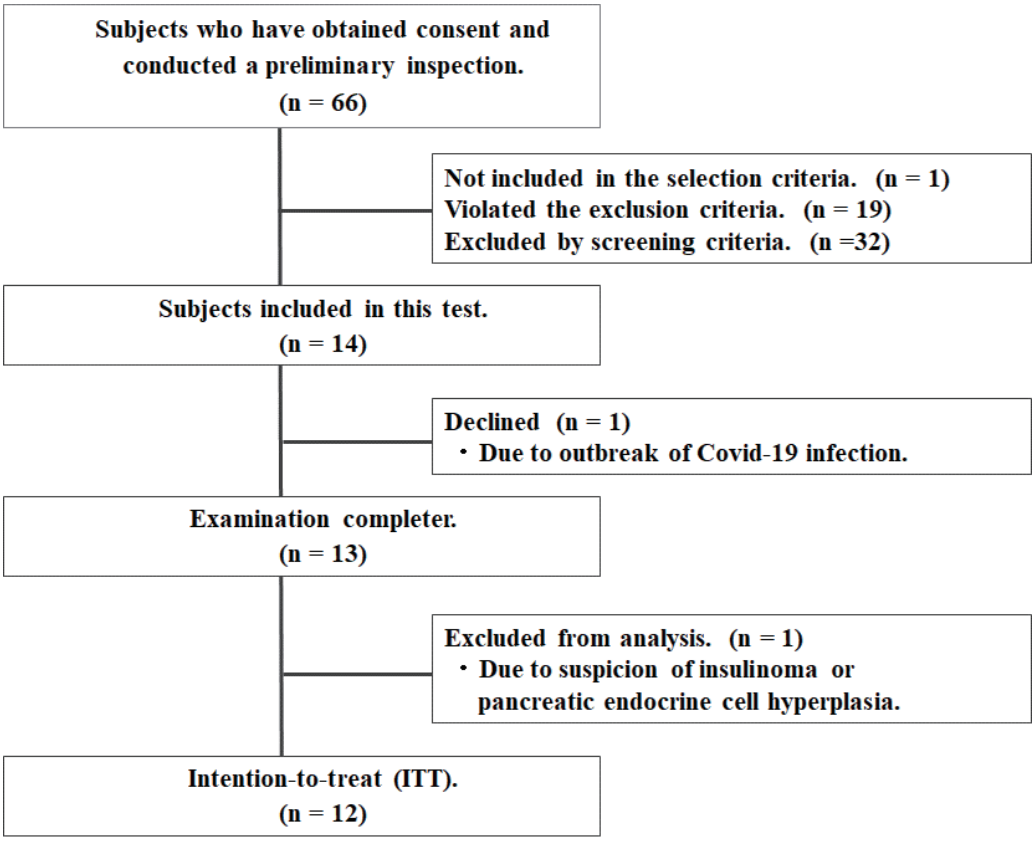
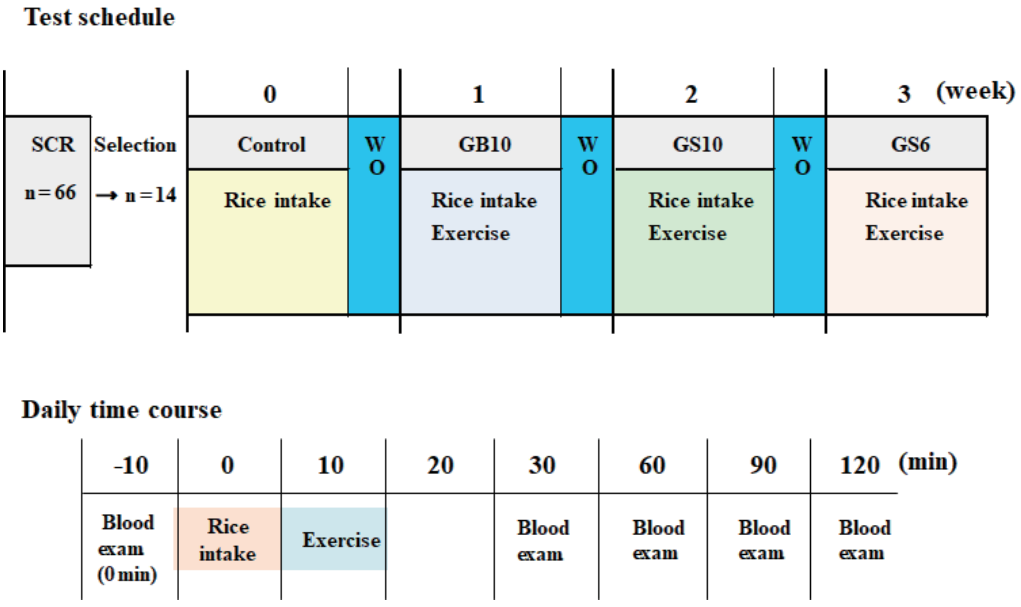


Fig. 1. **Changes in the number of test subjects.**



SCR, screening; WO, washu out; Exam, examination.

Fig. 2. **Test schedule.**

Evaluation iteme

Blood glucose and insulin were measured before, 30 min after loading, 60 min after loading, 90 min after loading, and 120 min after loading (5 times). The main evaluation items were changes in measured values, the amount of increase or decrease relative to before loading meal intake (0 min), the area under the curve (AUC) calculated using the trapezoidal method, incremental AUC (iAUC) under which the negative area is added as a negative value, Cmax which was the maximum blood concentration, and Tmax which was the maximum blood concentration reaching time after loading rice. HOMA-IR (homeostasis model assessment of insulin resistance) using the calculated value of fasting insulin level (μU/mL) x FBG level (mg/dL)/405 was used as the secondary endpoint. The test period was from February to March 2020.

Prior examination

Anthropometric measurements (height/weight/body fat percentage/BMI), blood pressure/pulse measurement, electrocardiography, doctor interviews, general urine tests, peripheral blood tests and biochemical tests using blood samples were performed. The items to be implemented included white blood cell count (WBC), red blood cell count (RBC), hemoglobin amount (Hb), hematocrit value (Ht), average red blood cell volume (MCV), average red blood cell hemoglobin amount (MCH), average red blood cell pigment concentration (MCHC), platelet count (Plt), total protein (TP), albumin quantification, urea nitrogen (BUN), creatinine (CRE), uric acid (UA), aspartate aminotransferase (AST), alanine aminotransferase (ALT), γ-glutamyl transpeptidase (γ-GTP), alkaline phosphatase (ALP), lactate dehydrogenase (LDH), creatinine kinase (CPK), total cholesterol (TC), triglyceride (TG), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), non-HDL cholesterol * calculated value, total bilirubin (T-Bil), FBG, HbA1c [NGSP], and immune-reactive insulin (IRI). The test using the blood sample was measured by Health Science Institute Co., Ltd. (Yokohama, Kanagawa, Japan).

Rice load: How to adjust and ingest loaded meals

The test was conducted according to a uniform protocol by the Japan Glycemic Index (GI) Study Group [8]. Commercially available packaged steamed rice "Sato no Gohan, Niigata Koshihikari 200 g" (Sato Food Industry, Niigata, Japan) was used. The packed rice was cooked in a microwave oven 15 min before the load inspection and stored in a warm storage. As a seasoning, 3 g of seasoned salt (Gomashio [salted sesami]; Marumiya Food Industry, Tokyo, Japan) was added to the rice and 150 mL of water was provided to the subjects. The composition of the loaded diet is shown in ***Table 1, 2***. The subjects were instructed to chew the rice about 30 times per bite. The intake time was 10 min.

Table 1. Loaded rice.

Ingredient	Steamed rice (200 g)
Calorie	294 kcal
Protein	4.2 g
Fat	0 g
Carbohydrate	67.8 g
Sodium	0 g

Table 2. Seasoning ingredient.

Ingredients	Salted sesame (3 g)
Calorie	15 kcal
Protein	0.42 g
Fat	1.2 g
Carbohydrate	0.5 g
Salt equivalent	0.71 g

Exercise procedure

GB10 group

1) Abdominal exercises: Rolled up in 3 seconds and returned in 3 seconds (2 min).
2) Waist twist: twisted in 3 seconds and returned in 3 seconds. Left and right (2 min).
3) Rowing: Pulled for 3 seconds and returned in 3 seconds (2 min).
4) Punching: Pressed in 3 seconds and returned in 3 seconds (2 min).
5) Leg press: Pressed in 3 seconds and returned in 3 seconds left and right (2 min).

GS10 group

1) Shoulder reflection: Raised in 3 seconds and lowered in 3 seconds (2 min).
2) Side vent: The body was bent sideways in 3 seconds and returned in 3 seconds (2 min).
3) Abduction: opened in 3 seconds and returned in 3 seconds. Left and right (2 min).
4) Knee extension: raised, held for 3 seconds and lowered. Left and right (2 min).
5) Marching: Raised, held for 3 seconds and lowered. Left and right (2 min).

GS6 group

1) Side vent: The body was bent sideways in 3 seconds and returned in 3 seconds (2 min).
2) Hip Abduction: Opened in 3 seconds and returned in 3 seconds. Left and right (2 min).
3) Marching: Raised and held for 3 seconds. Left and right (2 min).

Statistical Analysis

The statistical analysis was performed with a paired t-test using statistical analysis software SAS (SAS 9.4; SAS Institute Japan, Minato-ku, Tokyo) and SPSS (Statistics 25; IBM Japan, Chuo-ku, Tokyo). Calculated data are presented as mean ± standard error mean in the figures and mean ± standard deviation in the text and Tables. The measured values were analyzed by two-tailed paired t-test/Dunnett multiple comparison tests [9, 10]. Regarding the safety evaluation, time-course comparison and group comparison were performed by the paired t-test. A risk rate of less than 5% was considered a significant difference.

Ethical standards

This study was conducted in compliance with the Declaration of Helsinki (revised at the 2013 WMA Fortaleza General Assembly) and the ethical guidelines for human-based medical research (notification by Ministry of Education, Culture, Sports, Science and Technology [MEXT] and Ministry of Health, Labour and Welfare [MHLW]). This research obtained the approval of the Ethical Committee of the Society for Glycative Stress Research (GSE 2018-001), which has discussed the ethics and validity of the study. The clinical trial for this study was pre-registered (UMIN #000039165).

Results

Changes in blood glucose

The blood glucose level of GS10 was significantly lower than that of the control 30 min after eating ($p < 0.05$, ***Fig. 3***). GS6 showed significantly lower values than those of the control at 30 min ($p < 0.01$) and at 90 min ($p < 0.05$). There were no significant differences in glucose AUC, iAUC, Cmax, and Tmax (***Table 3***).

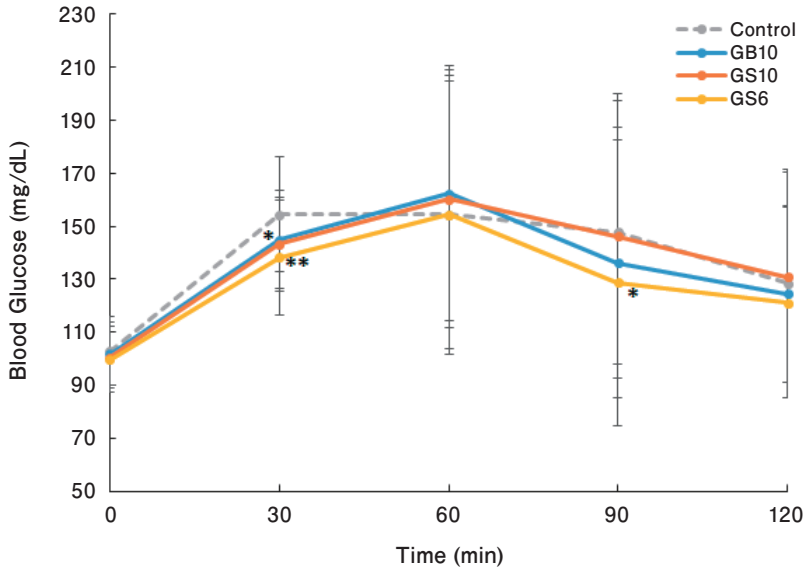


Fig. 3. Results are expressed as mean ± SD, n = 12, * $p < 0.05$, ** $p < 0.01$ vs control by two-tailed paired t-test/Dunnett multiple comparison tests.SD, standard deviation.

Table 3. AUC, iAUC, Cmax and Tmax of glucose.

	AUC (mg • min/dL)	iAUC (mg • min/dL)	Cmax (mg/dL)	Tmax (min)
Control	1,7161 ± 4,098	4,801 ± 3,267	170.4 ± 41.0	50.0 ± 26.6
GB10	1,6705 ± 3,834	4,495 ± 2,851	170.4 ± 40.0	52.5 ± 18.6
GS10	1,6958 ± 4,003	4,888 ± 2,942	173.4 ± 42.5	55.0 ± 21.5
GS6	1,595 ± 4,082†	3,983 ± 3,260	163.3 ± 42.7	52.5 ± 18.6

Results are expressed as mean ± SD, n = 12, † $p = 0.0598$ vs control by two-tailed paired t-test/Dunnett multiple comparison tests. AUC, area under the curve; iAUC, incremental area under the curve; Cmax, maximam value of glucose concentration; Tmax, time to maximum value of glucose concentration; SD, standard deviation.

Changes in insulin

Insulin levels of the GB10 ($p < 0.05$), GS10 ($p < 0.01$), and GS6 ($p < 0.005$) were significantly higher than those of the control group 60 min after eating (***Fig. 4***). Insulin AUC, iAUC, and Cmax were significantly higher in GS10 and GS6 than in the control group ($p < 0.05$, ***Table 4***). There were no significant differences in HOMA-IR between groups.

Safety

Fisher exact test was used for statistical analysis of the number of cases of side effects and adverse events. No side effects attributable to the test product were confirmed in all subjects.

Discussion

Glucose tolerance

Among Japanese with impaired glucose tolerance, those with low insulin secretion are common, while those with obesity were much fewer [11, 12]. Since insulin secretion decreases with age [13]. T2DM is more likely to develop simply by mild increase in insulin resistance due to overeating and lack of exercise [14, 15].

The reason for this is that the Japanese have a significantly lower insulin secretory capacity in the early stage after glucose loading, as compared with other ethnic groups. Furthermore, when the ATP-binding cassette transporter 1 (ABC1) of pancreatic β-cells is impaired, it becomes difficult to prevent the accumulation of excess cholesterol in the β-cell membrane, resulting in impaired insulin secretion [16]. It has been reported that some gene polymorphisms tend to cause insulin secretion disorders constitutionally.

Based on the glucose tolerance data in the Ningen Dock/Medical Checkups (data from the Nippon Kokan Hospital) and previous reports [11-16], the "process from normal to glucose intolerance resulting in T2DM" is outlined. Most subjects shift from normal weight to mild obesity/visceral obesity, during which the FBG level gradually approaches the upper limit of the standard range (Ningen Dock Society standard value of 99 mg/dL). Concurrently, fasting insulin gradually increased in those who initially had less than 5 μU/mL,

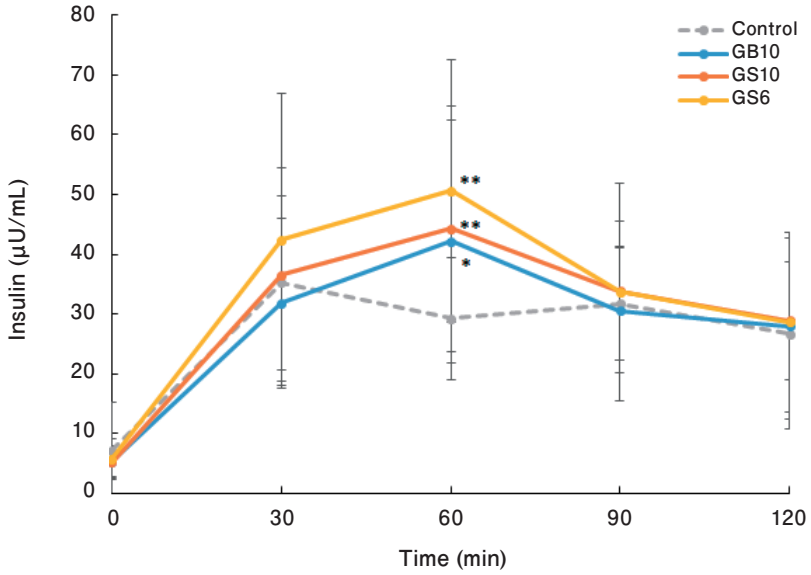


Fig. 4. Results are expressed as mean ± SD, n = 12, * $p < 0.05$, ** $p < 0.01$ vs control by two-tailed paired t-test/Dunnett multiple comparison tests.SD, standard deviation.

Table 4. AUC, iAUC, Cmax and Tmax of insulin and HOMA-IR.

	AUC (μU • min/mL)	iAUC (μU • min/mL)	Cmax (μU/mL)	Tmax (min)	HOMA-IR
Control	3,395 ± 1,052	2,530 ± 1,061	40.5 ± 13.1	70.0 ± 34.6	1.84 ± 2.01
GB10	3,638 ± 1,169	2,993 ± 1,015*	47.1 ± 17.0†	70.0 ± 14.8	1.37 ± 0.75
GS10	3,954 ± 1,217*	3,328 ± 1,023**	50.3 ± 18.0**	65.0 ± 25.0	1.31 ± 0.82
GS6	4,318 ± 1,856*	3,626 ± 1,592*	55.2 ± 19.8*	60.0 ± 25.6	1.46 ± 0.98

Results are expressed as mean ± SD, n = 12, † $p = 0.069$, * $p < 0.05$, ** $p < 0.01$ vs control by two-tailed paired t-test/Dunnett multiple comparison tests. AUC, area under the curve; iAUC, incremental area under the curve; Cmax, maximam value of insulin concentration; Tmax, time to maximum value of insulin concentration; HOMA-IR, homeostasis model assessment of insulin resistance; SD, standard deviation.

and HOMA-IR increased. This condition is dominant in insulin resistance. Therefore, postprandial hyperglycemia also gradually rises, and blood glucose spikes are frequently induced. Unless lifestyle factors are improved, this condition continues for several years, and the amount of insulin secretion gradually decreases. This is an intermediate state with impaired insulin secretion. As the bad lifestyle habits continue, insulin secretion decreases, FBG rises significantly above the standard range, and postprandial hyperglycemia exceeds 200 mg/dL. This is a state in which impaired insulin secretion is predominant and is a pre-stage for the onset of diabetes.

In this study, 12 middle-aged men and women without exercise habits (55.3 ± 5.2 years old) were analyzed, and three types of mild muscle exercise were performed as a control when postprandial blood glucose was observed without exercise. A non-crossover type repeated test was conducted for postprandial blood glucose transition, and a comparative analysis was performed. The glucose tolerance of the subjects showed slightly higher scores in FBG (103.0 ± 13.0 mg/dL) and fasting insulin (7.21 ± 8.12 μU/mL), and slightly higher in HOMA-IR (1.84 ± 2.01). Although fasting insulin levels do not show a decrease in insulin secretion, postprandial insulin secretion is low due to a lower insulin AUC. This finding suggests that postprandial insulin secretion may be potentially affected early on before fasting insulin levels decline.

Improved glucose tolerance

In this study, a non-crossing type repeated test was conducted on the postprandial blood glucose transition when three types of mild muscle exercise were performed just after the meal, and the data were compared with a control without exercise. The same participants took the test weekly in the following order.

Order	Group	Time	Calorie consumption*
• 1st	Control	0 min	0 kcal
• 2nd	GB10	10 min	35 kcal
• 3rd	GS10	10 min	35 kcal
• 4th	GS6	6 min	21 kcal

*Estimated calorie consumptions are calculated for a standard weight of 60 kg.

As a result, both exercises showed a decrease in blood glucose AUC and an increase in insulin AUC, alleviation of postprandial blood glucose elevation and improvement of insulin secretory ability. However, the results of comparing the three types of muscle exercises were different than expected. The effect of improving glucose tolerance was highest in GS6 (4th), which consumes the least calories.

The order of FBG, glucose Cmax and insulin AUC were GS6 (4th), GS10 (3rd), GB10 (2nd), and control (1st). In these items, glucose tolerance was most improved in the GS6 (4th group) which had an accumulation of exercise. The exercise time and the thickness of the rubber band did not affect the effect of improvement of glucose tolerance. It is considered that even if the exercise amount is small, a cumulative effect will appear by continuing performance once a week.

What is interesting is that the secretory effect of this postprandial insulin is different from dietary intervention. In the case of dietary intervention, the effect of suppressing blood sugar elevation by delaying glucose absorption is the primary effect [17-19]. It may, in part, be related to the effect of stimulating GIP (glucose-dependent insulinotropic polypeptide) secretion from the small intestine, but in the case of dietary intervention, insulin secretion remains lower than the control group. Conversely in the case of this exercise intervention, postprandial blood hyperglycemia is alleviated as a result of the increase in insulin secreted in response to the glucose elevation stimulation.

The present subjects are those with impaired insulin secretion compared with healthy young people, *i.e.*, middle-aged, having little or no exercise habit, overweight, or close to the metabolic syndrome boundary area. Since subjects had these characteristics, physical exercise might have improved their insulin secretion and alleviated postprandial hyperglycemia. The effect was further enhanced by the accumulation of exercise therapy once a week.

Benefits of exercise

It has been mainly considered that physical activity increases glucose consumption and glucose uptake to skeletal muscle may ameliorate hyperglycemia.

Regarding the relationship between exercise and insulin secretion, it has been reported that female students without exercise habits tend to increase insulin secretion due to exercise. Ingesting protein and lipids promotes GIP secretion [20], however, it seems that young people do not always increase insulin secretion [21-23].

There are few surveys targeting middle-aged and elderly people. Dietary treatment (1,500 ± 100 kcal, P : F : C = 7 : 27 : 56) and exercise therapy (aerobic exercise + muscle training with moderate intensity) have been conducted with 9 middle-aged patients with T2DM for 2-4 weeks, resulting that oGTT (oral glucose tolerance test) decreased AUC, while there was no significant change in HOMA-IR [24]. Changes in insulin secretion after glucose load have not been investigated in this study. The results that post-prandial insulin secretion was increased by muscular exercise in middle-aged people with latent insulin secretion as in this study are valuable.

Acute and chronic effects of exercise

An increase in glucose consumption due to exercise leads to suppression of postprandial hyperglycemia. This is an acute effect obtained by one-time exercise. Although it depends on the amount of exercise, a subject whose insulin secretion is normal often does not change or shows a decrease in insulin secretion due to exercise.

Regarding chronic effects, regular exercise causes weight loss and visceral fat reduction in obese persons and improves blood adipocytokine balance [25]. Continuation of exercise habits also induces a decrease in leptin concentration (especially in those with increased leptin resistance), decrease in resistin (especially in T2DM patients and those with high inflammatory cytokines), and an increase in adiponectin (especially in those with decreased visceral fat mass by exercise). These changes improve insulin resistance. However, adipose tissue loss cannot be expected with exercise similar to this study, and it is unlikely that this mechanism of action

is involved.
Exercise-induced increase in GLUT4 (glucose transporter type 4) improves glucose metabolism in skeletal muscle. As the mechanism, it is reported that the elevation of GLUT4 expression [26-29], the promotion of GLUT4 translocation [30, 31] and the increase of mitochondrial number are important. The improvement of insulin resistance can be explained by GLUT4 increase, however, it cannot explain the increase of insulin secretion by exercise.

As another factor, favorable effects of exercise via lipid metabolism are also expected. Its insufficiency causes accumulation of triglycerides and FACoA (fatty-acyl-coenzyme A) in skeletal muscle, inhibition of IRS-1 (insulin receptor substrate 1) tyrosine phosphorylation, and decreased PI3K (phosphoinositide 3-kinase) activity, resulting in impaired GLUT4 translocation [32]. In contrast, if adiponectin secretion from visceral fat is increased by exercise and TNF-α (tumor necrosis factor-α)/resistin is decreased, AMP kinase can be activated to increase fat burning (β-oxidation of fatty acid), that may improve insulin resistance and glucose tolerance [33].

When glucose tolerance decreases and glucose metabolism function declines, postprandial hyperglycemia is more likely to occur, leading to the condition with high glycative stress and high amounts of advanced glycation end products (AGEs). As a result, insulin secretion is reduced [34-36]. Experimentally, when AGEs are topically applied to pancreatic β cells, insulin mRNA decreases and the protein levels of intracellular proinsulin and insulin decrease [37]. Furthermore, in the process; preproinsulin → preinsulin → insulin inside pancreatic β-cells, carbonylation of lysine (Lys) and arginine (Arg) residues and succinylation of cysteine residue (-S) occur, which disturbs insulin production. [38]. Approximately 9 % of blood insulin in T2DM patients undergoes AGE modification (glycated insulin production) and loses insulin function, resulting in increased insulin resistance [39]. It is expected that exercise improves insulin secretion by reducing glycative stress.

Safety: adverse effects by exercise

When exercise intensity and load are excessive, glucagon and catecholamine secretion are increased, which acts on insulin antagonism. In more intense exercise, blood ketone body production, ROS (reactive oxygen species) production, and thiobarbituric acid reactive substances (TBARS) increase remarkably, and degenerative changes with organ damage and aging deteriorate [40-43]. In addition, the secretion of insulin antagonist hormones (counterregulatory hormone), *i.e.*, catecholamine, glucagon, growth hormone, and cortisol occurs during exercise. This reaction induces adaptation by continuing exercise, and the increase rate of insulin antagonistic hormone becomes moderated [44]. Therefore, for diabetic exercise therapy, moderate or lower intensity and a duration of 10 to 30 min are desirable [43]. There were no adverse events during this study. It is considered that the exercise therapy performed is effective for avoiding adverse events.

Research limitation

In this study, the changes in postprandial blood glucose immediately after the three kinds of exercise loads are compared with a control in a non-crossing type test. Compared with the crossover test, it cannot eliminate the factors of the cumulative effect and the elements of order from the analysis. Therefore, it was not able to compare the effects on glucose tolerance and insulin secretion among the three groups. On the other hand, the advantage is that the cumulative effect of the exercise could be observed.

Conclusions

This study showed that middle-aged people who tend to lack exercise but do muscle exercise using a super-soft rubber band for about 6 to 10 min after eating are able to reduce postprandial hyperglycemia, and its mechanism is based on increased insulin secretion. The results suggest that once a week, the persons with a lack of physical activity may continue their physical activity to restore the decreased insulin secretion. Insufficient exercise promotes skeletal muscle mass reduction with aging, and causes glycative stress through decreased glucose consumption and increased insulin resistance, therefore causing a vicious cycle in which insulin secretion from pancreatic β cells is further reduced [7]. It is quite possible that this cycle can be inhibited by continuing “muscle load training” as presented in this study.

Conflict of Interest Statement

The present study was partly supported by Kyowa Rubber Co.Ltd.

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