Nutritional Sciences 6(3), August, 2003, pp. 127-134

Effects of a Raw Diet on Plasma Glucose and Lipid Levels in Streptozotocin-induced Diabetic Rats

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This study investigates the effects of a raw diet (RD) on blood glucose and lipid metabolism in non-diabetic (normal) and streptozotocin (STZ)-induced diabetic rats. Male Sprague-Dawley rats were assigned to four groups (normal control, normal RD, diabetic control, and diabetic RD), for the four-week experimental period. The control groups were fed the AIN diet, and the RD groups were fed a diet consisting only of raw materials. Weight gain was statistically lower in the RD group than the control. Fasting plasma glucose was significantly lower in the diabetic RD group compared to the diabetic control group. The levels of triglycerides (TG), and of total cholesterol (TC) and LDL-cholesterol in the plasma, were lower in the RD groups than the control groups, but not significantly. There was a statistically significant decrease in the levels of TG and TC in the livers of the diabetic RD group, compared to the diabetic control group. The fecal levels of total lipids, TG, and TC were significantly higher in the RD groups, compared to the non-RD groups. It can be postulated that this raw diet may possess substantial hypoglycemic/hypolipidemic properties in diabetic rats.

Key Words : raw diet, plasma glucose, lipids, diabetes, rats

INTRODUCTION

Consumption of a raw diet can be defined as eating raw food with it being uncooked, which is opposite to eating a diet of cooked food.[1)] An alternative definition is: it is a method for eating vegetable food excluding animal food, as it is, without heating it, and further, it is a live meal without any process thereof and addition thereto that can contain various nutrients and vital substances, such as vitamins, minerals, enzymes, chlorophylls and so on, just as they are, and further natural vitality by preventing proteins, fat and the like from being transformed.[2)]

People took raw diet from the beginning of human history and the history of a diet of cooked food is not so long.[3)] Even though primitive men, who took raw diet, occasionally died due to a raid by wild animals or a natural disaster, failing to die of degenerative diseases. In a fundamental sense, taking a raw diet means eating raw vegetables or raw cereals just on the spot where they are harvested. However, today's raw diets for those living busy urban lifestyles can include freeze dried foods.[1,2)]

In studies of the nutritive status of people taking raw diets or vegetable diets, and of people taking raw staple foods, it has been shown that a raw diet can be superior to a diet of cooked food in many aspects.[1)] It is thought by many that raw diets are worth being recommended as a good food regimen of today which can be practically and properly applied to improve the diets of ordinary people; this opinion is based on studies such as the following: a study on changes in circulatory function, blood elements and weight between before and after taking exercise in people taking a raw diet[4)]; a study of differences in dietary behavior, nutritive conditions and condition of health, depending upon whether a raw diet is taken[5)]; and a study on the potential of a raw diet for prevention of cancers[2)].

As life patterns and diets change along with recent economic advancements, the mortality resulting from adult diseases, such as hypertension, arteriosclerosis, diabetes, etc. is on the rise. Also in Korea, as life patterns are gradually being westernized, the morbidity from adult diseases, particularly the morbidity from diabetes, sharply increase. According to various epidem- iologic data reported in the 1980's and 1990's, the morbidity from diabetes, which was estimated to be about 1 % in the 1960's, was shown to be about 3% and 5~8 % of the total population in the 1980's and 1990's, respectively. The annual average incidence rate of diabetes, which is currently 2.5%, is also assumed to have continuously increased along with improvements in the diets of people.[6)] Furthermore, the mortality resulted from

Accepted : July 8, 2003
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diabetes is continuously increasing; for example, such mortality was 7.8 per 100,000 population in 1987 and this increased to 17.4 in 1996, an increase of 2.2 times. It is thought that the mortality from diabetes will continue to increase in the future.[7]

Diabetes is a chronic metabolic disease bringing about hyperglycemia because glycolysis does not occur due to the hyposecretion of insulin from the β-cells of Langerhan's island in the pancreas, or through any anomaly of insulin receptors in tissues.[6,8] When symptoms persist, many complications, such as arteriosclerosis, kidney and nerve damage, and retinal change, are apt to take place.[9] Due to abnormal lipid metabolism related to the diabetic condition, an increase in the concentration of blood glucose and hyperlipidemia,[10] an increase in LDL-cholesterol and lipid peroxidation,[11] and variations in lipoproteins, can occur.[12] Particularly in the case of Type 2 diabetes which is a non-insulin dependent type and accounts for most cases of diabetes, diabetes causes complications of hyperlipidemia because increased amounts of LDL-cholesterol and triglycerides are produced due to a hyperglycemia-induced reduction in the decomposition of lipoproteins in the liver.[13,14] In this study, in order to investigate the possible clinical effects of a raw diet in rats, diabetes was artificially induced, and the effects of a raw diet on plasma glucose and lipid levels were studied.

METHODS

The experimental animals

Sixty male Sprague-Dawley rats weighing 15010g were used. A chow diet was supplied to all animals for a 2-week adaptation period. Then, the rats were divided into 4 groups (15 rats per group): a normal control group, a normal raw diet (RD) group, a diabetic control group, and a diabetic RD group. The chow diet fed in this experiment was the AIN-93G diet15) and its ingredients are as shown in Table 1. Both the chow and the raw feed were fed to the rats in powdered form. For the experimental period, the environmental conditions for the animals were set as follows: a temperature of 25±1℃; approximately 50% humidity; a 12-hour light and 12-hour darkness cycle (light 8:00 ~ 20:00); and unlimited supplies of feed and drinking water.

The experimental diet

Following previous work,[16] grains, fruits, mushrooms, seaweeds, green leaves, herbs, and sugars were selected as ingredients for the raw diet. Brown rice, barley, green peas, beans, glutinous brown rice, wheat, corn, glutinous millet, sorghum, dried barley sprouts, red beans, sesame, adlay (Job's tears), black beans, black sesame, apples, sea tangle, sweet potatoes, sweet pumpkins, shiitake mushrooms, kale, yams, pine needles, jujube, radish leaves, sea mustard, chestnuts, spinach, green tea, *Angelica keiskei*, bonnet bellflowers, rose of Sharon, burdock, carrots, mulberry leaves, laver, arrowroot, Houttuynlae Herba, *Saururus chinensis*, suki mushrooms, licorice roots, bananas, *maesil, Pleuropterus multflorus*, Hardy ruber, agaricus, raspberries, and *gasiogapi* were either freeze-dried or dried for 12 hours at 50℃. Oligosa- ccharides, salt, xylitol, spirulina, aloegel, *dongchungh- acho*, royal jelly, lactic acid bacteria, and propolis were mixed and ground together with the other ingredients.

Table 1. Composition of diets (g/kg)

Ingredients	
Corn Starch	529.486
Casein	200.000
Sucrose	100.000
Soybean oil	70.000
Cellulose	50.000
Mineral mixture*	35.000
Vitamin mixture**	10.000
L-Cystine	3.000
Choline bitartrate	2.500
T-butylhydroquinone	0.014

* Mineral mixture(per 1kg): Calcium carbonate, 357g; monopotassium phosphate, 196g; Potassium citrate, 70.78g; Sodium chloride, 74g; Magnesium oxide, 24g; Ferric citrate, 6.06g; Zinc carbonate, 1.65g; Manganous carbonate, 0.63g; Cupric carbonate, 0.30g; Potassium iodate, 0.01g; Ammonium paramolybdate, 0.00785g

**Vitamin mixture(per 1kg): Nicotinic acid, 3.0g; Ca Pantothenate, 1.6g; Pyridoxine HCl 0.7g; Thiamin HCl, 0.6g; Riboflavin 0.6g; Folic acid, 0.2g; D-Biotin, 0.02g; Vitamin B_{12}, 2.5g; Vitamin E, 15.0g; Vitamin A, 0.8g; Vitamin D_3, 0.25g; Vitamin K, 0.075g; Powdered sucrose, 974.655g

The inducement and identification of diabetes

Diabetes was induced in the two diabetic groups by letting the rats fast for 24 hours, and then, a 0.5ml solution of streptozotocin (STZ) was abdominally injected at a dose of 50 mg per 1 kg of body weight. The STZ solution was prepared by dissolving STZ in a citrate buffer (pH 4.0), and then kept in cold storage; the solution was used within 10 minutes after removing it from cold storage, because it rapidly becomes inert at room temperature. The inducement of diabetes was confirmed as follows: blood was collected from a vein under the condition of an empty stomach 24 hours after the STZ had been injected; the blood glucose level was then measured with a blood glucose measuring instrument. Rats having a blood glucose level of 300mg/dl or more were considered to be diabetic. In order to maintain similar treatment conditions, a sham injection using saline was made to the non-STZ-administered rats.

Preparation of samples

After the 4-week experimental period, no feed except water was supplied to each rat for 12 hours. Then, rats were lightly anesthetized with ethyl ether and, upon dissection, blood was collected from the heart. The collected blood was left at room temperature for about 30 minutes, and then plasma was separated from it by centrifuging it for 15 minutes at 3,000 rpm. The plasma was then used for measuring the lipid content, the value of hematocrit, and the levels of liver enzyme activity. The liver, the kidney and the pancreas were extracted from the experimental animals, and were washed with saline; water was then removed from these organs with gauze before they were placed in a deep freezer at -70℃ until further analysis.

Measurement of body weight, dietary intake, and food efficiency ratio

Each rat was weighed once per week during the experiment. The intake of food per day was measured at a given time every day. The food efficiency ratio (FER) was calculated by dividing the change in body weight by the intake of food.

$$\text{Food Efficiency Ratio (FER)} = \frac{\text{Weight Gained/Lost (g)}}{\text{The Amount of Food Consumed (g)}}$$

Blood gluc-ose

The blood glucose, the most frequently used indicator of diabetes, was measured by the glucose oxidase method

$$\text{Glucose (mg/dl)} = \frac{\text{Absorbance of Sample}}{\text{Absorbance of Standard Solution}} \times 200$$

Measurement of organ weight

After each rat was sacrificed, the liver, the kidney and the pancreas were extracted and their weights were measured.

Measurement of hematocrit, and of GOT (Glutamate Oxaloacetate Transaminase) and GPT (Glutamate Pyruvate Transaminase) in the plasma

To determine hematocrit, the blood was put into a capillary vessel by the microhematocrit method and was then precipitated with a microcapillary centrifuge; the packed cell volume was then measured by a microcapillary reader and the result was expressed as a percentage. The levels of GOT and GPT activity were measured by using a kit (Youngdong Pharmaceutical Co.) according to the Reitman and Frankel method.[17] Specifically, 1 ml of either the GOT or GPT substrates was placed in a test tube and was heated for 2~3 minutes in a water bath at 37℃. Then, 0.2 ml of plasma (diluted 5 times by volume with distilled water) was added and kept in a water bath at 37℃ - for 60 minutes in the case of measuring GOT reactions or 30 minutes in the case of measuring GPT reactions. Subsequently, 1 ml of the color coupler, 2,4-dinitrophenylhydrazine, was added and kept at room temperature for 20 minutes, after which 10.0 ml of 0.4N-NaOH was added to the mixture to stop the reaction. Thirty minutes after the reaction was stopped, the absorbance was measured at 505 nm, and this was converted into equivalent enzyme activity units by using the calibration curve.

Lipid components of plasma

The lipid levels of plasma (total lipids, triglycerides, total cholesterol, HDL-cholesterol, and LDL-cholesterol) were measured using kits (Youngdong Pharmaceutical Co.) from the Giegel et al.'s method.[18]

Measurement of lipid components of the liver

After blood was collected, the liver was extracted and weighed. Then, the remaining blood was washed out with a 0.9% NaCl solution, and water was removed with a filter paper. Potassium phosphate buffer (pH 7.4), which was equivalent to two times of the measured weight, was added to the liver and was homogenized with a Teflon tissue homogenizer (Glass-Col). The levels of total lipids, triglycerides, and total cholesterol were measured by using the homogenized sample in the same manner as in the case of plasma.

Measurement of lipid components of feces

The feces of the rats were collected for 3 days before they were sacrificed, and the weight of the collected feces was measured. One gram of the feces was lyophilized and then its weight was measured again. 0.2 g of the lyophilized feces was ground and the lipids were extracted using ethyl ether three times over a two-day period. Then, the extracted material was used for lipid analysis after being filtrated and vacuum con- centrated. The lipid analysis of the feces was undertaken using the same procedure as in the case of plasma.

RESULTS AND DISCUSSION

Body weights, dietary intakes and food efficiency ratio

The changes in the weights of the rats in each experimental group over the total 4-week period are shown in Table 2. The normal control group showed the greatest increase in body weight in comparison with the initial weight, while the diabetic control group showed the greatest reduction in weight. The normal RD group showed a significantly lower final body weight compared with the normal control group, while the diabetic RD group showed a relatively higher final body weight

Table 2. Initial and final body weights (g), dietary intake (g/day) and food efficiency ratio (FER)

Group	Initial body weight(g)	Final body weight(g)	Dietary intake (g/day)	FER
Normal control	205.4 ± 8.3$^{N.S.}$	337.5 ± 18.4^{a}	17.8 ± 2.4^{a}	0.27 ± 0.19^{a}
Normal RD	203.1 ± 7.3	276.6 ± 15.6^{b}	15.3 ± 1.9^{a}	0.17 ± 0.09a^{b}
Diabetic control	203.8 ± 8.9	187.6 ± 26.7^{c}	28.9 ± 4.2^{b}	-0.02 ± 0.03^{c}
Diabetic RD	204.5 ± 9.1	206.5 ± 23.5^{c}	25.4 ± 3.4^{b}	0.003 ± 0.001^{d}

N.S. : not significant
Values with different superscript within the same column are significantly different at 5% level.

compared with the diabetic control group: in fact, the diabetic RD group maintained its initial weight during the experiment, while the diabetic control group lost weight.

Many previous studies have shown that, if diabetes is induced due to the hyposecretion of insulin caused by administration of STZ, anomalies occur in energy metabolism and body weights are reduced.[19-23] Furuse *et al.*[24] and Fisher et al.[25] reported that both growth and weight decreased in rats in which diabetes had been induced by STZ.[24,25]Also, other studies[26,27] reported that any reduction in the weights of STZ-induced diabetic rats was caused by the atrophy of skeletal muscle due to a relative reduction of the maximum surface area of capillaries where the exchange of solute occurs; furthermore, this reduction in weight was not recovered as easily as when alloxan-induced diabetic rats were studied.

The dietary intakes and the food efficiency ratios for each of the experimental groups are also presented in Table 2. Compared with the normal control, the diabetic groups showed a substantially-increased food intake, and this is thought to be due to polyphagia, a major symptom of diabetes. The food efficiency ratio of the diabetic control group was shown to be negative. There was a significant difference in the food efficiency ratio between the normal control group and the diabetic groups; despite a higher food intake, the diabetic groups achieved signi- ficantly lower final weights than the normal groups. It is thought that this was due to degenerative changes in internal metabolism caused by diabetes.[28] There was a significant difference in the food efficiency ratio between the diabetic control group and the diabetic RD group, with the food efficiency ratio achieving a positive score in the diabetic RD group.

Fasting blood glucose

As shown in Table 3, there was no significant difference in fasting blood glucose levels between the normal control group and the normal RD group. However, compared with the blood glucose levels of the normal control group, the blood glucose levels of the two diabetic groups were significantly increased, by 3.5 times in the diabetic control group and by 2.6 times in the diabetic RD group. In the case of the diabetic groups, administration of the raw diet significantly reduced blood glucose levels.

Table 3. Plasma fasting glucose level

Group	Plasma fasting glucose(mg/dl)
Normal control	127.4 ± 8.3^{a}
Normal RD	130.7 ± 11.8^{a}
Diabetic control	439.8 ± 19.8^{b}
Diabetic RD	328.5 ± 17.0^{c}

Values with different superscript within the same column are significantly different at 5% level.

Any increase in blood glucose levels under diabetes may be caused by resistance to internal insulin,[29] and it was reported that the new synthesis of glucose was increased, and utilization of glucose was decreased, through the inhibited action of pyruvate dehydrogenase.[30-33]

Organ weights

Respective weights of the liver, the pancreas and the kidney per 100 g of the weight of the rat are shown in Table 4. In the case of the diabetic control group, the weight of the liver was significantly increased, and the weight of the pancreas decreased, in comparison with the other groups. This result is in agreement with the results of previous studies[34-36] which showed that the weight of the liver per unit body weight increased due to reductions in body weight resulting from diabetes, rather than to the enlargement of the liver. There was little difference in weight between the livers of the diabetic RD group and the normal groups. The weight of the pancreas was significantly decreased in the case of the diabetic control group in comparison with all the other groups; however, the weights of the pancreas in the diabetic RD group maintained similar levels to those in the normal groups.

Table 4. Organ weights

Group	Liver (g/100 BW)	Pancreas (g/100 BW)	Kidney (g/100 BW)
Normal control	3.45 ± 0.45[a]	0.49 ± 0.18[a]	1.20 ± 0.33[N.S.]
Normal RD	3.66 ± 0.56[a]	0.56 ± 0.23[a]	1.18 ± 0.34
Diabetic control	4.78 ± 0.89[b]	0.40 ± 0.14[b]	1.22 ± 0.27
Diabetic RD	3.76 ± 0.78[a]	0.47 ± 0.20[a]	1.24 ± 0.19

Values with different superscript within the same column are significantly different at 5% level.
N.S. : not significant

Table 5. Hematocrit levels and plasma GOT/GPT

Group	Hematocrit (%)	GOT (unit)	GPT (unit)
Normal control	42.1 ± 5.6 [N.S.]	63.97 ± 12.87[a]	30.27 ± 2.50[a]
Normal RD	43.7 ± 6.3	61.87 ± 8.29[a]	29.14 ± 8.24[a]
Diabetic control	44.2 ± 5.9	114.92 ± 15.30[b]	58.20 ± 11.26[b]
Diabetic RD	45.3 ± 6.5	90.10 ± 11.83[c]	39.24 ± 10.27[a]

N.S. : not significant
Values with different superscript within the same column are significantly different at 5% level.

Table 6. Plasma triglycerides, total cholesterol, HDL-cholesterol, and LDL-cholesterol

Group	Triglycerides (mg/dl)	Total cholesterol (mg/dl)	HDL-cholesterol (mg/dl)A	LDL-cholesterol (mg/dl)
Normal control	35.3 ± 20.8[ac]	66.2 ± 35.9[ab]	17.9 ± 2.3[N.S.]	11.4 ± 4.2[N.S.]
Normal RD	28.7 ± 5.6[a]	54.7 ± 8.8[a]	19.2 ± 5.8	12.1 ± 5.8
Diabetic control	110.8 ± 54.7[b]	169.7 ± 80.8[c]	17.8 ± 2.9	13.7 ± 3.4
Diabetic RD	60.2 ± 25.9[c]	119.5 ± 50.2[cd]	18.2 ± 3.2	10.9 ± 5.5

Values with different superscript within the same column are significantly different at 5% level.
N.S. : not significant

Hematocrit values, and GOT and GPT activity of plasma

Values of hematocrit, and of GOT and GPT activity of the plasma, are shown in Table 5. The hematocrit values were not significantly different between the normal and diabetic groups, and this result is in agreement with previous studies showing that the hematocrit values of rats with diabetes were very similar to that of normal rats.[23,37]

In order to detect the effect of diabetes on the biochemical functions of the liver, the levels of GOT and GPT activities of plasma were measured. When the liver is damaged, these enzymes leak out of liver cells in large quantities so their concentrations in the blood are increased; thus, the levels of these enzymes in the blood can be used as indicators of damage to the liver.[38] In diabetic subjects, various pathological liver conditions are frequently observed; particularly, diabetes induces complications fatal to the liver, such as fatty liver, hepatic cirrhosis and hepatitis.[21] The diabetic control group showed higher values of GOT and GPT in the plasma than other groups. Administration of the raw diet had no effect on the levels of GOT and GPT activity in the normal group, while it significantly reduced activity in the diabetic groups.

Plasma triglycerides, total cholesterol, HDL-cholesterol, and LDL-cholesterol

Results for plasma triglycerides, total cholesterol, HDL-cholesterol, and LDL-cholesterol are shown in Table 6.

Plasma triglyceride levels increased 3.1 and 1.7 times in the diabetic control group and the diabetic RD group, respectively, compared to the normal control group. Hypertriglyceridemia is frequently found in all diabetics, irrespective of the age and the weight.[39] There was no significant difference in plasma triglyceride levels between the normal control group and the normal RD group, whereas triglyceride levels in the diabetic RD group were significantly lower than in the diabetic control group.

A significant difference in levels of total plasma cholesterol was found between normal and diabetic groups, and the administration of the raw diet did not affect these values. Hyperlipidemia, which is characterized by a high level of total cholesterol and trigly-

ceride concentration in plasma, is a complication associated with diabetes.[39,40] When glucosuria is not controlled, the activities of hydroxymethyl glutaryl-CoA (HMG-CoA) reductase are decreased in the liver, and are increased in the intestines, resulting in the increased migration of cholesterol to circulating blood.[41] It is thought that the significantly-higher total cholesterol concentration of the plasma in the diabetic groups, compared with the non-diabetic groups, resulted from the increased migration of cholesterol to circulating blood, due to a reduction in cholesterol metabolism in the liver and an increase in cholesterol synthesis in the intestines. Another study reported that due to the administration of STZ, glycometabolism was not performed smoothly, and that-due to the accumulation of acetyl-CoA-the synthesis of fat was increased so that intrahepatic lipid accumulated to cause serious fatty degeneration, as a result, the outflow of total lipid and triglycerides in blood was increased to bring about an imbalance in lipid metabolism.[42] In the cases of HDL-cholesterol and LDL-cholesterol levels, there were no significant differences between all experimental groups. Another study has reported that an anomaly in lipid metabolism frequently observed in diabetic subjects is that values of triglycerides, total cholesterol and LDL-cholesterol in blood are increased while the value of HDL-cholesterol in blood is decreased.[21]

Triglycerides (TG) and total cholesterol(TC) of the liver

Table 7 presents values for triglycerides and total cholesterol in the liver. Compared with other groups, the diabetic control group showed significantly higher values for both triglycerides and total cholesterol in the liver. In the case of the diabetic groups, the administration of the raw diet maintained the normal values for triglycerides and total cholesterol in the liver. These results are similar to the results reported in previous studies.[21,43,44] However, one previous study found very different results[45], stating that, since glucose fails to be used as an energy source while the fat of the liver and muscle is used as an energy source, liver lipid levels may be curtailed along with a reduction in weight. These results indicate that diabetes may increase the risk of arteriosclerosis and hyperlipidemia.

Table 7. Triglyceride (TG) and total cholesterol (TC) in liver

Group	TG (mg/g liver)	TC (mg/g liver)
Normal control	16.22 ± 2.12^{a}	4.17 ± 0.36^{ab}
Normal RD	17.08 ± 2.87^{a}	4.08 ± 0.97^{a}
Diabetic control	21.16 ± 3.56^{b}	6.98 ± 1.27^{a}
Diabetic RD	18.17 ± 1.65^{a}	3.89 ± 0.29^{b}

Values with different superscript within the same column are significantly different at 5% level.

Total lipids (TL), triglycerids (TG) and total cholesterol (TC) of feces

The lipid content of the feces in each experimental group is shown in Table 8. The total lipid content of feces was significantly lower in the diabetic control group compared with the other groups. In the case of the groups in which diabetes was induced, the outputs of total lipids, triglycerides, and total cholesterol were significantly higher in the raw diet group. The raw diet group gave values of total lipids, triglycerides, total cholesterol of feces, which were similar to those of either the normal control group or the normal experimental group. Our results are in agreement with previous results which showed that raw diets promote excretion by curtailing internal resorption of triglycerides and cholesterol.[46-50] The raw diet may further inhibit internal resorption of triglycerdies and cholesterol by combining directly with, and increasing excretion of, intestinal triglycerides and cholesterol.

CONCLUSION

In short, in the non-diabetic groups, final body weights were significantly lower in the group fed the raw diet compared to the group fed the regular powdered chow diet. In the STZ-induced diabetic groups, the following significant effects of feeding the raw diet rather than the regular powdered chow diet, were observed: an increase in food efficiency ratio, a reduction in fasting blood glucose concentration, a reduction in the liver weight per unit body weight, and an increase in the pancreas weight per unit body weight; further, the raw diet resulted in decreases in values of GOT/GPT activities and TG levels

Table 8. Total lipid (TL), triglycerides (TG), and total cholesterol (TC) in feces

Group	TL (mg/g)	TG (mg/g)	TC (mg/g)
Normal control	108.85 ± 20.56^{ab}	29.15 ± 2.52^{a}	62.87 ± 7.68^{ab}
Normal RD	190.25 ± 30.28^{a}	27.48 ± 5.79^{a}	59.28 ± 8.94^{a}
Diabetic control	135.27 ± 17.53^{b}	5.94 ± 2.02^{b}	10.28 ± 8.12^{b}
Diabetic RD	219.18 ± 16.84^{a}	31.97 ± 4.12^{a}	64.98 ± 6.12^{a}

Values with different superscript within the same column are significantly different at 5% level.

of plasma, decreases in levels of TG and total cholesterol in the liver, and increases in the levels of total lipids, triglycerids, and total cholesterol in the feces.

Further to this study, it is expected that a study on humans of the clinical effects of consuming raw diets will be conducted in the future. It may eventually be possible to recommend raw diets for treating and preventing adult diseases, as an alternative to the popular diets of today, thereby contributing greatly to improvements in dietary behavior and public health.

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