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# Daily consumption of banana marginally improves blood glucose and lipid profile in hypercholesterolemic subjects and increases serum adiponectin in type 2 diabetic patients

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In this study, we explored the effects of consumption of banana in thirty hypercholesterolemic and fifteen type 2 diabetic subjects. They were given a daily dose of 250 or 500 grams of banana for breakfast for 12 weeks. Fasting serum lipid, glucose and insulin levels were measured initially as well as every 4 weeks. Daily consumption of banana significantly lowered fasting blood glucose (from 99±7.7 to 92±6.9 and 102±7.3 to 92±5.7 mg·dL$^{-1}$ ($p<0.05$) after consuming banana 250 or 500 g/day for 4 wk, respectively) and LDL-cholesterol/HDL-cholesterol ratio (from 2.7±0.98 to 2.4±0.85 and 2.8±0.95 to 2.5±0.79, $p<0.005$) in hypercholesterolemic volunteers. Analysis of blood glycemic response after eating banana showed significantly lower 2 h-postprandial glucose level compared to baseline in hypercholesterolemic volunteers given a dose of 250 g/day. The changes of blood glucose and lipid profile in diabetic patients were not statistically significant, but for plasma levels of adiponectin, there were significantly increased (from 37.5±9.36 to 48.8±7.38 ng·ml$^{-1}$, $p<0.05$) compared to baseline. Although it remains to be confirmed with larger group of volunteers, this pilot study has demonstrated that daily consumption of banana (@ 250 g/day) is harmless both in diabetic and hypercholesterolemic volunteers and marginally beneficial to the later.

**Keywords:** Cholesterol, Diabetes, Hypocholesterolemics, Hypoglycemics, Metabolic syndrome, *Musa* spp., Obesity, Plantain

Modern day lifestyles increase the risk of metabolic syndrome including abdominal obesity, high blood pressure, high fasting blood glucose and an unfavorable lipid profile, which is characterized by high plasma levels of total cholesterol (TC), LDL-cholesterol (LDL-C), triglyceride (TG), but low levels of HDL-cholesterol (HDL-C)[1]. Further, it also leads to coronary artery diseases, stroke and type 2-diabetes. Adiponectin, a key adipocytokine involved in metabolic syndrome, is known to possess anti-atherogenic, anti-inflammatory and anti-diabetic properties. Low plasma concentrations of adiponectin has been shown to be associated with obesity-related diseases, including atherosclerotic cardiovascular diseases, type 2 diabetes mellitus, hypertension and unfavorable lipid profile[2,3].

Dietary modification is recommended as a first approach to reduce risk factors associated with metabolic syndrome with the specific goals of achieving a favorable lipid profile and normalized blood glucose level. Daily consumption of various vegetables, fruits or their products has been demonstrated to deliver positive results[4]. Several studies have advocated a "food-to-eat" approach rather than a "food-to-restrict" approach for risk reduction and improved dietary adherence[5]. In this context, banana (*Musa* sp.) fruits with its minerals (iron, potassium, calcium, magnesium, phosphorus, sulphur and copper)[6], vitamins (A, C, B6 and B12, tryptophan, flavonoid and phenolic compounds) and dietary fiber provide a potential source to depend up on. Earlier workers have demonstrated the hypocholesterolemic[7,8], hypoglycemic[9,10] and antioxidant[11,12] activities of banana both *in vivo* and *in vitro*. Banana is rich in potassium thus may potentially help reducing the risk of stroke[13,14]. Banana has also been projected as a potential source for treatment of shigellosis[15], gastric ulcer[16-19], diabetes mellitus[20-22], depression and oxidative stress induced neuro-degenerative disease[23]. In the present study, we evaluated the effect of daily banana intake on lowering the risk factors associated with metabolic

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syndrome. We hypothesized that daily consumption of banana may lead to an improvement of individual's insulin sensitivity and lipid profile.

## Materials and Methods

### *In vitro* experiments

*Sample preparation*—Three sets each of banana cultivars commonly consumed in Thailand; "Hoam Thong" [*Musa* (AAA group)] "Num-War" (*Musa sapientum* Linn.) and "Khai" [*Musa* (AA group)] were randomly selected from three local markets. Unripe bananas were peeled, edible parts chopped off and frozen in liquid nitrogen, while the remaining parts were allowed to ripe before freezing. Subsequently, all fruits were lyophilized at −45 °C for 60 h, milled and stored at −70 °C until analysis.

*Determination of antioxidant capacity*—Dried milled banana were extracted with 80% methanol and concentrated in a rotary vacuum evaporator at 50 °C under reduced pressure. Ferric reducing ability (FRAP) assay was applied according to Benzie and Strain[24] to measure the total reducing power of electron-donating substances. Briefly, 6 ml of freshly prepared FRAP reagent (0.1 M acetate buffer: 0.02 M $FeCl_3$: 0.01 M TPTZ = 10: 1: 1) was mixed with 20 µl of dried fruit extract, incubated at 37 °C for 30 min and absorbance was recorded at 593 nm. FRAP values were obtained by comparing with standard curves created by Trolox (0-35 µg) and reported as mg Trolox equivalent per 10 gram of sample (dry wt).

The antioxidant capacity of banana extract was also measured based on its ability to quench the long-lived ABTS radical cation ($ABTS^{\bullet+}$)[25]. $ABTS^{\bullet+}$ was generated by mixing 7 mM ABTS stock solution with 2.45 mM potassium persulphate (final concentration) and allowing the mixture to stand in the dark at room temperature for 12-16 h before use. The $ABTS^{\bullet+}$ solution was diluted with deionized water and 95% ethanol (1:1) to an absorbance of 0.70 (± 0.02) at 734 nm. Twenty microliters of the banana extract was mixed with 6 ml of the diluted $ABTS^{\bullet+}$ solution. The decreasing rate of absorbance, which represents the rate of $ABTS^{\bullet+}$ quenching by antioxidant in the extract, was recorded at 1 min after mixing. Results were reported as mg Trolox equivalent (TE) per gram of sample (dry wt).

*Determination of indigestible fraction*—The indigestible fraction was determined according to Saura-Calixto *et al*[26]. Briefly, 300 mg of lyophilized fruit was digested with pepsin in HCl-KCl buffer, pH 1.5 for 1 h at 40 °C further digested by α-Amylase in Tris-maleate buffer (0.1 M, pH 6.9) at 37 °C for 16 h. Samples were centrifuged (15 min, 3000 g) and supernatants removed. Residues were washed twice with 10 mL of distilled water and all supernatants combined. The residues were dried overnight at 105 °C and quantified gravimetrically as the insoluble indigestible fraction (iIF). Supernatants were dialyzed against water for 48 h at 25 °C (12000-14000 MWCO; Dialysis Tubing Visking, Medicell International Ltd., London, UK). Dialysates were then hydrolyzed with 1.0 M sulfuric acid at 100 °C for 90 min, and the soluble indigestible fraction (sIF) was measured.

### *In vivo* experiment

*Subjects*—Thirty hypercholesterolemic volunteers and 15 type 2 diabetes patients according to the WHO criteria were recruited into the study. All hypercholesterolemic subjects had total serum cholesterol higher than 200 $mg \cdot dL^{-1}$ (5.17 $mmol \cdot L^{-1}$) and were not receiving lipid-lowering medications. All subjects had maintained stable weight during the three month prior to enrollment and did not have renal/liver diseases or heart problems. Pregnant or breast feeding women were not included. All fifteen diabetic patients were receiving oral hypoglycemic agents (12 metformin alone; 1 each on sitagliptin and metformin; glucophage; and glipizide, respectively). Five diabetic patients were taking lipid lowering drugs. A criterion for exclusion was modification of medical treatment during the study. All subjects provided written informed consent and the study was approved by the Ethics Committee at the Faculty of Associated Medical Sciences, Chiang Mai University.

*Experimental design*—Prior to this study, preliminary investigation was performed with 14 healthy volunteers, randomly divided into test and control groups (7 volunteers each). The test group had banana ($500\ g \cdot day^{-1}$) at any time of the day, while the control group maintained their usual diet styles without banana throughout the 8 wk study. After the study, the test group showed significantly reduced fasting plasma glucose level. The preliminary data also indicated hypocholesterolemic effect of banana (data not shown). Moreover, the body weight and BMI values of test group consuming $500\ g \cdot day^{-1}$ of banana daily significantly increased, possibly due to the high content of carbohydrate and sugar in banana. Therefore, an additional test group with lower dose of banana ($250\ g \cdot day^{-1}$) was also included in the actual

study. All the volunteers in test groups (both 250 $g \cdot day^{-1}$ and 500 $g \cdot day^{-1}$) were requested to consume banana before having their breakfast and also to maintain their body weight and total calorie intake throughout the study.

Hypercholesterolemic volunteers were randomly assigned to two groups of 15 each and were given banana for breakfast at either 250 (about 1 banana) or 500 $g \cdot day^{-1}$ (about 2 bananas), respectively for 12 weeks followed by washout period of 8 weeks. The third group of 15 type 2 diabetic patients were given a dose of 250 $g \cdot day^{-1}$ (already proven to help reducing blood cholesterol and glucose) banana for 4 weeks followed by washout period of 8 weeks. The amount of banana chosen for this study was based on the tolerable amount that can be consumed daily by a person and also our preliminary investigation. All participants maintained their bodyweight, diet and normal exercise habits. Overnight fasted venous blood samples were collected at baseline and every 4 weeks as well as after a washout period of 8 weeks. Plasma and sera were obtained and stored at −70 °C until analysis. Body weight and blood pressure were also measured on every visit at approximately the same time (7-8 a.m.) of the day.

*Dietary assessment*—To monitor dietary intake, the subjects were put on a 3-day food record prior to each study visit. All items and portions of food consumed including name and method of cooking on 2 'weekdays' and one 'weekend' day were recorded. Average intake of energy, protein, fat carbohydrate, dietary fiber, magnesium and cholesterol were calculated using "Inmucal-nutrient"(Mahidol University, Thailand).

*Clinical Laboratory Assays*—Fasting blood samples were obtained at the onset of the study and every 4 weeks throughout the study. Samples of plasma and sera were kept at −70 °C until analysis. Sodium fluoride plasmas were analyzed for glucose using the hexokinase-based method (Roche Diagnostic Systems). Serum samples were analyzed for total cholesterol, HDL-C, LDL-C and triglyceride were analyzed using Olympus AU400 chemistry auto-analyzer with reagent kits purchased from Roche Diagnostic Systems, Inc. All samples were subjected to analysis at the same time in order to avoid imprecision of the assay. The serum samples were also analyzed for parameters of liver function [alanine transaminase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), total bilirubin, direct bilirubin and albumin] and kidney function [blood urea nitrogen, (BUN) and creatinine] at baseline and at the end of the study.

Fasting serum insulin (Mercodia Insulin ELISA, Uppsala, Sweden) levels were determined using ELISA. The assay has detection limit of 1 $mU \cdot L^{-1}$ with intra- and inter-assay coefficients of variation of 1.5-3.2% and 3.3-5.0%, respectively. Insulin homeostasis modeling assessment (HOMA)[27] was utilized as an estimate of % beta-cell function and insulin resistance (IR). Fasting blood glucose and insulin levels were also used to calculate Quantitative Insulin-Sensitivity Check Index (QUICKI)[28].

*Glycemic response examination*—Blood glucose level in response to eating 250 g of banana at breakfast was determined prior and during the last weeks of banana consumption using finger-prick blood monitor (TerumoFinetouch® Blood Glucose Meter). Subjects had 10 min to consume their banana and glucose concentration was determined before and at 15, 30, 60, 120, 150 and 180 min following initiation of breakfast. At least two blood glucose readings were taken at each time point. A third measurement was taken if glucose values were > $5mg \cdot dL^{-1}$ apart. Increment areas under the curve for glucose response curves were determined using methodologies outlined by Wolver & Jenkins[29].

Fasting serum adiponectin concentrations were measured by and ELISA according to manufacturer's instructions (US biological, Massachusetts, USA) with intra- and inter-assay coefficients of variation of 1.4-2.6% and 2.8-4.5%, respectively.

*Statistical analyses*—Statistical analysis was performed using SPSS version 15 for windows (SPSS Inc, Chicago, III). Descriptive values were presented as mean ± SD. Comparisons between groups were assessed using the Mann-Whitney U test and pair-wise comparisons were tested using the Wilcoxon singed-rank test.

## Results

### *In vitro* experiment

*In vitro*—experiments have shown the most suitable banana cultivar to be used for *in vivo* experiment. The antioxidant capacity of banana assessed both with ABTS and FRAP assays was consistent and comparable with all three cultivars. Ripe banana possessed significantly higher antioxidant capacity than unripe bananas (Table 1). Unripe bananas contained a high percentage of insoluble indigestible fractions (iIF) whereas ripe

Table 1—Percentage of indigestible fraction (IF) and antioxidant capacities of banana cultivars (*Hoam-Thong*, *Khai*, *Num-War*) at different ripening stage (ripe, unripe)

| Cultivar of banana | | Soluble IF (%) mean (SD) | Insoluble IF (%) mean (SD) | Antioxidant capacity assessed by ABTS assay (mM TE/ 10g dried weight) | Antioxidant capacity assessed by FRAP assay (mM TE/ 10g dried weight) |
|---|---|---|---|---|---|
| *Hoam-Thong*: | unripe | 11.2 (1.99) | 77.3 (3.34) | 692.8 (6.3) | 148.9 (5.4) |
| | ripe | 20.0 (2.18)* | 23.2 (2.46)* | 1254.0 (213.0)* | 222.1 (47.9)* |
| *Khai*: | unripe | 6.8 (0.84) | 81.9 (2.66) | 790.0 (9.0) | 164.8 (5.5) |
| | ripe | 18.5 (2.95)* | 22.9 (0.42)* | 1194.6 (307.8)* | 190.8 (53.7)* |
| *Num-War*: | unripe | 5.7 (0.67) | 83.0 (5.28) | 312.0 (6.9) | 67.0 (2.7) |
| | ripe | 19.0 (3.08)* | 20.7 (1.56)* | 979.9 (104.1)* | 189.2 (1.0)* |

* $P< 0.05$

Table 2—Volunteer characteristics at baseline and during the study

| Volunteer groups | Age (years) Mean (SD) | Body weight (kg) Mean (SD) | BMI ($kg\cdot(m^2)^{-1}$) Mean (SD) | Blood pressure | |
|---|---|---|---|---|---|
| | | | | Systolic (mmHg) Mean (SD) | Diastolic (mmHg) Mean (SD) |
| Healthy hypercholesterolemic volunteers consuming banana 250g·day$^{-1}$ (n=15) | | | | | |
| baseline | 44.8 (10.31) | 61.5 (10.9) | 24.8 (4.03) | 120 (9.6) | 79 (7.8) |
| after 4 weeks | | 61.3 (10.7) | 24.7 (4.29) | 120 (12.4) | 77 (10.9) |
| after 8 weeks | | 61.2 (10.9) | 24.6 (4.41) | 122 (6.3) | 79 (8.5) |
| after 12 weeks | | 61.5 (10.7) | 24.8 (4.28) | 124(12.1) | 82 (9.9) |
| after washout for 8 weeks | | 61.0 (10.5) | 24.5 (4.29) | 127 (9.3) | 82 (11.9) |
| Healthy hypercholesterolemic volunteers consuming banana 500g·day$^{-1}$ (n=15) | | | | | |
| baseline | 43.1 (7.51) | 59.6 (11.8) | 24.0 (3.94) | 124 (14.4) | 79 (11.8) |
| after 4 weeks | | 59.8 (11.6) | 24.3 (4.11) | 125 (15.2) | 78 (12.6) |
| after 8 weeks | | 59.6 (11.8) | 24.2 (4.09) | 126 (12.9) | 80 (11.7) |
| after 12 weeks | | 59.3 (12.1) | 24.1 (4.24) | 124 (16.3) | 78 (15.5) |
| after washout for 8 weeks | | 59.3 (11.9) | 24.1 (4.17) | 124 (12.0) | 78 (10.5) |
| Diabetic patients consuming banana 250g·day$^{-1}$ (n=15) | | | | | |
| baseline | 52.8 (5.23) | 61.8 (13.3) | 25.8 (4.71) | 134 (10.8) | 81 (6.6) |
| after 4 weeks | | 62.3 (13.0) | 25.9 (4.59) | 132 (24.6) | 82 (16.4) |
| after washout for 8 weeks | | 61.7 (12.8) | 25.7 (4.54) | 134 (21.4) | 78 (7.6) |

bananas had a high percentage of soluble indigestible fractions (sIF). sIF was found in the range of 5.7-11.2% in unripe banana and ripe banana had iIF in the range of 20.7-23.2% . Hoam-Thong banana contained the highest percentage of sIF.

***In vivo* experiment**

*Subject characteristics*—Mean body weight and BMI of all groups of volunteers after the experiment did not significantly differ from baseline (Table 2). Throughout the study, values of kidney and liver function indices remained within the normal range for all subjects, and were not significantly different from the baseline levels (Table 3).

*Dietary intake*—All groups of volunteers showed a significant increase in magnesium intake during the period of banana consumption. The calorie intake in diabetic patients consuming 250 g of banana a day and hypercholesterolemic volunteers consuming 500 g of banana a day increased significantly compared to baseline value (Table 4). Similarly, dietary fiber intake showed increased values in both groups of hypercholesterolemic volunteers. However, in diabetic patients it was not statistically significant.

*Effect of banana intake on lipid profile*—The mean serum levels of triglyceride, total cholesterol (TC), HDL-C and LDL-C at baseline, every 4 weeks after starting banana consumption and after the washout period of 8 weeks are shown in Table 5. Although the mean levels of TC and LDL-C were reduced from the baseline values in all groups of volunteers consuming

Table 3—Results of liver function tests and renal function test of volunteers at baseline and at the end of banana consumption

| Tests | Reference range | Healthy hypercholesterolemic volunteers consuming banana 250g·day$^{-1}$ (n=15) | | Healthy hypercholesterolemic volunteers consuming banana 500g·day$^{-1}$ (n=15) | | Diabetic patients consuming banana 250g·day$^{-1}$ (n=15) | |
|---|---|---|---|---|---|---|---|
| | | Baseline Mean (SD) | End Mean (SD) | Baseline Mean (SD) | End Mean (SD) | Baseline Mean (SD) | End Mean (SD) |
| BUN (mg·dL$^{-1}$) | 0.7-18.0 | 12.4 (2.85) | 12.5 (3.54) | 12.6 (2.82) | 12.4 (2.21) | 14.9 (4.23) | 14.3 (6.2) |
| Creatinine (mg·dL$^{-1}$) | 0.6-1.3 | 0.8 (0.14) | 0.7 (0.13) | 0.8 (0.14) | 0.8 (0.17) | 0.8 (0.21) | 0.7 (0.18) |
| Albumin (g·dL$^{-1}$) | 3.5-5.0 | 4.6 (0.27) | 4.4 (0.22) | 4.4 (0.21) | 4.3 (0.23) | 4.1 (0.47) | 3.9 (0.46) |
| Total bilirubin (mg·dL$^{-1}$) | 0.2-1.1 | 0.9 (0.42) | 0.8 (0.38) | 0.7 (0.25) | 0.7 (0.25) | 0.6 (0.24) | 0.4 (0.14) |
| Direct bilirubin (mg·dL$^{-1}$) | 0.0-0.3 | 0.1 (0.08) | 0.1 (0.08) | 0.1 (0.05) | 0.1 (0.04) | 0.1 (0.06) | 0.1 (0.03) |
| AST (U·L$^{-1}$) | 10-42 | 20 (4.7) | 22 (6.2) | 20 (4.8) | 22 (5.9) | 24 (6.9) | 23 (5.8) |
| ALT (U·L$^{-1}$) | 10-40 | 16 (3.8) | 22 (8.4) | 18 (7.5) | 21 (8.6) | 26 (14.6) | 15 (5.6) |
| ALP (U·L$^{-1}$) | 32-92 | 67 (15.6) | 75 (18.9) | 58 (14.9) | 62 (15.1) | 69 (23.8) | 68 (19.8) |

BUN, blood urea nitrogen; AST, aspartate aminotransferase; ALT, alanine aminotransferase; ALP, alkaline phosphatase

Table 4—Dietary nutrient intakes of volunteers at baseline and during the study

| Volunteer groups | Total energy (Kcal·day$^{-1}$) Mean (SD) | Carbohydrate (g) Mean (SD) | Fat (g) Mean (SD) | Protein (g) Mean (SD) | Cholesterol (mg) Mean (SD) | Dietary fiber (g) Mean (SD) | Magnesium (mg) Mean (SD) | Selenium (µg) Mean (SD) |
|---|---|---|---|---|---|---|---|---|
| Healthy hypercholesterolemic volunteers consuming banana 250g·day$^{-1}$ (n=15) | | | | | | | | |
| baseline | 1813 (470) | 224 (70) | 66 (18) | 78 (24) | 253 (157) | 1.7 (3.6) | 27.3 (34.25) | 0.4 (1.33) |
| after 4 weeks | 1805 (407) | 264 (63) | 53 (16)* | 77 (26) | 258 (160) | 12.7 (3.4)** | 82.1 (31.1)* | 1.2 (3.11) |
| after 8 weeks | 1907 (508) | 273 (66) | 54 (19)* | 74 (24) | 263 (227) | 12.4 (3.1)** | 81.6 (34.0)* | 1.9 (3.20) |
| after 12 weeks | 2069 (565) | 285 (62) | 61 (26) | 85 (29) | 233 (118) | 12.0 (4.4)* | 82.6 (29.6) ** | 1.4 (2.45) * |
| after washout for 8 weeks | 2079 (711) | 227 (60) | 75 (28) | 83 (26) | 276 (186) | 9.6 (4.1) | 33.1 (29.1) | (1.37) |
| Healthy hypercholesterolemic volunteers consuming banana 500g·day$^{-1}$ (n=15) | | | | | | | | |
| Baseline | 1800 (640) | 244 (76) | 57(28) | 75 (33) | 199 (167) | 8.9 (5.0) | 33.0 (49.79) | 0.5 (1.33) |
| after 4 weeksafter 8 weeks | 2035 (321)* | 308 (40)* | 55 (21) | 80 (30) | 230 (128) | 18.3 (4.6)** | 143.3 (38.14)** | 1.1 (4.67) |
| after 12 weeks | 2140 (351)* | 317 (40)* | 58 (21) | 83 (23) | 257 (110) | 18.0 (4.7)** | 145.2 (50.94)** | 2.1 (3.96) * |
| after washout for 8 weeks | 2297 (609)* | 325 (79)* | 66 (22) | 96 (32) | 278 (186) | 17.7 (9.6)* | 132.8 (65.2)** | 0.4 (0.31) |
| | 1936 (575) | 233 (43) | 72 (30) | 82 (32) | 354 (286)* | 11.2 (4.6) | 33.0 (33.56) | 4.0 (8.45) |
| Diabetic patients consuming banana 250g·day$^{-1}$ (n=15) | | | | | | | | |
| baseline | 1800 (382) | 250 (69) | 53 (20) | 78 (13) | 239 (112) | 12.6 (5.2) | 153.8 (129.45) | 0.5 (0.67) |
| after 4 weeks | 2203 (542)* | 312 (44)* | 62 (32) | 97 (34) | 272 (143) | 13.9 (4.1) | 251.4 (78.16)* | 1.83 (2.69)* |
| after washout for 8 weeks | 1902 (411) | 250 (55) | 61 (20) | 88 (21) | 268 (130) | 13.5 (4.9) | 137.9 (119.65) | 0.2 (0.43) |

*P* values: *< 0.05; **<0.005

banana, it was statistically significant ($P$<0.05) only in the group of hypercholesterolemic volunteers consuming 500 g of banana a day. The mean values of HDL-C slightly increased from baseline in all groups. However, it was statistically significant only in a group of hypercholesterolemic volunteers after consuming 250 g·day$^{-1}$ of banana for 12 weeks. Interestingly, the ratio of TC/HDL-C and LDL-C/HDL-C in both groups of healthy hypercholesterolemic volunteers showed significant reduction from baseline compared to after 4 weeks of banana consumption and continued to decrease throughout the study. Moreover, after the washout period of 8 weeks the ratio of TC/HDL-C and LDL-C/HDL-C returned to baseline levels thus indicating the specificity of the banana effect. Although the same pattern of changes in lipid profile was observed in the diabetic patients it was not statistically significant.

*Effect of banana intake on glucose metabolism*—After 4 weeks of banana consumption, fasting plasma glucose levels of hypercholesterolemic volunteers significantly decreased ($P$<0.05 by Wilcoxon sign rank test) by about 10% of the baseline values and

Table 5—Effect of banana consumption on lipid profile

| Volunteer groups | Triglyceride (mg/dL) Mean (SD) | Total cholesterol (TC) (mg/dL) Mean (SD) | HDL-C (mg/dL) Mean (SD) | LDL-C (mg/dL) Mean (SD) | TC/ HDL-C ratio Mean (SD) | LDL-C/ HDL-C ratio Mean (SD) |
|---|---|---|---|---|---|---|
| Healthy hypercholesterolemic volunteers consuming banana 250g·day$^{-1}$ (n=15) | | | | | | |
| baseline | 117 (48.5) | 231 (33.2) | 62 (13.4) | 160 (33.9) | 3.9 (1.09) | 2.7 (0.98) |
| after 4 weeks | 127 (64.2) | 224 (33.1) | 65 (14.8) | 144 (29.7) | 3.6 (1.04)** | 2.4 (0.85)** |
| after 8 weeks | 119 (71.9) | 223 (39.1) | 65 (15.6) | 151 (34.1) | 3.6 (1.29)* | 2.4 (0.81)* |
| after 12 weeks (end) | 135 (65.8) | 220 (27.3) | 71 (19.1)* | 142 (32.5) | 3.4 (1.68)* | 2.1 (0.70)** |
| after washout for 8 weeks | 144 (89.3) | 219 (32.8) | 56 (15.9)* (¶) | 153 (31.6) | 4.1 (1.06)*(¶) | 2.9 (1.21)**(¶) |
| Healthy hypercholesterolemic volunteers consuming banana 500g·day$^{-1}$ (n=15) | | | | | | |
| baseline | 132 (59.7) | 241 (37.8) | 63 (9.1) | 170 (44.1) | 3.9 (0.84) | 2.8 (0.95) |
| after 4 weeks | 118 (38.0) | 231 (36.1)* | 65 (11.1) | 156 (38.1) | 3.6 (0.77)** | 2.5 (0.79)** |
| after 8 weeks | 137 (60.3) | 222 (36.2)* | 65 (8.7) | 159 (43.5) | 3.5 (0.83)** | 2.6 (0.86) |
| after 12 weeks (end) | 145 (79.9) | 223 (31.5)* | 69 (10.5) | 162 (34.1) | 3.4 (0.91)** | 2.5 (1.38)* |
| after washout for 8 weeks | 130 (55.1) | 221 (37.8)* | 56 (8.5)*(¶) | 162 (44.9) | 4.1 (1.12)*(¶) | 3.1 (1.25)*(¶) |
| Diabetic patients consuming banana 250g·day$^{-1}$ (n=15) | | | | | | |
| baseline | 174 (130.4) | 163 (45.6) | 37 (10.2) | 107 (36.6) | 4.5 (1.41) | 3.0 (1.03) |
| after 4 weeks (end) | 187 (134.8) | 153 (42.7) | 37 (12.6) | 101 (35.1) | 4.3 (0.97) | 2.8 (0.82) |
| after washout for 8 weeks | 182 (154.4) | 165 (45.5)*(¶) | 41 (13.0)**(¶) | 105 (26.5) | 4.1 (1.14) | 2.6 (0.57)* |

*Mean values were significantly different from the "baseline" mean values". *P* values: *<0.05, **<0.005; (¶) in comparison to "end" mean values"

continued to decrease during the entire period of banana consumption as well as the washout period (Table 6). Although the mean value (±SD) of fasting plasma glucose in diabetic patients reduced from 131 (± 44.8) to 120 (± 30.2) mg·dL$^{-1}$, it was not statistically significant. No significant change of fasting insulin levels was observed except the group of hypercholesterolemic volunteers that had 250 g·day$^{-1}$ of banana for 12 weeks. An increase in insulin sensitivity, as revealed by a reduction of HOMA-IR concomitantly with increased QUICKI value, was observed in hypercholesterolemic volunteers after consuming 500 g of banana a day for 12 weeks and after the washout period. The percentage of beta-cell function measured by HOMA showed significant increase in hypercholesterolemic volunteers. Although glycemic response did not show any significant difference, there was a significant reduction of 2 h postprandial glucose level in hypercholesterolemic volunteers on 250 g·day$^{-1}$ of banana after 12 weeks (Table 6).

*Effect of banana intake on plasma levels of adiponectin*—Serum level adiponectin at baseline and at the end of the banana consumption period did not differ significantly in hypercholesterolemic volunteers. Diabetic patients had less serum adiponectin compared to healthy hypercholesterolemic volunteers. After 4 weeks of banana consumption, the diabetic patients possessed significantly increased serum level adiponectin (Table 7).

## Discussion

Indigestible Fractions (IF) is the part of plant food that escape digestion and absorption in the small intestine, but are susceptible to bacterial fermentation in the colon[26]. Although sIF and iIF are undigested and not absorbed into the bloodstream, only sIF, but not iIF, exhibited the ability to reduce blood cholesterol level and reduce the risk of heart diseases[30,31]. In our study, the ripe "*Hoam-Thong*" variety was chosen for further *in vivo* investigation as they had high level of antioxidant activity and high soluble indigestible fraction compared to the other two varieties viz. "*Num-War*" and "*Khai*".

Banana possesses an *in vitro* binding capacity of bile acid comparable to a FDA-approved oat bran cereal for lowering blood cholesterol[32]. It is also a good source of flavonoids[33]. Flavonoids from banana have been demonstrated to reduce concentrations of cholesterol, phospholipids, free fatty acids, and triglycerides in the serum, liver, kidney, and brain of the experimental animals[34].

The hypocholesterolemic effect of banana consumption in animal model was first reported by Usha *et al* (1984). Rats given neutral-detergent fiber (NDF) prepared from unripe bananas showed significantly lower levels of serum cholesterol and triglyceride in both cholesterol-containing and

Table 6—Effect of banana consumption on glucose metabolism and indices of insulin resistivity

| Volunteer groups | Fasting blood glucose (mg/dL) | | Fasting blood insulin (mU/L) | | HOMA-IR Mean (SD) | HOMA-β Mean (SD) | QUICKI Mean (SD) | Glycemic response (IAUC, mg.min/dL) Mean (SE) | 2hr-postprandial glucose (mg/dL) Mean (SD) |
|---|---|---|---|---|---|---|---|---|---|
| | Mean (SD) | Range | Mean (SD) | Range | | | | | |
| Healthy hypercholesterolemic volunteers consuming banana 250g·day$^{-1}$ (n=15) | | | | | | | | | |
| baseline | 99 (7.7) | 85-114 | 3.1 (1.6) | 0.92-6.85 | 0.76 (0.38) | 32.9 (24.2) | 0.42 (0.044) | 2817 (354) | 96 (11.5) |
| after 4 weeks | 92 (6.9)* | 80-105* | 3.5 (1.8) | 1.17-7.2 | 0.81 (0.45) | 42.6 (20.6) | 0.41 (0.044) | | |
| after 8 weeks | 88 (8.3)* | 73-107* | 4.1 (3.2) | 0.77-13.84 | 0.94 (0.86) | 55.7 (28.2)** | 0.42 (0.058) | | |
| after 12 weeks (end) | 92 (8.5)* | 83-112* | 4.3 (2.0)* | 2.31-9.67 | 1.02 (0.59) | 52.4 (13.6)* | 0.39 (0.029) | 2573 (300) | 84 (10.0)** |
| after washout for 8 weeks | 90 (7.6)* | 78-102* | 3.3 (2.0) ¶ | 0.55-9.03 | 0.74 (0.45) | 46.6 (37.2)* | 0.42 (0.059) | | |
| Healthy hypercholesterolemic volunteers consuming banana 500g·day$^{-1}$ (n=15) | | | | | | | | | |
| baseline | 102 (7.3) | 93-119 | 3.3.(2.1) | 0.93-7.49 | 0.85 (0.58) | 30.0 (17.3) | 0.42 (0.053) | 2749 (389) | 93 (15.7) |
| after 4 weeks | 92 (5.7)* | 80-100* | 3.2 (1.7) | 0.87-7.86 | 0.74 (0.41) | 39.5 (19.0)* | 0.42 (0.045) | | |
| after 8 weeks | 91 (10.1)* | 76-115* | 3.7 (2.5) | 0.55-9.74 | 0.87 (0.66) | 47.8 (26.3)* | 0.42 (0.063) | | |
| after 12 weeks (end) | 91 (7.6)* | 81-109* | 2.8 (1.6) | 0.34-5.9 | 0.64 (0.41)* | 36.9 (17.8) | 0.45 (0.087)* | 3719 (853) | 95 (31.3) |
| after washout for 8 weeks | 90 (7.6)* | 83-111* | 2.8 (1.7) | 0.6-6.71 | 0.66 (0.43)* | 36.1 (20.2) | 0.43 (0.061) | | |
| Diabetic patients consuming banana 250g·day$^{-1}$ (n=15) | | | | | | | | | |
| baseline | 131 (44.8) | 83-222 | 14 (16.9) | 0.52-65 | 4.2 (5.9) | 111.8 (156.5) | 0.34 (0.062) | 5511 (660) | 182 (66.8) |
| after 4 weeks (end) | 120 (30.2) | 83-201 | 16.9 (17.6) | 4.8-61 | 5.0 (5.9) | 152.4 (247.6) | 0.32 (0.034) | | |
| after washout for 8 weeks | 130 (39.0) | 84-226 | 15.1 (16.9) | 2.4-62 | 4.9 (5.5) | 90.6 (99.6) | 0.33 (0.051) | 6557 (1027) | 155 (59.8) |

*Mean values were significantly different from the "baseline" mean values". *P* values: *<0.05, **<0.005; ¶ in comparison to "end" mean values"

Table 7—Effect of banana consumption on plasma levels of adiponectin

| Volunteer groups | Fasting serum levels of adiponectin (ng·ml$^{-1}$) | |
|---|---|---|
| | Mean (SD) | range |
| Healthy hypercholesterolemic volunteers consuming banana 250g·day$^{-1}$ (n=15) | | |
| Baseline | 44.8 (7.17) | 32.9-56.2 |
| after 12 weeks (end) | 45.3 (10.1) | 33.2-57.4 |
| Healthy hypercholesterolemic volunteers consuming banana 500g·day$^{-1}$ (n=15) | | |
| baseline | 36.9 (2.50) | 33.0-39.7 |
| after 12 weeks (end) | 39.6 (4.65) | 33.4-46.9 |
| Diabetic patients consuming banana 250g·day$^{-1}$ (n=15) | | |
| baseline | 37.5 (9.36) | 27.9-53.4 |
| after 4 weeks (end) | 48.8 (7.39)* | 36.4-57.8* |

* $P$ <0.05

cholesterol-free diets compared to rats fed on a fiber-free diet, while NDF from ripe bananas had no such effect[8]. However, Horigome *et al*[7] reported that freeze-dried banana pulp, both unripe and ripe, showed a marked cholesterol lowering effect when incorporated into a diet in rats fed on a cholesterol-containing diet, while the banana pulp dried in a hot-air oven (65 °C) had no effect.

Our study has demonstrated that daily intake of ripe banana significantly reduced the ratio of TC/HDL-C and LDL-C/HDL-C in hypercholesterolemic volunteers. Although bodyweight and BMI of all the participants were not significantly different from the baseline values, volunteers consuming bananas daily showed increasing total calorie intake, which may have been due to the high content of carbohydrate in bananas. The marginal improvement of lipid profile in the hypercholesterolemic subjects could be attributed to the increased amount of dietary fiber and magnesium during the period of banana consumption. Magnesium regulates a specific enzyme called HMG-CoA reductase, a rate limiting step of cholesterol synthesis in the body[35]. In addition, magnesium is also necessary for lecithin cholesterol

acyl transferase (LCAT) activity, which lowers LDL-C and triglyceride levels and raises HDL-C levels[36-38].

The hypoglycemic effect of various parts of banana, including flower[9,10,22], root[21,39] and unripe fruit[20,40] is not uncommon. Ripe banana is restricted in the diet prescribed for diabetic patients owing to its high content of free sugar. In this study, we have demonstrated that ingestion of about 1-2 bananas (250 g) a day can significantly lower fasting blood glucose of normoglycemic volunteers by 10% within 4 weeks of the study period and the levels continued to decrease even after the washout period of 8 weeks. A similar reduction was seen in the group of diabetic patients, although it was not statistically significant, which may due to the fact that all of the diabetic patients were already taking the prescribed hypoglycemic agents at the time and might thus masked the hypoglycemic effect of banana. The hypoglycemic effect of banana is believed to mediate through several active ingredients including dietary fiber[41,42], flavonoids[22] and magnesium[43-45]. Magnesium possesses both direct and indirect roles in regulating the secretion and the signal transduction of insulin thus involving in the process of insulin resistance development[46]. Moreover, flavonoids from banana may increase insulin sensitivity by potentiating the insulin signaling pathway. The docking analysis results indicated that banana flavonoids may potential be activators of insulin receptor tyrosine kinase[47].

Our present study has demonstrated that daily consumption of banana significantly increases serum adiponectin levels in diabetic patients. Adiponectin is an important autocrine/paracrine factor in adipose tissue associated with favorable metabolic effects (i.e. greater insulin sensitivity, reduced visceral adipose mass, reduced plasma triglycerides and increased HDL-cholesterol). Adiponectin influences plasma lipoprotein levels by altering the levels and activity of key enzymes (lipoprotein lipase and hepatic lipase) responsible for the catabolism of triglyceride-rich lipoproteins and HDL[48]. Increased serum adiponectin is thus consistent with the improvement of an individual's lipid profile and reduction of insulin resistance. However, the reason why significant changes were only observed in the diabetic patients, but not in the healthy hypercholesterolemic volunteers may partly be due to the differences of the baseline values and adiponectin metabolism between these two groups of volunteers.

Our study reveals that daily consumption of 1-2 bananas (@ 250grams·day-1) is harmless for diabetic patients, and marginally beneficial to non-diabetic but hypercholesterolemic volunteers.

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