Original Article

# Plasma adiponectin concentrations are associated with dietary glycemic index in Malaysian patients with type 2 diabetes

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Adiponectin, an adipocyte-derived hormone has been implicated in the control of blood glucose and chronic inflammation in type 2 diabetes. However, limited studies have evaluated dietary factors on plasma adiponectin levels, especially among type 2 diabetic patients in Malaysia. The aim of this study was to investigate the influence of dietary glycemic index on plasma adiponectin concentrations in patients with type 2 diabetes. A cross-sectional study was conducted in 305 type 2 diabetic patients aged 19-75 years from the Penang General Hospital, Malaysia. Socio-demographic information was collected using a standard questionnaire while dietary details were determined by using a pre-validated semi-quantitative food frequency questionnaire. Anthropometry measurement included weight, height, BMI and waist circumference. Plasma adiponectin concentrations were measured using a commercial ELISA kit. Data were analyzed using multiple linear regression. After multivariate adjustment, dietary glycemic index was inversely associated with plasma adiponectin concentrations (β =-0.272, 95% CI -0.262, -0.094; $p$<0.001). It was found that in individuals who consumed 1 unit of foods containing high dietary glycemic index that plasma adiponectin level reduced by 0.3 μg/mL. Thirty two percent (31.9%) of the variation in adiponectin concentrations was explained by age, sex, race, smoking status, BMI, waist circumference, HDL-C, triglycerides, magnesium, fiber and dietary glycemic index according to the multiple linear regression model ($R^2$=0.319). These results support the hypothesis that dietary glycemic index influences plasma adiponectin concentrations in patients with type 2 diabetes. Controlled clinical trials are required to confirm our findings and to elucidate the underlying mechanism.

**Key Words: dietary glycemic index, adiponectin, type 2 diabetes, food frequency questionnaire, linear regression**

## INTRODUCTION

Adiponectin, also known as adipoQ, apM1, Acrp30 and GBP28, is a circulating hormone predominantly produced by the adipose tissue.[1] Unlike most cytokines produced from adipose tissue, adiponectin is presented at low plasma level in obese and diabetic animals and humans.[2-4] Many pharmacological, experimental and epidemiological studies suggest that adiponectin may have potent anti-diabetic, anti-inflammatory and anti-atherosclerotic effects.[5-7] Studies in animals show that adiponectin improves glucose utilization through reducing blood free fatty acids, enhancing insulin action, stimulating glucose utilization, increasing hepatic fatty acid oxidation and decreasing hepatic fatty acids synthesis.[5,8,9] High circulating adiponectin levels have been associated with improved blood glucose and lipid control[10,11] and reduced inflammatory markers in patients with diabetes.[12]

In light of the potential beneficial effect of adiponectin on endocrine-related disorders, it is important to identify modifiable lifestyle factors that may affect adiponectin blood concentrations, including diet-related parameters. Recently, numerous studies reported the effects of dietary factors on adiponectin concentrations. Several short-term human trials indicate that a diet high in fiber[13,14] and moderate alcohol intake[15,16] could increase adiponectin concentration, whereas a diet with high glycemic index might adversely affect plasma adiponectin levels in animal models.[17] Other long-term studies indicate that diets low in glycemic load and glycemic index may increase plasma adiponectin concentrations in diabetic patients.[18,19] These are consistent with previous findings that diets with low-glycemic load or index have a favorable

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effect in healthy population.[16] However, no Malaysian study has investigated the effects of dietary glycemic index on adiponectin levels in patients with type 2 diabetes. As unhealthy eating habits is one of the major causes for the complications of type 2 diabetes, the outcome of this study will enable us to understand the relationship of dietary glycemic index on plasma adiponectin concentrations of Malaysian patients with type 2 diabetes. In addition, recent studies reported that adiponectin concentrations were significantly higher in Europeans (adjusted mean 12.94) and Aboriginal people (11.87) than in South Asians (9.35) and the Chinese population (8.52).[20] Using a multiethnic population-based sample, this study will provide information on the variation of adiponectin concentrations among the three main ethnic groups of Malaysia.

## MATERIALS AND METHODS

### *Study population*

We conducted a cross-sectional survey among 305 diabetic patients. The patients were identified through the diabetes clinic at Penang General Hospital, Malaysia. The Penang State is located in the northern region of Peninsular Malaysia, where majority of the population are Chinese. The patients aged between 19 to 75 years comprised of 149 Chinese (48.9%), 91 Indians (29.8%) and 65 Malays (21.3%) that were recruited between May 2011 and December 2011. Inclusion criteria were: 1) Malaysian diagnosed with type 2 diabetes for at least 2 years and above, who were free of fatal coronary heart disease, non-fatal myocardial infarction and stroke; 2) Fasting blood glucose 6 - <10 mmol/L, and HbA1c 6.5% - <8.0%; 3) taking oral hypoglycemic agents only (α-glucosidase inhibitors (AGIs), biguanides, dipeptidyl peptidase-4 (DPP-4) inhibitors and insulin secretagogues). Exclusion criteria were: 1) type 1 diabetes; 2) type 2 diabetic patients who were on exogenous insulin treatment and thiazolidinedione (TZDs) which may affect adiponectin concentrations; 3) those with chronic complications of uncontrolled diabetes with FBS >10 mmol/L and HbA1c > 8.0 % such as nephropathy, neuropathy and cardiovascular disease. The study protocol was approved by the Universiti Sains Malaysia (USM) Human Research Ethics Committee and the Ministry of Health (MOH) Malaysia Clinical Research Centre.

### *Data collection*

The purpose and procedures of the study were explained to the participants and after obtaining written informed consent from all of the subjects, a fasting blood sample was collected and anthropometric and body composition measurements were done. Demographic, medical and lifestyle information were obtained using a standardized questionnaire.

### *Anthropometry assessment*

Anthropometric measurements were taken with the patients in lightweight clothing with shoes removed. Height and weight were measured in duplicate and recorded to the nearest 0.5 cm and 0.1 kg, respectively. Body mass index (BMI) was calculated as weight (in kilograms) divided by the square of height (in meters).[21] Height was measured using a stadiometer (SECA, Germany) and weight was measured using a digital weighing scale (SECA, Germany). After a normal expiration, waist circumference was measured at a level midway between the inferior margin of the lowest rib and the iliac crest by using measuring tape.[22] The World Health Organization cut-off points of waist circumference (men >94 cm; women >80 cm) were used to assess risk of metabolic complications.

### *Biochemistry assessment*

Venous blood samples were taken from the median cubital vein and were collected from all patients after a 12-h overnight fast. Plasma glucose was analyzed by using hexokinase method while the concentration on total-cholesterol, HDL-C and triglycerides were assessed enzymatically with commercially available reagents using auto-analyzer. Concentration on LDL-C was calculated by the Friedewald equation.

### *Assessment of plasma adiponectin concentrations*

Adiponectin concentrations were measured using an enzyme-linked immunosorbent assay (ELISA) kit (Symansis Limited, New Zealand) in duplicate. The sensitivity of the assay was 7.8 ng/mL. The intraassay and interassay coefficients of variation were less than 5% and 9% respectively for adiponectin.

### *Dietary assessment*

Detailed dietary information was obtained using a pre-validated semi-quantitative food-frequency questionnaire (FFQ). The FFQ consisted of 126 food items that were listed in 15 food groups that was evaluated in the Malaysian Adult Nutrition Survey (MANS) involving 6,742 subjects, comprising of 3,274 men and 3,468 women and conducted nation-wide between October 2002 and December 2003, to assess the habitual food intake.[23] However, the validity and reliability of MANS' FFQ to measure GI/GL was not performed in that study. The GI values for food consumed by the population were listed in a separate sheet attached along with the pre-validated FFQ. The FFQ was administered by interview on a one-to-one basis where patients were asked on the frequency of intake of each food item either 'per day, per week, per month, per year or never' on the food items listed. Patients were also requested to respond to the number of servings consumed each time they ate the food. Nutrient intakes were computed by multiplying the frequency response with the nutrient content of the specified portion sizes. Values for nutrients were derived from the Malaysian Food Database with Nutritionist Pro™ software product. The GI values of some foods were obtained from the research studies done in Malaysia.[24-26] However the GI values for most of the carbohydrate foods were referred from the International Table of Glycemic Index and Glycemic Load Values.[27] All food consumed by the population was available and therefore no replacement or estimation of the glycemic index (GI) value of the foods was conducted. Glucose was used as the reference (GI for glucose=100). When more than one GI value was available, the mean GI values were used. In this study, we calculated the average meal GI of each patient by multiply-

ing the available carbohydrate content of each food by the number of servings consumed per day, then multiplied this value by the GI value of each food to get the glycemic load (GL). The overall dietary GI was then calculated by dividing the glycemic load by the total carbohydrate consumed.[18]

***Statistical analysis***

Continuous variables are presented as mean values (standard deviations); and categorical variables, as absolute frequencies. The normality of distribution was tested by using histogram. Linear regression model was used to evaluate associations between dietary glycemic index and plasma adiponectin concentrations. We further adjusted for the potential confounding variables: age, sex, race, smoking status, HDL-C, triglycerides, BMI, waist circumference, fiber and magnesium intake. Multiple linear regression analysis was applied to evaluate the explanatory ability of the principal components extracted in relation to adiponectin levels after adjusting for potential confounders. The results from the regression models are presented as standardized β coefficients. All statistical evaluations were computed using the Statistical Package for the Social Sciences (SPSS) Statistics version 19.0 (SPSS, Inc., Chicago, IL, USA). Two sided $p<0.05$ was considered statistically significant at 95% confidence interval.

**RESULTS**

The characteristics of the study subjects are shown in Table 1. The populations were patients diagnosed with

**Table 1.** Characteristics of study subjects ($n$ = 305)

| Characteristics | Total† ($n$=305) | Men† ($n$=156) | Women† ($n$=149) | $p$-value* |
|---|---|---|---|---|
| Age (year) | 59.6 (7.4) | 59.0 (7.7) | 60.1 (7.0) | 0.184 |
| Race | | | | |
| Malays | 65 (21.3) | 32 (20.5%) | 33 (22.1%) | |
| Chinese | 149 (48.9) | 78 (50.0%) | 71 (47.7%) | |
| Indians | 91 (29.8) | 46 (29.5%) | 45 (30.2%) | |
| Anthropometric parameters | | | | |
| Weight (kg) | 68.7 (14.5) | 73.5 (14.7) | 63.6 (12.3) | <0.001 |
| Height (cm) | 161 (8.1) | 166 (6.3) | 155 (5.7) | <0.001 |
| BMI (kg/m$^2$) | 26.5 (5.0) | 26.6 (5.0) | 26.3 (4.9) | 0.609 |
| WC (cm) | 92.6 (11.4) | 95.5 (11.4) | 89.3 (10.3) | <0.001 |
| HC (cm) | 99.6 (9.1) | 101 (8.6) | 98.3 (9.3) | 0.025 |
| WHR | 1.1 (0.1) | 0.94 (0.06) | 0.91 (0.06) | <0.001 |
| Smoking status | | | | |
| Current smokers | 31 (10.1) | 24 (15.4%) | 7 (4.7%) | |
| Never smoked | 256 (83.7) | 114 (73.1%) | 142 (95.3%) | |
| Past smokers | 18 (5.9) | 18 (11.5%) | 0 (0%) | |
| Dietary intake (mean) | | | | |
| Energy (kcal/d) | 1632 (471) | 1691 (484.4) | 1571 (449) | 0.026 |
| Carbohydrate (g/d) | 346 (97.8) | 361 (101.7) | 331 (91.5) | 0.009 |
| Protein (g/d) | 41.7 (15.4) | 43.2 (15.8) | 40.2 (15.0) | 0.083 |
| Fat (g/d) | 9.3 (10.2) | 8.8 (10.2) | 9.8 (10.3) | 0.400 |
| Fiber (g/d) | 4.9 (6.6) | 4.8 (6.8) | 4.9 (6.5) | 0.934 |
| Magnesium (mg/d) | 50.2 (87.7) | 49.4 (89.0) | 51.1 (86.6) | 0.864 |
| Glycemic load (unit) | 245 (109) | 250 (90.7) | 240 (126) | 0.420 |
| Glycemic index (unit) | 69.7 (7.7) | 70.6 (7.5) | 68.8 (7.9) | 0.046 |
| Glycemic index quartiles (unit) | | | | |
| Q1 | 59.0 (6.4) | 59.4 (6.1) | 58.7 (6.7) | |
| Q2 | 69.7 (1.0) | 69.7 (1.1) | 69.7 (1.0) | |
| Q3 | 72.4 (0.9) | 72.3 (0.8) | 72.5 (0.9) | |
| Q4 | 77.7 (3.4) | 78.2 (3.9) | 77.1 (2.4) | |
| Plasma adiponectin (μg/mL) | 10.3 (5.1) | 9.3 (4.8) | 11.3 (5.1) | 0.001 |
| Adiponectin quartiles (μg/mL) | | | | |
| Q1 | 4.7 (1.1) | 4.7 (1.1) | 4.8 (1.1) | |
| Q2 | 7.8 (1.0) | 7.7 (1.0) | 7.9 (1.0) | |
| Q3 | 11.3 (1.2) | 11.2 (1.0) | 11.4 (1.3) | |
| Q4 | 17.2 (3.6) | 17.3 (3.8) | 17.2 (3.6) | |
| Glucose studies‡ | | | | |
| FBS (mmol/L) | 7.3 (2.7) | 7.4 (2.7) | 7.2 (2.7) | 0.359 |
| HbA1c (%) | 7.9 (1.4) | 8.0 (1.4) | 7.8 (1.4) | 0.420 |
| Lipid studies‡ | | | | |
| Total cholesterol (mmol/L) | 4.8 (0.9) | 4.7 (0.8) | 4.9 (0.9) | 0.004 |
| TGs (mmol/L) | 1.7 (0.9) | 1.8 (0.9) | 1.7 (0.9) | 0.328 |
| HDL-C (mmol/L) | 1.2 (0.5) | 1.1 (0.2) | 1.2 (0.6) | 0.006 |
| LDL-C (mmol/L) | 2.9 (0.8) | 2.8 (0.7) | 3.0 (0.8) | 0.062 |

† Mean (SD) or n (%)

‡ indicates fasting values

$n$: total number of subjects, SD: standard deviation, BMI: body mass index, WC: waist circumference, WHR: waist-to-hip ratio, FBS: fasting blood sugar, HbA1c: glycated hemoglobin.

* Independent t-test was applied to determine the difference between men and women.

**Table 2.** Spearman correlation coefficients between dietary factors, biomarkers and adiponectin concentrations (n=305)

| Characteristics | *r* | *p*-value |
|---|---|---|
| Energy intake | - 0.16 | 0.004* |
| Carbohydrate intake | - 0.20 | <0.001** |
| Protein intake | - 0.37 | 0.520 |
| Fat intake | 0.14 | 0.016* |
| Fiber intake | 0.23 | <0.001** |
| Magnesium intake | 0.13 | 0.020* |
| Glycemic index | - 0.35 | <0.001** |
| Glycemic load | - 0.36 | <0.001** |
| FBS (mmol/L) | - 0.07 | 0.182 |
| HbA1c (%) | - 0.24 | <0.001** |
| Total cholesterol (mmol/L) | 0.03 | 0.581 |
| TGs (mmol/L) | - 0.38 | <0.001** |
| HDL-C (mmol/L) | 0.39 | <0.001** |
| LDL-C (mmol/L) | 0.04 | 0.526 |

* Significant at the 0.05 level
** Significant at the 0.001 level

type 2 diabetes at least 2 years with moderate glycemic control, in which the mean HbA1c and FBS were reported at 7.9% (1.4) and 7.3 mmol/L (2.7) respectively. The mean plasma adiponectin level of the overall population was 10.3 μmol/L; and we noticed a significant difference in mean adiponectin concentrations between men and women ($p$<0.001), reported at 9.30 μg/mL (4.85) and 11.3 μg/mL (5.11), respectively. The mean dietary glycemic index was 69.7 unit (Table 1). Both plasma adiponectin and glycemic index were normally distributed in this population.

In correlation analysis (Table 2), we found a small but significant correlation for glycemic index (r= -0.35, $p$<0.001), glycemic load (r= -0.36, $p$<0.001), energy intake (r= -0.16, $p$<0.05), carbohydrate intake (r= -0.20, $p$<0.001), fat intake (r=0.14, $p$<0.05), fiber intake (r=0.23, $p$<0.001), and magnesium intake (r=0.13, $p$<0.05) with adiponectin concentrations. Figure 1 illustrates inverse linear relationships between plasma adiponectin and glycemic index generated by scatter plots and regression lines. To evaluate potential associations between adiponectin levels and glycemic index, after controlling for potential confounders (age, sex, race, smoking status, HDL-C, triglycerides, BMI, waist circumference, fiber and magnesium intake), multiple regression analysis (MLR) was performed. The inverse association between dietary glycemic index and plasma adiponectin levels detected in bivariate analysis remained statistically significant after adjusting for the aforementioned confounders (standardized β coefficient= -0.272, $p$<0.001). Results are presented in Table 3. It was found that in individuals who consumed 1 unit of foods containing high dietary glycemic index that the plasma adiponectin level reduced by 0.3 μg/mL. Thirty two percent (31.9 %) of the variation in adiponectin concentrations is explained by age, sex, race, smoking status, HDL-C, triglycerides, BMI, waist circumference, glycemic index, fiber and magnesium intake according to the MLR model ($R^2$=0. 319, $p$<0.001).

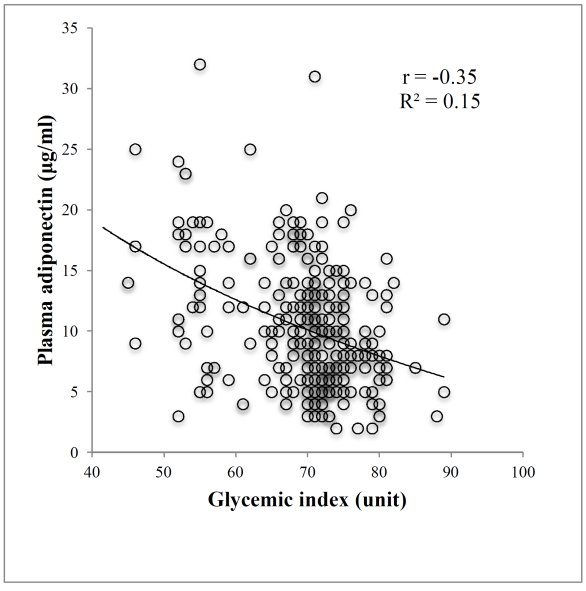


**Figure 1.** Inverse linear relationships between plasma adiponectin and dietary glycemic index

## DISCUSSION

In this cross-sectional study we found that there was an association between dietary glycemic index and plasma adiponectin concentrations in diabetic patients. A high glycemic index diet was associated with lower level of plasma adiponectin. The observed associations remained significant after adjusting for potential confounders including age, sex, race, BMI, waist circumference, HDL-C, triglycerides and smoking status. Although the strength of these associations was modest, our data suggest that dietary glycemic index is related to plasma adiponectin concentrations and support the hypothesis that dietary factors may modulate adiponectin concentrations; a potential mediator related to better glycemic control.[10-12]

Our findings of inverse associations of dietary glycemic index with plasma adiponectin are consistent with prior studies.[16,18,19] Results from Lu Qi *et al.* and Pischon *et al.* indicate that diets with low glycemic index or glycemic load have a favorable effect in both healthy and diabetic individuals.[16,18,19] Pischon *et al.* found a carbohydrate-rich diet with a high glycemic load is associated with lower adiponectin concentrations in 532 men with no history of cardiovascular disease. This finding is in line with an earlier study conducted by Lu Qi *et al.*, who examined the predictive roles of glycemic load or glycemic index among type 2 diabetic men in the Health Professional Follow-up Study (HPFS) cohort.[18] They found that dietary glycemic index was inversely associated with adiponectin concentrations among men with type 2 diabetes. These results are highly consistent with a more recent findings in women with type 2 diabetes, which showed that glycemic load or glycemic index may be important determinants for blood concentrations of adiponectin in patients with type 2 diabetes in the United States.[18,19]

The precise mechanism underlying the protective effects of glycemic-related dietary factors is not fully understood. However, the glycemic index was suggested to affect plasma adiponectin through modulation of blood glucose, because a diet high in glycemic index may increase blood glucose.[28,29] Blood glucose has been inversely associated with the expression of adiponectin in adipose tissue.[30] Hence, a glucose-enriched diet markedly

**Table 3.** Results of the multiple regression model evaluating association between dietary glycemic index and other confounding factors against plasma adiponectin concentrations ($n$ = 305)

| Variables | Multiple Linear Regression | |
|---|---|---|
| | $p$-value | β (95% CI) |
| Glycemic index | <0.001** | - 0.272 (-0.262, -0.094) |
| Fiber | <0.001** | 0.415 (0.155, 0.478) |
| Magnesium | <0.001** | - 0.408 (-0.035, -0.012) |
| Age | 0.002* | 0.162 (0.041, 0.181) |
| Sex | 0.087 | 0.095 (-0.139, 2.054) |
| Race | 0.294 | - 0.052 (-1.060, 0.322) |
| BMI | 0.267 | 0.091 (-0.071, 0.257) |
| WC | 0.440 | - 0.066 (-0.104, 0.045) |
| Smoking status | 0.313 | 0.052 (-0.675, -2.100) |
| TG | <0.001** | - 0.238 (-1.883, -0.777) |
| HDL-C | 0.072 | 0.092 (-0.088, 2.083) |

Dependent variable: plasma adiponectin concentrations; β is standardized coefficients. Enter multiple linear regression method applied. Model assumptions are fulfilled. There were no interactions amongst independent variables. No multicollinearity detected.
Coefficient of determination ($R^2$) = 0.319. Adjusted coefficient of determination (adjusted $R^2$) = 0.294. Standard error of the estimate = 4.26.
* Significant at the 0.05 level
** Significant at the 0.001 level

reduces adiponectin expression in adipose tissue.[31] In addition, our data also demonstrates that the adiponectin-modulation effect of dietary glycemic index was independent of fiber and magnesium intakes. Because fiber and magnesium share similar food sources such as whole grains, it is difficult to identify the independent effects of these nutrients on adiponectin concentrations. These findings are similar with those previously reported for adiponectin levels and cereal fiber, whole grain cereals or glycemic index and glycemic load.[15,18,19]

Our study has both strengths and limitations. The strengths of the study include the use of state-of-art methodology to measure adiponectin, and statistical control of potential confounders. Dietary records were measured using the FFQ, which is not only a useful method to categorize intake of individuals based on their food consumption,[32,33] but also to evaluate the habitual energy and nutrient intake to determine the relationship between changes in food habits and chronic diseases.[34] The performance of the FFQ in assessing the individual foods from which the glycemic index and load were derived was previously documented.[35] The validity of using MANS' FFQ to determine the calculated dietary GI/GL was not performed in this study; however, potential measurement error from using FFQ does not seem to bias our results because reporting error is not likely associated with the biological measurements. When compared with other dietary assessment tools, FFQ is often regarded as a "gold standard" or "reference method" for collecting dietary data.[36] Drug treatment for diabetes can alter adipokine concentration, and thus we excluded these patients from our analysis. Our sample size of 305 patients has provided enough power to detect statistical significant in primary outcome measures in our study based on the Power and Sample Size Calculation Program with a significance level (α) of 0.05 for 2 sided-tests and 80% power. Among the limitations of this study is its cross-sectional design, in which one cannot establish a cause-effect relationship and/or elucidate underlying mechanisms. Although we established a temporal relation between diet and adiponectin, our study was associative and thus more apt for hypothesis generating than for drawing inferences about causal pathways. Furthermore, potential epidemiological issues especially the bias induced by prevalent diabetes disease may confound the association between dietary factors and biomarkers; however, the FFQ was obtained before the blood samples were drawn; thus, reverse causality seems unlikely. Nevertheless, the implications of the relationship are not clear, and thus controlled trials are needed to determine if the relationships are causal. Although we carefully adjusted the known and potential confounders, residual confounding remains a possibility. We used surrogate measures of adiposity (e.g., body mass index and waist circumference), which are merely proxy indicators of visceral fat, a stronger predictor of insulin resistance; therefore, we cannot exclude the possibility of residual confounding. It is currently unknown whether the long-term storage of blood samples affects plasma adiponectin concentrations; however, adiponectin concentrations were not significantly related to storage time and storage time is unlikely to be related to dietary factors.[16] In this study we found that the adiponectin concentrations among Malaysians are lower when compared with Caucasians. This finding is similar to a previous study by Andrew *et al.*[20]; thus suggesting adiponectin gene variability and gene polymorphism in this population. Our findings reflect different pathogenic pathways including the type, amount and distribution of adipose tissue accumulation as well as dietary and genetic influences that may exist among Malaysians. The metabolic disturbances behind these differences is not known but may be related to differences in adipocyte properties. On the other hand there were no significant differences in the adiponectin concentrations within the three main ethnic groups of Malaysia; thus further studies that comprise of a bigger sample size may be warranted to determine the differences in adiponectin concentrations among the different races of the Malaysian population.

To the best of our knowledge, this investigation is the first to compare adiponectin levels with dietary glycemic index in a multi-ethnic population of Malaysia. As unhealthy eating habits, especially prolong intake of high glycemic index diet is one of the major contributing factors for the rising prevalence of type 2 diabetes mellitus in Malaysia, the interesting results of this study will be useful for educating the Malaysian diabetic population regarding the significance of the GI concept and its application in planning healthy meals.

In conclusion, Malaysians have an unfavorable adipokine profile compared with Caucasians and display a greater increase in insulin resistance with decreasing levels of adiponectin concentrations. In addition, it was found that there was a significant inverse association between dietary glycemic index and plasma adiponectin concentrations in Malaysian patients with type 2 diabetes. Even though the strength of these associations was modest, our study supports the hypothesis that dietary factors

may regulate plasma adiponectin concentrations. Further studies, especially controlled clinical trials and experimental studies, are warranted to confirm our findings as well as to elucidate underlying mechanism.

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**AUTHOR DISCLOSURES**

None of the authors had any personal or financial conflicts of interest.

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Original Article

# Plasma adiponectin concentrations are associated with dietary glycemic index in Malaysian patients with type 2 diabetes

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# 馬來西亞第 2 型糖尿病患之膳食升糖指數與血漿脂聯素濃度之相關性

脂肪細胞衍生的激素-脂聯素，被認為會調控第 2 型糖尿病患之血糖濃度及慢性發炎反應。然而，僅有限的研究評估飲食因子對血漿中脂聯素濃度的影響，更缺少針對馬來西亞第 2 型糖尿病患者的研究。本研究的目的在於評估膳食升糖指數對於第 2 型糖尿病患血漿脂聯素的影響。此橫斷面研究，受試者為 305 位，於馬來西亞檳城總醫院就醫，年齡介於 19-75 歲的第 2 型糖尿病患。使用標準化問卷蒐集社會人口學資訊，膳食評估則使用具效度的半定量食物頻率問卷。體位測量包括體重、身高、身體質量指數及腰圍。血漿脂聯素濃度是使用市售的酵素結合免疫吸附(ELISA)試劑組檢測。統計方法為多元線性迴歸。校正多變項後，膳食升糖指數與血漿脂聯素濃度呈顯著負相關(β= -0.272；95%CI： -0.262, -0.094；$p$<0.001)。個體每攝取一單位高升糖指數之食品，血漿脂聯素下降 0.3 μg/mL。依據多元線性迴歸模式($R^2$=0.319)，血漿脂聯素濃度 31.9%的變異量可由年齡、性別、種族、抽菸、身體質量指數、腰圍、高密度脂蛋白膽固醇、三酸甘油酯、鎂、膳食纖維及升糖指數所解釋。這些結果證實，膳食升糖指數會影響第 2 型糖尿病患者血漿中脂聯素的濃度。對於此結果的驗證以及可能機制的闡明，仍需要臨床試驗進一步釐清。

**關鍵字：膳食升糖指數、脂聯素、第 2 型糖尿病、食物頻率問卷、線性迴歸**