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Original article

The relationship between plasma total antioxidant capacity and dietary antioxidant status in adults with type 2 diabetes

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Summary. *Background and aim:* There is limited information available on the association of plasma total antioxidant capacity (TAC) with dietary TAC in DM. The aim of this study was to evaluate the plasma TAC and its association with dietary antioxidant status in adults with Type 2 diabetes. *Methods:* Thirty outpatients diagnosed with type 2 DM (diabetics, n = 29) and 15 healthy subjects (control, n = 15) aged 40-70 with BMI≤ 30 kg/m^2 were recruited to the study. Energy and nutrients intake, anthropometric measurements, dietary and plasma TAC, and some biochemical parameters were evaluated. The calculation of dietary TAC was based on previously published databases in which modified version of the FRAP. *Results:* Serum triglyceride, uric acid, and HbA1c levels in diabetics were higher than controls. A negative and statistically significant correlation was found between plasma TAC and HbA1c for diabetics. A negative and statistically significant correlation was observed between dietary TAC, HbA1c and fasting plasma glucose in diabetics. A positive and statistically significant correlation was found between plasma TAC and dietary intake of niacin in diabetics. No remarkable differences were found between dietary and plasma TAC in either group.*Conclusion:* This study provides evidence that dietary TAC is not an important modulator of antioxidant status in diabetic subjects. But, it showed that the increase in niacin and antioxidant taken with foods can be effective in controlling HbA1c and fasting plasma glucose.

Key words: antioxidant, HbA1c, metabolic control, TAC, type 2 diabetes mellitus

Introduction

According to the World Health Organization (WHO), approximately 150 million people have diabetes mellitus (DM) worldwide, and this number may double by the year 2025 due to population growth, age, unhealthy diet, obesity and sedentary lifestyle (1). There is concern about an emerging diabetes epidemic in Turkey. A cross-sectional survey, "The Turkish Epidemiology Survey of Diabetes, Hypertension, Obesity and Endocrine Disease" (TURDEP-II) conducted in 2010, included 26.499 randomly sampled adults aged ≥ 20 years showed that the prevalence of diabetes was 16.5% translating to 6.5 million adults with diabetes in Turkey (2).

The role of oxidative stress and inflammation in several chronic diseases is receiving increasing attention due to identified links with chronic diseases such as atherosclerosis, obesity, or type 2 DM (3). One of the pathogenic mechanisms that can explain this increased risk in DM is the imbalance between pro-oxidants and antioxidants, which results in oxida-

tive stress (OS) (4). Hyperglycemia results in glucose auto-oxidation, no enzymatic glycation and monocyte dysfunction, which lead to increased production of free radicals (5). This is further aggravated by the decreased levels of antioxidants (6) and leads to oxidative damage, illustrated by the high levels of lipid and DNA peroxidation products found in diabetic patients (7).

Food intake has been related to oxidative stress modulation (8, 9), being described as energy restriction that might decrease the levels of oxidative stress mediators (10). Antioxidant intake has been suggested to protect against oxidative damage (11).

Dietary antioxidants have been hypothesized to have a protective effect against the development of DM by inhibiting peroxidation chain reactions (12). Fruit and vegetable consumption has long been reported to be associated with lower incidence and mortality rates of several chronic diseases (13, 14). One hypothesis of the protective effects is that all types of antioxidant compounds, including vitamin C, vitamin E, carotenoids, and phytochemicals such as flavonoids and proanthocyanidins could protect cells from free radical–induced oxidative damage (15).

An increasing number of studies have reported that an increase in plasma TAC is associated with intake of fruits and vegetables which are rich in antioxidants (16, 17). Given that the concentration of single antioxidants may not reflect the total antioxidant power of food, the concept of TAC was introduced (18). Dietary TAC has been shown to be a good indicator of diet antioxidant status (19, 20).

Nevertheless, there is limited information available on the association of plasma TAC with dietary TAC in DM. It has been hypothesized that dietary TAC is a good indicator of diet quality with respect to reflecting the antioxidant capacity of a diet, as well as a predictor of plasma antioxidant status. Thus, the aim of this study was to evaluate the plasma TAC and association with dietary TAC in adults with type 2 DM.

Methods

Study population and design

This descriptive study was performed at Internal Medicine Clinic of Kayseri Acıbadem Hospital, a tertiary referral Centre in Turkey, the between 2013 and 2014.

Thirty outpatients diagnosed with type 2 DM (diabetic group, n = 30) and 15 healthy subjects (control group, n = 15) aged 40-70 with BMI ≤ 30 kg/m^2, and who did not report any physical activities were recruited to this study (5). All controls were in good health as determined by a medical history questionnaire, physical examination and normal results of clinical laboratory tests. All diabetics were on different medications, mainly metformin, and they were not on special diets. Of 45 subjects, 1 diabetic patients had missing information thus data of the 44 participants were used.

Demographic characteristics like age, gender, marital status (married, divorced, and widowed), financial status (average annual income during the past three years), and occupational status as well as education level were obtained by questionnaire during a face to face interview. Anthropometric measurements, dietary and plasma TAC, and some biochemical parameters were evaluated. The subjects in both groups were matched according to socio-demographic features, sex and age.

Exclusion criteria were derived as follows: 1) individuals who were administered supplements during the 6 months prior to this study 2) patients with evidence of any diabetic complication and history of chronic conditions or diseases, including cardiovascular disease, certain cancers, and chronic obstructive pulmonary disease, 3) alcohol consumption and smoking 4) those who were on insulin treatment.

In accordance with the Declaration of Helsinki, after a clear explanation of the study protocol, all subjects gave written informed consent to participate in this study, which was approved by the ethics committee of Faculty of Medicine in Erciyes University (Kayseri, Turkey) (reference 2012/571).

Dietary assessment

All participants recorded their 24-hour food consumption for three consecutive days including at least one weekend day. Nutrient Database (BeBiS, Ebispro for Windows, Germany; Turkish Version/BeBiS 7) was used to determine energy and nutrient intake (21). Volumes and portion sizes were estimated with 2-dimensional food models and with a portion size picture

booklet including 120 photographs of food, each with 3–5 different portion sizes (22).

The calculation of total dietary TAC was based on previously published databases in which a modified version of the FRAP assay that allowed quantification of most water- and fat-soluble antioxidants (23) was considered for each food. This Antioxidant Food Database was used because the samples were procured from local stores and markets in Scandinavia, USA and Europe and from the African, Asian and South American continents. We calculated total dietary TAC of 3100 foods, and recorded them in the Nutrient Database (BeBiS, Ebispro for Windows, Germany; Turkish Version/BeBiS 7).

Anthropometric measurements

Anthropometric measurements were determined according to WHO criteria (24). Body weight and height were measured and BMI was calculated (BMI=body weight (kg)/height (m^2)). All subjects were weighed while wearing light clothing and without shoes, using a calibrated digital flat scale (Seca-803, USA). Standing height was measured without shoes to the nearest 0.5 cm using a measuring tape.

Assessment of biochemical parameters

Biochemical analysis

Following an 8 hour overnight fast, blood samples were collected between 08.30 and 10.30 am. Routine blood tests including serum fasting plasma glucose (FPG), triglyceride (TG), total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), glycated hemoglobin (HbA1c), aspartate aminotransferase (AST), alanine aminotransferase (ALT), and uric acid were analyzed in the Acıbadem Hospital laboratory.

Serum FPG, TG, TC, and HDL-C were determined with kits using Architect c16000 auto analyzer (Abbott Diagnostics, USA). The reported serum low-density lipoprotein cholesterol (LDL-C) data were calculated using the Friedewald equation as described elsewhere (25). HbA1c was measured by boronate affinity assay using the NycoCard HbA1c Kit (Axisshield, Oslo, Norway). Serum AST and ALT were determined using a commercial kit, Latrozyme TA-LQ (Dai Iatron Co, Tokyo, Japan). Serum uric acid was assessed by using colorimeter on a Konelab Autoanalyser (Thermo Scientific). The serum was separated by centrifugation and stored at 80°C then shipped on dry ice to the laboratory where TAC analysis was performed. TAC of plasma was determined based on the Trolox equivalent antioxidant capacity assay using a colorimetric commercial kit (Cayman Chemical Corporation, Ann Arbor, MI, USA).

Statistical analysis

Data were analyzed by SPSS, version 21.0 (Inc., Chicago, IL, USA). Normal distribution of data was determined with Shapiro-Wilk test. Chi-square analysis was used to compare the difference of variables between groups and Mann Whitney U test was used for non-normally distributed data by showing median and 25–75 percentiles. Spearman correlation was performed to determine the association of dietary and plasma TAC as well as energy and nutrient intake and biochemical parameters. $p<0.05$ was set as statistically significant.

Results

Mean ages of subjects were 55.86±5.69 years for diabetics, and 51.73±7.27 years for controls. 31.0% of diabetics (n=9) and 33.3% of controls (n=5) were male, while 69.0% of diabetics (n=20) and 66.7% of controls (n=10) were female. Socio-economic status, educational level, and occupation were similar in both groups. Median levels of body weight and height were higher in diabetics than in controls ($p<0.05$) while median level of BMI was not significantly different between groups ($p>0.05$) (Table 1). Dietary intakes of energy and nutrients were not significantly different between groups ($p>0.05$) (Table 2). Although statistically insignificant, dietary TAC in diabetics (3.09±1.81 mmol/day) was higher than in controls (2.97±1.76 mmol/day) (Table 2). Serum triglyceride ($p<0.05$), uric acid ($p<0.05$), and HbA1c ($p<0.01$) levels in diabetics were higher than controls whereas LDL-C were lower ($p>0.05$) (Table 3). Although statistically insignificant, plasma TAC level of controls (0.94±0.34 mmol) were lower than diabetics (1.00±0.42 mmol) ($p>0.05$) (Table 1). Negative and statistically significant correla-

Table 1. Anthropometric measurements and biochemical parameters of diabetics and controls

Parameters	Diabetics (n=29)	Controls (n=15)	p
	X±SD Median-25%-75%	X±SD Median-25%-75%	
Age (year)	55.86±5.69 57.00 (54.00- 60.00)	51.73±7.27 51.00 (45.00-58.00)	0.068
Weight (kg)	74.66±9.59 75.30 (68.90-81.00)	67.08±6.34 66.75 (61.10-69.91)	0.016
Height (cm)	163.10±8.17 164.00 (158.00-168.00)	156.17±6.28 156.00 (150.00-160.00)	0.012
BMI (kg/m²)	27.95±1.88 28.60 (27.50-29.45)	27.64±2.14 27.90 (26.80-29.51)	0.618
FPG (mg/dl)	112.19±64.09 97.00 (65.25-129.75)	81.75±54.58 64.00 (50.00-105.75)	0.073
TG (mg/dl)	205.07±112.25 196.00 (118.00-285.00)	138.53±83.23 118.00 (86.00-159.00)	0.032
TC (mg/dl)	219.79±37.10 224.00 (187.50-249.00)	238.93±38.62 234.00 (204.00-258.00)	0.117
HDL (mg/dl)	46.07±10.12 45.00 (39.50-53.50)	57.53±30.35 49.00 (42.00-58.00)	0.154
LDL (mg/dl)	134.31±35.47 129.00 (113.50-159.50)	156.93±30.75 150.00 (131.00-177.00)	0.042
AST (U/L)	17.93±8.88 17.00 (13.50-20.50)	18.77±7.19 16.00 (13.00-23.50)	0.795
ALT (U/L)	16.45±10.11 13.00 (10.50-17.00)	22.86±21.44 16.50 (10.25-29.50)	0.377
Uric acid (mg/dl)	5.29±1.69 5.20 (3.85-6.20)	4.13±0.85 3.90 (3.65-4.65)	0.018
HbA1c (%)	7.51±1.82 6.90 (6.30-9.00)	5.59±0.28 5.50 (5.40-5.80)	0.002
Plasma TAC (mmol)	1.00±0.42 1.00 (0.71-1.32)	0.94±0.34 0.96 (0.61-1.15)	0.626

tion was found between plasma TAC and HbA1c (r=-0.376, p=0.053) for diabetics, and LDL-C (r=-0.647, p=0.012) for controls (Table 3). When all participants were considered, a negative association was observed between plasma TAC and dietary intake of vitamin E in controls (r=-0.537, p=0.048). Also, a positive and statistically significant correlation was found between plasma TAC and dietary intake of niacin in diabetics (r=0.487, p=0.007). No remarkable differences were found between dietary TAC and plasma TAC in the groups (p>0.05) (Table 3). Negative and statistically significant correlation was observed between dietary TAC, HbA1c and fasting plasma glucose in diabetics (r=-0.531, p=0.004) (Table 4).

Discussion

To our knowledge, this is the first study designed to evaluate the dietary antioxidant intake and its association with plasma antioxidant status in adults with type 2 diabetes in Turkey.

Table 2. Energy and nutrients intakes of diabetics and controls

Parameters	Diabetics (n=29)	Controls (n=15)	p
	X±SD Median-25%-75%	X±SD Median-25%-75%	
Energy (kcal/d)	1660.56±588.73 1432.90 (1156.55-2095.30)	1696.59±485.25 1554.60 (1276.90-2173.00)	0.840
CHO (g/d)	193.76±73.68 181.70 (136.10-247.70)	190.89±48.97 179.30 (152.10-223.90)	0.893
CHO (%)	48.00±7.10 49.00 (42.00-53.00)	46.73±5.05 47.00 (43.00-49.00)	0.543
Protein (g)	57.12±19.74 53.00 (41.95-72.90)	65.17±22.56 65.20 (42.30-88.10)	0.229
Protein (%)	14.38±3.29 14.00 (12.00-16.00)	15.87±4.31 15.00 (12.00-18.00)	0.313
Fat (g/d)	70.85±29.20 66.40 (46.20-88.50)	72.58±26.79 66.50 (47.10-101.10)	0.849
Fat (%)	37.69±7.14 37.00 (33.00-44.00)	37.40±4.64 38.00 (35.00-41.00)	0.738
Fiber (g/d)	20.04±7.57 21.50 (12.85-26.40)	21.21±8.35 20.30 (14.40-25.50)	0.642
TC (mg)	237.04±125.80 237.30 (141.30-334.50)	274.71±99.96 234.80 (198.00-337.40)	0.321
Vitamin A (mcg)	1328.22±1103.31 1190.90 (606.65-1574.60)	1198.04±571.50 988.80 (861.20-1622.00)	0.853
Vitamin E (mg)	14.46±6.17 14.60 (9.80-18.60)	14.85±6.55 13.90 (9.00-19.60)	0.846
Thiamine (mg)	0.83±0.30 0.80 (0.65-1.05)	0.89±0.31 1.00 (0.60-1.10)	0.498
Riboflavin (mg)	1.37±0.55 1.30 (0.90-1.80)	1.46±0.59 1.60 (0.90-1.80)	0.601
Niacin (mg)	18.18±6.71 17.70 (13.25-21.45)	20.51±10.75 18.00 (10.10-28.50)	0.453
Vitamin C (mg)	135.68±87.23 129.60 (74.95-170.40)	154.89±84.31 140.80 (116.10-134.80)	0.488
Ca (mg)	652.99±273.81 592.80 (443.25-794.10)	675.49±343.58 690.40 (327.10-932.60)	0.814
Mg (mg)	248.57±107.16 249.90 (168.20-317.65)	241.85±97.47 266.60 (162.50-305.50)	0.840
Fe (mg)	29.38±99.77 10.80 (7.90-15.10)	11.10±3.72 10.30 (8.60-14.30)	0.931
Zn (mg)	8.05±2.94 7.40 (5.55-10.30)	9.63±3.84 9.40 (5.90-12.40)	0.193
Dietary TAC (mmol/day)	3.09±1.81 3.00 (1.45-4.80)	2.97±1.76 2.30 (1.60-4.00)	0.931

Table 3. Relationship between plasma and dietary TAC as well as dietary TAC and nutrient intakes and biochemical parameters in diabetics and controls

	Plasma TAC (mmol/L)			
	Diabetics (n=29)		**Controls (n=15)**	
	r	p	r	p
Dietary TAC (mmol/day)	0.293	0.123	0.482	0.081
Energy (Kkal)	0.239	0.214	-0.301	0.296
CHO (g)	0.254	0.184	0.204	0.485
CHO (%)	-0.053	0.786	0.520	0.056
Protein (g)	0.179	0.352	-0.314	0.274
Protein (%)	-0.067	0.730	-0.307	0.285
Fat (g)	0.248	0.194	-0.248	0.392
Fat (%)	0.007	0.972	-0.262	0.365
Fiber (g)	-0.101	0.601	0.156	0.594
TC (mg/dl)	0.123	0.524	0.321	0.263
Vitamin A (mcg)	0.017	0.929	0.253	0.383
Vitamin E (mg)	0.034	0.861	-0.537	0.048
Thiamine (mg)	0.163	0.398	0.115	0.695
Riboflavin (mg)	0.168	0.385	0.188	0.521
Niacin (mg)	0.487	0.007	-0.336	0.240
Vitamin C (mg)	0.193	0.316	-0.295	0.306
Ca (mg)	0.132	0.494	-0.002	0.994
Mg (mg)	0.116	0.550	0.090	0.759
Fe (mg)	0.009	0.962	0.235	0.418
Zn (mg)	0.155	0.423	0.279	0.334
FPG (mg/dl)	-0.158	0.440	0.336	0.312
AST (U/L)	0.002	0.990	-0.232	0.468
ALT (U/L)	-0.137	0.479	0.302	0.316
Uric Acid (mg/dl)	0.141	0.465	0.319	0.313
HbA1c (%)	-0.376	0.053	0.220	0.601
TG (mg/dl)	0.119	0.540	-0.029	0.923
TC (mg/dl)	-0.177	0.359	-0.427	0.128
HDL-C (mg/dl)	-0.020	0.918	-0.117	0.690
LDL-C (mg/dl)	-0.245	0.200	-0.647	0.012

The synergistic effect of antioxidants in human plasma is known to provide greater protection against free radical aggression than any single antioxidant alone (26). The current study measured the total antioxidant activity of plasma because of its established ability to withstand oxidative stress (27). Although statistically insignificant, plasma TAC level was higher in patients with diabetes compared with controls consistent with the results of Savu et al. as well as Kharroubi et al. (28, 29). As shown in limitations, similar plasma TAC levels may be due to insufficient individuals in both groups.

Table 4. Association between dietary total antioxidant capacity (TAC) and biochemical parameters in diabetics and controls

	Dietary TAC (mmol/day)			
	Diabetics (n=29)		Controls (n=30)	
	r	p	r	p
HbA1c (%)	-0.531	0.004	-0.247	0.522
FPG (mg/dL)	-0.406	0.036	0.112	0.577
TG (mg/dL)	-0.042	0.825	-0.364	0.052
HDL (mg/dL)	0.083	0.662	-0.225	0.240
AST (U/L)	-0.049	0.798	-0.464	0.034
ALT (U/L)	0.237	0.207	-0.173	0.429
Uric acid	0.073	0.703	0.493	0.011

Opara et al. (30) reported a decrease in antioxidant levels in diabetic subjects with complications while Srivatsan et al. (31) found an increase in antioxidant levels in diabetic subjects without complications. Most diabetic subjects in our study seem to have no obvious complications. This is consistent with the notion that the increase in free radicals seems to be associated first with an increase in antioxidant levels and with the progression of the disease the antioxidant levels decrease and complications eventually develop. The association between serum uric acid and diabetes mellitus and their findings are not consistent. Some studies reported that there is a positive association between high serum uric acid levels and diabetes (32-34), whereas other studies reported no association (35), or an inverse relationship (36, 37). Significantly increased plasma uric acid concentrations were found in patients with diabetes in the current study.

In type 2 diabetes, chronic exposure to hyperglycemia and insulin resistance has been implicated in altered oxidative metabolism. Several different mechanisms have been proposed to explain why oxidative stress is increased in diabetes mellitus; these mechanisms fall into two general categories: increased production of ROS and decreased antioxidant defences (38). In some previous studies (29,39), no or positive association was found between plasma TAC and HbA1c. However, in the present study, there was a negative correlation between HbA1c and plasma TAC (Table 3). This result was consistent with the results of Wahba et al. (40) and Peerapatdit et al. (41) which can be explained by the fact that hyperglycemia decreases antioxidant levels.

Nutrition is a potent tool in regulating glucose metabolism. Food, through fruit and vegetable consumption, can be a great source of antioxidants that protect the body against oxidative damage and insulin resistance (42). The results of the present work suggest that higher total dietary antioxidant intake is correlated with lower levels of HbA1c and fasting plasma glucose in diabetic individuals (Table 4). Niacin is one of the vitamins belonging to vitamin B complex. Niacin is found in both plant and animal foods. Due to the contribution of tryptophan, foods containing balanced protein may contribute to high niacin equivalent. Niacin compounds provide potential health benefits like combating cardiovascular disease, diabetes, osteoarthritis, neurological problems, and skin diseases (43). A positive and statistically significant correlation was found between plasma TAC and dietary intake of niacin in diabetics in this study. To our knowledge, there has not been a study designed to evaluate between the relationship dietary niacin intake and plasma TAC level in diabetics, thus further research is needed on this topic. Although, several observational studies showing a positive correlation between dietary and plasma TAC (15, 44-49), in our study, no remarkable differences were found between dietary and plasma TAC. FAO/WHO Expert Consultation on diet, nutrition and the prevention of chronic diseases recommends the intake of a minimum of 400 g of fruit and vegetables per day (50). According to Dietary Guidelines for Turkey, vegetable or fruit must be consumed at least 5 servings daily (51). Since the individuals did not consume the recommended amount of vegetables and fruits (diabetics=192 g, controls= 200 g), a significant relationship may have not been found between the diabetics and controls. This is consistent with results from a number of intervention studies (52-54). Studies that monitored dynamic change of plasma TAC immediately following consumption of tea, coffee, red wine, nuts, fruits, and vegetables found a significant increase of plasma TAC that reached its peak 1 or 2 hours after consumption (45,46,48,49). However, studies on the long-term effect of consuming TAC-rich foods on fasting plasma TAC levels reported inconclusive results (17, 52, 54). To date, few studies have examined whether dietary TAC modulates plasma TAC. Our study provides evidence that dietary TAC is not an important modulator of antioxidant status in diabetic subjects.

In conclusion no remarkable differences were found between dietary and plasma TAC in diabetic patients. But, it was shown that the increasing in TAC taken with foods can be effective in controlling HbA1c and fasting plasma glucose.

Limitations

This analysis was performed in a small sample. Longitudinal data on plasma total antioxidant and dietary antioxidant status among diabetic patients is needed.

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