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The effect of dietary total antioxidant capacity of individuals with type 2 diabetes on metabolic and oxidative parameters: A cross-sectional study

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Ethics Committee Approval
The study was approved by the Scientific Research and Publication Ethics Committee of Toros University with decision number 170 dated October 26, 2022.
All procedures in this study involving human participants were performed in accordance with the 1964 Helsinki Declaration and its later amendments.

Conflict of Interest
No conflict of interest was declared by the authors.

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Abstract

Background/Aim: The aim of this study is to determine the dietary total antioxidant capacity (DTAC) values and levels of certain serum oxidative parameters in individuals with previously and newly diagnosed type 2 diabetes and to evaluate the impact of these findings on glycemic values and metabolic parameters.
Methods: This study was conducted with a total of 97 participants aged 19-64, comprising 35 individuals with a previous type 2 diabetes diagnosis, 32 individuals with a recent type 2 diabetes diagnosis, and 30 healthy participants. During face-to-face interviews, participants provided descriptive information, physical activity levels, and anthropometric measurements. DTAC was calculated from three-day dietary intake records using various methods. Serum samples were collected for the analysis of glycemic, lipid, and oxidative parameters.
Results: The results show that DTAC values (specifically derived from total radical-trapping antioxidant potential (TRAP) and total phenolics (TP) values)) and serum TAC levels tend to decrease with both prolonged diabetes age and when compared to individuals without diabetes ($P<0.05$). DTAC values were found to have a significant effect on some oxidative parameters like TAC, paraoxonase 1, and arylesterase ($P<0.05$), while serum oxidative parameters were found to have no significant effect on glycemic and lipid parameters.
Conclusion: It was concluded that low DTAC may be a risk factor related to oxidative stress depending on type 2 diabetes and diabetes age.

Keywords: type 2 diabetes, dietary total antioxidant capacity, glycemic control, oxidative parameters, metabolic parameters

Introduction

Type 2 diabetes mellitus (DM) is a metabolic disease characterized by hyperglycemia and insulin resistance [1]. DM, which progresses with chronic hyperglycemia, creates an inflammatory environment in the body and creates an important ground for the formation of reactive oxygen derivatives (ROS). The increase in ROS production and the decrease in antioxidant concentrations lead to oxidative stress, which can be associated with elevated plasma glucose levels and complications arising from diabetes [2].

Experimental and clinical studies have shown that oxidative stress (OS) plays an important role in the pathogenesis of type 2 DM. High levels of free radicals and insufficient antioxidant defense mechanisms can damage cellular organelles and enzymes, increase lipid peroxidation, and lead to the development of insulin resistance [3, 4]. While it has been reported that individuals with type 2 DM have decreased total antioxidant capacity and increased levels of oxidative stress biomarkers [5], increasing the intake of common dietary antioxidants has been shown to reduce insulin resistance and promote glycemic improvement [6, 7]. Dietary compounds with antioxidant activity may exert antioxidant effects cumulatively or synergistically [7]. Fruits and vegetables, fatty seeds, wine, tea, and coffee are the foods that contribute the most to the overall antioxidant capacity of the diet [8]. Dietary antioxidants both prevent cellular oxidative damage by preventing excessive free radical formation and alleviate the progression of oxidative stress-induced conditions by preventing cellular degeneration from further progressing after damage [9].

Since assessing antioxidants individually may not provide a complete picture of a diet's overall antioxidant potential and might ignore synergistic interactions, researchers have introduced a cumulative approach known as dietary total antioxidant capacity (DTAC), which evaluates the collective effectiveness of all dietary antioxidants in combating reactive compounds [10, 11]. In studies conducted with different sample groups, the protective role of high DTAC values against oxidative stress has been addressed, and it has been found that high DTAC values may reduce the risk of hypertension, dyslipidemia, and retinopathy in cross-sectional studies [12, 13], while cohort studies have shown an association with lower cancer and cardiovascular risk [14, 15].

Today, there is no gold standard for measuring ROS-mediated tissue damage. Instead of separately evaluating the antioxidant and oxidant effects against antioxidant presence, which is costly, time-consuming, and challenging to measure technically or still undiscovered, it is recommended to measure total antioxidant capacity (TAC) and total oxidant capacity (TOC). Therefore, both TAC and TOC serve as logical approaches to the evaluation of OS [16]. In addition, the Oxidative Stress Index (OSI), which more clearly defines oxidant-antioxidant imbalances in chronic inflammatory diseases, has been developed [17].

Considering the duration of diabetes, no studies evaluating the effects of DTAC values on serum oxidative, glycemic, and lipid parameters in type 2 diabetics have been discovered. The purpose of this study is to determine the DTAC values of individuals with type 2 DM who have been diagnosed either recently or previously, and to evaluate the effects of these findings on glycemic levels and lipid parameters.

Materials and methods

This cross-sectional and comparative case-control study was conducted between October 2022 and March 2023 with a total of 97 individuals, including 35 participants with a previous diagnosis of type 2 diabetes mellitus (with a diabetes duration of at least five years), 32 participants with a recent diagnosis of type 2 diabetes mellitus, and 30 healthy participants. All participants were followed at the Internal Medicine and Endocrinology outpatient clinics of Mersin University Faculty of Medicine. The inclusion criteria for healthy individuals participating in the study required that they present to the hospital for routine checks, have no diagnosis of any disease, and fall within the same age range as individuals with type 2 diabetes, while those with inflammatory conditions (such as rheumatoid arthritis) or chronic diseases. Individuals diagnosed with cancer, users of oral antidiabetic agents other than biguanide derivatives, pregnant and lactating women, as well as individuals who smoke or take antioxidant dietary supplements were excluded from the study. For the study to be conducted, ethical approval was obtained from the Scientific Research and Publication Ethics Committee of Toros University with decision number 170 dated October 26, 2022. Written informed consent was obtained from participants before the study commenced. To determine the sample size, a power analysis was conducted using G*Power software with an alpha (α) level of 0.05, power (1-β) of 0.98, and a medium effect size (d=0.50). The analysis determined that a total of 90 observations would achieve an approximate test power of 100% for this study.

In the study, face-to-face interviews were conducted with the individuals to inquire about their descriptive characteristics (age, gender, marital status, education duration) and physical activity status. Anthropometric measurements (body weight, height, waist and hip circumference) were taken. In addition, DTAC values (FRAP1, FRAP2, TRAP, TEAC, H-ORAC, L-ORAC, Total-ORAC, TP values) were calculated by using three-day food consumption records of individuals, two days on weekdays and one day on weekends, and serum samples were taken for serum glycemic (fasting blood glucose (FBG), glycated hemoglobin (A1c)), lipid (total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides) and oxidative parameters (TAC, TOC, PON-1, ARES).

Anthropometric measurements

Body weight, height, waist and hip circumference measurements were taken during face-to-face interviews [18]. BMI values (body weight (kg)/height (m^2)) were calculated using body weight and height measurements and evaluated according to the World Health Organization (WHO) classifications (BMI: <18.5 kg/m^2 is weak, between 18.5-24.9 kg/m^2 is normal, between 25.0-29.9 kg/m^2 is overweight, ≥30.0 kg/m^2 is obese) [19]. In addition, the waist-hip ratio was calculated by proportioning the waist and hip circumference measurements, and the waist-hip ratio was calculated by proportioning the height measurements. The individuals' waist-to-height ratios were classified according to the classification developed by Ashwell et al. [20] (<0.5 normal,

0.5-0.6 risk, and ≥0.6 high risk), while the waist-to-hip ratios were assessed based on WHO criteria (waist-to-hip ratio: men: <0.9; women: <0.85) [21].

Calculation of total antioxidant capacity of diets

In calculating the total antioxidant capacity of diets, three different databases were utilized to assess the ferric-reducing antioxidant power (FRAP), Trolox equivalent antioxidant capacity (TEAC), total radical-trapping antioxidant potential (TRAP), and oxygen radical absorbance capacity (ORAC) of the foods [22-24]. The estimated FRAP values according to the database created by Carlsen et al. [22] were named FRAP-1 analysis, and the FRAP values estimated according to the database created by Pellegrini et al. [23, 24] were named FRAP-2. According to the database created by the United States Department of Agriculture (USDA), ORAC values were evaluated by hydrophilic-ORAC (H-ORAC), lipophilic-ORAC (L-ORAC), total-ORAC and total Phenolics (TP) analyses [25]. When calculating the total antioxidant capacity of the diets, the FRAP1, FRAP2, TRAP, TEAC, H-ORAC, L-ORAC, Total-ORAC, and TP values for each food item listed in the databases were defined in the Nutrition Information System (BeBİS) program [26], and 24-hour feedback from individuals was collected by dieticians, one day in person and one day by telephone. The dieticians used the BeBiS program to determine the average daily total antioxidant capacity of their diets from their two-day food consumption records. In cases where DTAC values could not be determined, the values of the nutrients with the most similarities were taken.

Biochemical parameters

In the study, measurements of FBG, A1c, total cholesterol, LDL cholesterol, HDL cholesterol, and triglycerides were conducted on serum samples obtained after a 12-hour fasting period during routine analyses. Serums obtained after centrifugation from blood samples were portioned into eppendorf tubes and stored at -20°C until analysis to evaluate TAC, TOC, paraoxanase 1 (PON1), and arylesterase (ARES) enzyme activity levels. The analysis of these samples, gradually solubilized, was performed using a spectrophotometric method with an automatic microplate reader (Mindray BS300), employing suitable local commercial kits (RelAssay® Diagnostics, Diagen, Ankara). The formula OSI =TOS/(TAS×100) was used to calculate the oxidative stress index (OSI) [17].

Statistical analysis

During the statistical analysis phase of the study, the relationships between categorical variables (n x r) were examined; in cases where at least one of the expected values of the cells was less than five, the Fisher test was applied, while the Pearson chi-square test of independence was used when all cells were greater than five. The skewness and kurtosis values of the variables were calculated on a group basis, and it was observed that these values fell within the range indicating suitability for normal distribution. Therefore, parametric tests were preferred in the study. The independent samples t-test was used to compare the means between two quantitative variables, while the independent samples ANOVA test was used to compare the means between three groups. Multiple comparisons were examined with the Tukey test in cases where the significance values were less than 0.05 in the ANOVA test. In the later stage of the study, logistic regression analysis was applied to examine the effects of independent variables on dependent variables. In logistic regression analysis, independent (quantitative) variables were divided into two groups, high and low, according to their median values prior to being used in the model. In all calculations and interpretations, the statistical significance level was considered as $P<0.05$. Statistical analysis of the data was performed using R software [27] and the IBM SPSS 26 statistical package program [28].

Results

The distribution of the general characteristics of the study participants is presented in Table 1. While no significant relationship was found between the mean age of the participants in the study, it was determined that individuals with a previous diagnosis of type 2 diabetes had shorter education durations, whereas the control group had longer education durations compared to the other groups ($P<0.001$). In terms of gender distribution, analysis showed that among individuals with a previous diagnosis of type 2 diabetes, females comprised 68.6%, while males constituted 56.2% of those with a new diagnosis ($P<0.001$). However, the rate of individuals with type 2 diabetes with a family history of the disease (82.9%) was higher than those with newly diagnosed type 2 diabetes (59.4%) ($P<0.001$). Evaluation of individuals with type 2 diabetes in terms of treatment methods showed that the rate of those treated with diet and oral antidiabetic drugs (OAD) (71.4%; 65.6%, respectively) and the rate of those receiving diet and insulin treatment (28.6%; 18.8%, respectively) was higher in individuals with both previously and newly diagnosed type 2 diabetes. However, it was determined that the rate of those treated with diet alone (15.6%) among individuals with newly diagnosed type 2 diabetes was lower than other treatment methods ($P<0.001$). According to the findings, it was determined that the waist/hip ratios, waist/height ratios, and BMI values of the participants in the control group were significantly lower than those of individuals with previously diagnosed type 2 diabetes ($P<0.001$) (Table 1).

There was no statistically significant difference between the FRAP1, TEAC, H-ORAC, L-ORAC, Total ORAC, TOC, PON-I and OSI values of the individuals with type 2 diabetes and the individuals in the control group. In addition, it was determined that the TRAP and TP values of the previously diagnosed type 2 diabetic individuals were significantly lower than both the control group ($P=0.004$) and the newly diagnosed type 2 diabetic individuals ($P=0.003$), and the FRAP2 values were significantly lower than the control group only ($P=0.005$). It was further determined that the levels of FBG, A1c, and LDL-K in the control group were significantly lower than in the other groups. The total cholesterol level was significantly lower only when compared to individuals newly diagnosed with type 2 diabetes ($P<0.001$), while the HDL cholesterol level was higher than in the other groups (previous diagnosis $P=0.002$; new diagnosis $P=0.01$). It was shown that the TAC levels of individuals with a previous diagnosis of type 2 diabetes were also significantly lower than those of both the newly diagnosed type 2 diabetes and the control group ($P<0.001$), while the ARES levels decreased significantly based on the ranking of previous diagnosis, new diagnosis, and control groups ($P<0.001$) (Table 2).

Table 1: Comparison of general and anthropometric characteristics of individuals participating in the study

Variables	Previous Diagnosis (n=35)	New Diagnosis (n=32)	Control Group (n=30)	*P*	*P1*	*P2*	*P3*
Age (year)	43.9 (7.3)	41.3 (10.8)	39.7 (7.4)	0.770A	0.853	0.562	0.879
Gender				<0.001c**			
Male	11 (31.4%)	18 (56.2%)	3 (10%)				
Female	24 (68.6%)	14 (43.8%)	27 (90%)				
Duration of education (year)	8.9 (4.1)	8.3 (5.2)	15.8 (3.6)	<0.001A**	0.046*	<0.001**	<0.001**
Family history of DM				<0.001f**			
Yes	6 (17.1%)	13 (40.6%)	30 (100%)				
No	29 (82.9%)	19 (59.4%)	0 (0%)				
Additional diseases							
Obesity	15 (42.9%)	12 (37.5%)	1 (3.3%)	<0.001f**			
Hypertension	15 (42.9%)	10 (31.3%)	0 (0%)	<0.001f**			
CVD	5 (14.3%)	8 (25.0%)	0 (0%)	0.010f**			
Kidney defects	1 (2.9%)	2 (6.3%)	0 (0%)	0.522f			
Eye diseases	3 (8.6%)	1 (3.1%)	0 (0%)	0.322f			
Thyroid diseases	7 (20.0%)	5 (15.6%)	0 (0%)	0.023f*			
Treatment method				<0.001f**			
Diet	0 (0%)	5 (15.6%)	0 (0%)				
Diet + OAD	25 (71.4%)	21 (65.6%)	0 (0%)				
Diet + insulin treatment	10 (28.6%)	6 (18.8%)	0 (0%)				
Regular diet				0.498f			
Yes	5 (14.3%)	3 (27.3%)	0 (0%)				
Occasional	17 (48.6%)	3 (27.3%)	0 (0%)				
No	13 (37.1%)	5 (45.5%)	1 (100%)				
Waist/hip ratio	0.95 (0.10)	0.92 (0.10)	0.89 (0.10)	<0.001A**	0.419	<0.001**	0.146
Waist/height ratio	0.62 (0.10)	0.61 (0.10)	0.59 (0.10)	<0.001A**	0.993	<0.001**	0.804
BMI (kg/m²)	31.2 (4.9)	30.2 (6.5)	28.8 (5.3)	<0.001A**	0.753	<0.001**	0.465
Energy intake (kcal)	1657.9 (447.6)	1799.1 (715.1)	1859.2 (412.3)	0.306A			
Physical activity level	1.65 (0.32)	1.69 (0.13)	1.73 (0.14)	0.596A			

P: Overall significance; *P*1: New diagnosis vs Previous diagnosis; *P*2: Control vs Previous diagnosis; *P*3: Control vs New diagnosis; A: ANOVA; C: Chi-square test; F: Fisher's exact test; DM: Diabetes Mellitus; OAD: Oral Antidiabetic Drug; BMI: Body Mass Index; CVD: Cardiovascular Disease. **P*<0.05, ***P*<0.01, ****P*<0.001

Table 2: Dietary total antioxidant capacities and biochemical parameters of the individuals participating in the study

Variables	Previous Diagnosis (n=35)	New Diagnosis (n=32)	Control Group (n=30)	*P*	*P1*	*P2*	*P3*
FRAP1	7.6 (3.5)	9.9 (7.3)	8.4 (3.8)	0.206	0.183	0.809	0.516
FRAP2	2.0 (1.0)	2.4 (1.4)	2.9 (1.2)	0.008*	0.343	0.005*	0.179
TRAP	55.6 (47.8)	78.2 (88.7)	77.4 (46.5)	0.003*	0.003*	0.004*	0.898
TEAC	60.7 (52.3)	64.7 (58.0)	67.2 (37.2)	0.870	0.943	0.862	0.979
H-ORAC	15512.6 (6290.5)	19789.6 (10434.3)	18517.9 (6541.4)	0.083	0.077	0.288	0.805
L-ORAC	2219.1 (2701.0)	2895.5 (5467.5)	3561.9 (4275.2)	0.480	0.800	0.811	0.446
Total ORAC	18596.5 (9966.1)	22618.0 (11041.9)	22282.3 (9085.6)	0.198	0.238	0.310	0.991
TP	1116.9 (435.3)	1524.4 (789.8)	1696.1 (669.6)	<0.001**	0.029*	<0.001**	0.546
FBG (mmol/L)	8.7 (3.7)	9.2 (5.0)	5.2 (0.4)	<0.001**	0.804	<0.001**	<0.001**
A1c (%)	7.3 (1.5)	7.7 (2.1)	5.3 (0.5)	<0.001**	0.168	<0.001**	<0.001**
Total-K (mg/dL)	201.2 (52.3)	227.5 (44.1)	180.9 (43.2)	<0.001**	0.071	0.194	<0.001**
LDL-K (mg/dL)	121.5 (37.7)	134.8 (41.1)	91.7 (35.2)	<0.001**	0.360	0.006*	<0.001**
HDL-K (mg/dL)	50.6 (11.3)	52.3 (11.7)	61.6 (14.5)	<0.001**	0.846	0.002*	0.012*
Triglyceride (mg/dL)	195.4 (199.6)	210.7 (132.8)	93.0 (51.6)	0.003*	0.903	0.015*	0.005*
TAC (mmol/L)	1.5 (0.2)	1.6 (0.2)	1.7 (0.2)	<0.001**	<0.001**	<0.001**	0.861
TOC (µmol/L)	6.4 (4.1)	5.9 (1.9)	5.2 (2.3)	0.269	0.852	0.245	0.541
PON-1 (U/L)	322.6 (198.9)	319.4 (205.4)	323.1 (227.4)	0.997	0.998	1.000	0.997
ARES (µmol/L)	626.1 (91.8)	579.9 (64.7)	481.9 (36.1)	<0.001**	<0.001**	<0.001**	0.021*
OSI	0.4 (0.2)	0.4 (0.1)	0.4 (0.1)	0.785	0.938	0.766	0.934

P: Overall significance; *P1:* New diagnosis - Previous diagnosis; *P2:* Control - Previous diagnosis; *P3:* Control - New diagnosis; A: ANOVA test; T: Independent samples t-test; Tukey test was used in multiple comparisons; FRAP: Ferric Reducing Antioxidant Activity; TEAC: Trolox Equivalent Antioxidant Capacity; TRAP: Total Radical Capture Antioxidant Potential; H-ORAC: Hydrophilic Oxygen Radical Absorption Capacity; L-ORAC: Lipophilic Oxygen Radical Absorption Capacity; TP: Total Phenolics; FBG: Fasting Blood Glucose; A1c: Glycated Hemoglobin; Total Cholesterol: Total-K; LDL-K: Low Density Lipoprotein; HDL-K: High Density Lipoprotein; TAC: Total Antioxidant Capacity; TOC: Total Oxidant Capacity; PON-1: Paraoxonase 1; ARES: Arylesterase; OSI: Oxidative Stress Index; **P*<0.05, ***P*<0.01, *P*<0.001

Linear regression models showing the effect of the dietary antioxidant capacities of the individuals participating in the study on their blood oxidative parameters are presented in Table 3. Findings from individuals with previous type 2 diabetes indicated that only the TRAP value had a significant effect on ARES level (*P*=0.004). Upon examining this effect, it was determined that a high TRAP value decreased the ARES level by 108.885 units compared to a low TRAP value. In individuals newly diagnosed with type 2 diabetes, FRAP1, FRAP2, TRAP, TEAC, H-ORAC, L-ORAC, and Total ORAC values did not have a statistically significant effect on TAC, PON-1, and ARES levels. However, FRAP1, and TEAC values exerted a substantial effect on TOC and OSI (respectively *P*=0.03, *P*=0.05; *P*=0.03, *P*=0.04). When this effect was examined, it was found that the high FRAP1 value increased the TOC level by 3.499 units and the OSI level by 0.220 units compared to the low FRAP1 value, while the high TEAC group decreased the TOC level by 3.874 units and the OSI level by 0.252 units compared to the low TEAC group. In the control group, the Total ORAC value was found to have a significant effect on TAC level, TEAC value on PON-1 level, and L-ORAC values on the ARES level (respectively *P*=0.02; *P*=0.01; *P*=0.02). When these effects were examined individually, it was seen that a high Total ORAC value increased the TAC level by 0.280 units compared to a low Total ORAC value; a high TEAC value increased the PON-1 level by 426.534 units compared to a low TEAC value; and a high L-ORAC value increased the ARES level by 36.919 units compared to a low L-ORAC value.

Table 4 shows the linear regression models showing the effect of serum oxidative stress parameters of the study groups on glycemic control and lipid profiles. Considering the values of individuals with previously diagnosed and newly diagnosed type 2 diabetes, it was determined that the levels of TAC, TOC, PON-1, ARES, and OSI did not have a significant effect on the levels of FBG, A1c, LDL-K, HDL-K, and Total-K. Considering the values for individuals with both previously and newly diagnosed type 2 diabetes, it was observed that TAC, TOC, PON-1, ARES, and OSI levels did not significantly affect FBG, A1c, LDL-C, HDL-C, and Total-C levels. Again, in the control group, PON-1, ARES and OSI values did not have a significant effect on HDL-K

Table 3: Linear regression models showing the effect of dietary antioxidant capacity on blood oxidative parameters

Response	Regressor	Previous diagnosis					New diagnosis					Control group				
		B	*P*	95%CI		R^2	B	*P*	95%CI		R^2	B	*P*	95%CI		R^2
				Lower	Upper				Lower	Upper				Lower	Upper	
TAC	**Intercept**	1.668	**<0.001**	1.498	1.837	0.097	1.664	**<0.001**	1.499	1.829	0.184	1.545	**<0.001**	1.425	1.665	0.417
	FRAP1 ***(Ref=Low)***	0.009	0.951	-0.298	0.316		0.012	0.947	-0.379	0.355		-0.149	0.141	-0.350	0.053	
	FRAP2 ***(Ref=Low)***	-0.055	0.593	-0.261	0.152		0.043	0.596	-0.123	0.210		-0.064	0.407	-0.222	0.093	
	TRAP ***(Ref=Low)***	0.041	0.705	-0.178	0.259		-0.008	0.950	-0.256	0.241		-0.077	0.587	-0.365	0.211	
	TEAC ***(Ref=Low)***	-0.095	0.448	-0.348	0.158		0.065	0.744	-0.341	0.471		0.095	0.412	-0.140	0.330	
	H-ORAC ***(Ref=Low)***	0.184	0.252	-0.138	0.507		-0.028	0.820	-0.277	0.221		-0.173	0.067	-0.358	0.013	
	L-ORAC ***(Ref=Low)***	0.030	0.791	-0.199	0.259		0.089	0.336	-0.097	0.275		-0.041	0.552	-0.182	0.100	
	Total ORAC ***(Ref=Low)***	-0.109	0.399	-0.370	0.152		-0.191	0.171	-0.471	0.088		0.280	**0.016***	0.059	0.502	
TOC	**Intercept**	6.453	**<0.001**	3.344	9.561	0.063	5.563	**<0.001**	4.131	6.994	0.285	5.343	**<0.001**	3.432	7.254	0.185
	FRAP1 ***(Ref=Low)***	-0.655	0.813	-6.282	4.972		3.499	**0.032***	0.317	6.680		0.225	0.885	-2.983	3.434	
	FRAP2 ***(Ref=Low)***	-0.634	0.734	-4.422	3.155		0.228	0.747	-1.216	1.673		1.550	0.213	-0.958	4.057	
	TRAP ***(Ref=Low)***	2.029	0.308	-1.976	6.035		2.109	0.055	-0.051	4.268		-1.997	0.376	-6.581	2.587	
	TEAC ***(Ref=Low)***	-0.594	0.795	-5.227	4.040		-3.874	**0.033***	-7.400	-0.348		0.378	0.836	-3.360	4.117	
	H-ORAC ***(Ref=Low)***	0.405	0.889	-5.507	6.316		-0.872	0.413	-3.033	1.289		1.247	0.391	-1.707	4.201	
	L-ORAC ***(Ref=Low)***	0.738	0.721	-3.463	4.939		-0.632	0.427	-2.247	0.983		-0.815	0.460	-3.063	1.434	
	Total ORAC ***(Ref=Low)***	-1.450	0.539	-6.229	3.330		0.350	0.769	-2.078	2.779		-1.050	0.544	-4.581	2.481	
PON-1	**Intercept**	276.142	**<0.001**	127.555	424.729	0.076	315.872	**<0.001****	146.823	484.920	0.150	182.528	**<0.001**	32.871	332.186	0.476
	FRAP1 ***(Ref=Low)***	-56.362	0.671	-325.333	212.609		-100.096	0.588	-475.839	275.647		-123.528	0.319	-374.808	127.753	
	FRAP2 ***(Ref=Low)***	-46.298	0.604	-227.395	134.799		4.685	0.955	-165.891	175.262		71.284	0.460	-125.093	267.661	
	TRAP ***(Ref=Low)***	88.387	0.352	-103.067	279.841		-115.210	0.361	-370.278	139.859		-317.689	0.080	-676.684	41.307	
	TEAC ***(Ref=Low)***	55.340	0.612	-166.116	276.796		258.230	0.213	-158.240	674.701		426.534	**0.006***	133.754	719.313	
	H-ORAC ***(Ref=Low)***	57.227	0.681	-225.338	339.792		-91.317	0.468	-346.609	163.976		145.787	0.205	-85.559	377.133	
	L-ORAC ***(Ref=Low)***	64.536	0.515	-136.253	265.325		-56.797	0.545	-247.532	133.938		113.531	0.195	-62.561	289.623	
	Total ORAC ***(Ref=Low)***	-72.529	0.520	-300.973	155.915		107.231	0.448	-179.635	394.096		-39.627	0.769	-316.183	236.929	
ARES	**Intercept**	611.942	**<0.001**	556.797	667.088	0.402	564.442	**<0.001**	511.046	617.837	0.144	476.432	**<0.001**	451.167	501.697	0.404
	FRAP1 ***(Ref=Low)***	87.104	0.085	-12.720	186.928		14.156	0.808	-104.526	132.837		16.818	0.420	-25.602	59.239	
	FRAP2 ***(Ref=Low)***	13.130	0.692	-54.081	80.342		47.200	0.083	-6.678	101.078		11.243	0.489	-21.909	44.395	
	TRAP ***(Ref=Low)***	-108.885	**0.004***	-179.940	-37.830		1.730	0.965	-78.835	82.295		-35.129	0.242	-95.734	25.476	
	TEAC ***(Ref=Low)***	73.492	0.078	-8.698	155.682		-15.778	0.807	-147.324	115.767		-13.311	0.582	-62.737	36.116	
	H-ORAC ***(Ref=Low)***	-67.069	0.200	-171.938	37.800		3.898	0.921	-76.738	84.534		21.248	0.271	-17.807	60.304	
	L-ORAC ***(Ref=Low)***	-19.426	0.597	-93.945	55.094		-9.254	0.754	-69.499	50.991		36.919	**0.017***	7.191	66.646	
	Total ORAC ***(Ref=Low)***	49.099	0.245	-35.684	133.882		-13.848	0.755	-104.456	76.761		-27.603	0.233	-74.291	19.085	
OSI	**Intercept**	0.388	**<0.001**	0.206	0.570	0.041	0.346	**<0.001**	0.248	0.444	0.301	0.347	**<0.001**	0.212	0.482	0.206
	FRAP1 ***(Ref=Low)***	-0.031	0.850	-0.361	0.299		0.220	**0.047***	0.003	0.437		0.057	0.605	-0.170	0.285	
	FRAP2 ***(Ref=Low)***	-0.021	0.847	-0.243	0.201		-0.006	0.894	-0.105	0.092		0.130	0.142	-0.047	0.308	
	TRAP ***(Ref=Low)***	0.090	0.441	-0.145	0.324		0.123	0.097	-0.024	0.271		-0.117	0.463	-0.441	0.208	
	TEAC ***(Ref=Low)***	-0.002	0.987	-0.274	0.270		-0.252	**0.041***	-0.493	-0.011		0.005	0.972	-0.260	0.269	
	H-ORAC ***(Ref=Low)***	-0.019	0.909	-0.366	0.327		-0.044	0.548	-0.191	0.104		0.114	0.269	-0.095	0.324	
	L-ORAC ***(Ref=Low)***	0.034	0.776	-0.212	0.281		-0.061	0.263	-0.172	0.049		-0.047	0.543	-0.207	0.112	
	Total ORAC ***(Ref=Low)***	-0.055	0.691	-0.335	0.225		0.069	0.398	-0.097	0.235		-0.138	0.265	-0.388	0.112	

B: Regression coefficient, CI: Confidende interval, R^2: Coefficient of determination, Ref: Reference group; FRAP: Ferric Reducing Antioxidant Activity; TEAC: Trolox Equivalent Antioxidant Capacity; TRAP: Total Radical Capture Antioxidant Potential; H-ORAC: Absorbing Capacity of Hydrophilic Oxygen Radical; L-ORAC: Absorbing Capacity of Lipophilic Oxygen Radical; TP: Total Phenolics.; TAC: Total Antioxidant Capacity; TOC: Total Oxidant Capacity; PON-1: Paraoxanase 1; ARES: Arylesterase; OSI: Oxidative Stress Index; *P<0.05, **P<0.01, P<0.001

Table 4: Linear regression models showing the effect of serum oxidative stress parameters on glycemic control and cholesterol values

Response	Regressor	Previous diagnosis					New diagnosis					Control group				
		B	*P*	95% CI		R^2	B	*P*	95% CI		R^2	B	*P*	95% CI		R^2
				Lower	Upper				Lower	Upper				Lower	Upper	
FBG	**Intercept**	149.745	**<0.001**	81.488	218.002	0.130	130.395	**0.013**	29.987	230.804	0.047	92.021	**<0.001**	85.730	98.311	0.176
	TAC ***(Ref=Low)***	-22.347	0.435	-80.141	35.447		24.379	0.533	-54.889	103.647		1.466	0.696	-6.184	9.116	
	TOC ***(Ref=Low)***	32.465	0.289	-28.956	93.887		-37.602	0.499	-150.307	75.104		3.867	0.334	-4.230	11.963	
	PON-1 ***(Ref=Low)***	-6.407	0.791	-55.310	42.497		10.207	0.781	-64.341	84.756		-4.966	0.148	-11.827	1.894	
	ARES ***(Ref=Low)***	35.787	0.135	-11.788	83.361		35.444	0.359	-42.591	113.480		0.870	0.764	-5.047	6.786	
	OSI ***(Ref=Low)***	-28.535	0.403	-97.337	40.267		35.027	0.530	-78.179	148.234		0.833	0.837	-7.437	9.102	
A1c	**Intercept**	7.028	**<0.001**	5.559	8.497	0.186	6.314	**<0.001**	4.112	8.516	0.168	5.171	**<0.001**	4.827	5.515	0.389
	TAC ***(Ref=Low)***	-0.374	0.543	-1.618	0.870		-0.065	0.940	-1.803	1.674		0.420	0.049	0.002	0.838	
	TOC ***(Ref=Low)***	-0.220	0.736	-1.542	1.102		1.940	0.119	-0.531	4.412		0.333	0.134	-0.110	0.775	
	PON-1 ***(Ref=Low)***	-0.622	0.236	-1.675	0.430		0.461	0.567	-1.174	2.096		-0.436	**0.024***	-0.811	-0.062	
	ARES ***(Ref=Low)***	0.743	0.149	-0.281	1.767		1.103	0.197	-0.608	2.815		-0.086	0.588	-0.409	0.237	
	OSI ***(Ref=Low)***	0.420	0.567	-1.061	1.900		-0.774	0.527	-3.257	1.708		0.086	0.698	-0.366	0.538	
LDL-K	**Intercept**	103.541	**<0.001**	66.413	140.668	0.085	133.585	**<0.001**	90.515	176.655	0.158	87.836	**<0.001**	58.049	117.624	0.232
	TAC ***(Ref=Low)***	2.888	0.852	-28.548	34.325		4.086	0.807	-29.915	38.088		10.977	0.538	-25.247	47.202	
	TOC ***(Ref=Low)***	-4.608	0.780	-38.018	28.801		24.687	0.304	-23.657	73.032		11.952	0.526	-26.386	50.291	
	PON-1 ***(Ref=Low)***	-16.861	0.205	-43.461	9.740		-4.832	0.759	-36.809	27.145		9.710	0.543	-22.776	42.195	
	ARES ***(Ref=Low)***	-9.703	0.449	-35.581	16.174		-18.791	0.259	-52.264	14.682		22.004	0.118	-6.011	50.019	
	OSI ***(Ref=Low)***	5.524	0.765	-31.900	42.949		-1.627	0.946	-50.186	46.933		9.763	0.612	-29.395	48.921	
HDL-K	**Intercept**	48.441	**<0.001**	37.486	59.397	0.222	55.323	**<0.001**	42.915	67.732	0.134	52.004	**<0.001**	41.358	62.650	0.338
	TAC ***(Ref=Low)***	6.619	0.155	-2.657	15.895		-1.682	0.727	-11.477	8.114		15.033	**0.025***	2.086	27.979	
	TOC ***(Ref=Low)***	-4.930	0.315	-14.789	4.928		10.389	0.137	-3.539	24.316		-14.778	**0.036***	-28.480	-1.076	
	PON-1 ***(Ref=Low)***	4.899	0.212	-2.950	12.748		0.801	0.860	-8.412	10.013		0.175	0.975	-11.435	11.785	
	ARES ***(Ref=Low)***	-6.592	0.088	-14.228	1.044		-5.169	0.281	-14.812	4.474		6.956	0.165	-3.056	16.968	
	OSI ***(Ref=Low)***	3.838	0.483	-7.205	14.881		-10.093	0.150	-24.083	3.896		10.855	0.123	-3.140	24.849	

B: Regression coefficient, CI: Confidende interval, R^2: Coefficient of determination, Ref: Reference group; FBG: Fasting Blood Glucose; A1c: Glycated Hemoglobin; Total-K: Total Cholesterol; LDL-K: Low Density Lipoprotein; HDL-K: High Density Lipoprotein; TAC: Total Antioxidant Capacity; TOC: Total Oxidant Capacity; PON-1: Paraoxanase 1; ARES: Arylesterase; OSI: Oxidative Stress Index; *P<0.05, **P<0.01, P<0.001

level, while TAC and TOC levels had a significant effect on HDL-K level (respectively P=0.03; P=0.04). Accordingly, it was found that the high TAC level increased the mean HDL-K level by 15.033 units compared to the low TAC level, while the high TOC level decreased the HDL-K level by 14.778 units compared to the low TOC level.

Discussion

In this study, it was found that DTAC values and serum TAC levels in individuals with type 2 diabetes tend to decrease both with increasing diabetes duration and compared to non-diabetic individuals, while DTAC values have a significant effect on certain oxidative parameters, and serum oxidative parameters do not have an effect on glycemic and lipid parameters.

Oxidative stress occurs as a result of the imbalance between ROS production and destruction and is shown as a potential predictor of both type 2 diabetes and the risk of complications [29, 30]. Conversely, chronic hyperglycemia creates a risk factor for ROS formation, causing type 2 diabetes to deepen and increase the likelihood of complications [31]. The effect of hyperglycemia in ROS accumulation can occur in different ways. The most effective factor is suggested to be the increased use of the glycolytic pathway due to rising hyperglycemia, which, in turn, leads to an accumulation of ROS through heightened electron pressure on the mitochondrial electron transport system [32]. Another way is that increased ROS production is linked to insulin resistance and plays a role in β-cell dysfunction by providing pancreatic β-cell apoptosis [33, 34]. ROS accumulation due to increased hyperglycemia also plays an important role in the formation of diabetes complications [34]. Serum TOC and TAC, which measure the synergistic and cumulative effects of all oxidants and antioxidants, are known to be associated with type 2 diabetes [35]. Although ROS levels rise in individuals with type 2 diabetes with increased diabetes duration, serum antioxidant levels may increase, decrease, or remain the same [35-39]. In this study, in accordance with the work of Kharroubi et al. [39], it was concluded that the serum TAC levels of individuals with a previous diagnosis of type 2 diabetes were lower compared to those in the newly diagnosed and control groups (Table 2). It was further determined that serum TAC levels had no effect on glycemic biomarkers and lipid profile (Table 4). In addition to practical methods that measure total oxidant and total antioxidant levels in serum, PON1 and ARES enzymes are also enzymes that are encoded by the same gene and act as antioxidants in the esterase group with similar active centers. Although it is known that PON1 shows a polymorphic change, the ARES enzyme does not show a genetic polymorphic change. The PON1 enzyme also has antioxidant function due to its ability to protect LDL cholesterol from oxidation and its capacity to neutralize other radicals, including hydrogen peroxide. ARES, in contrast, is accepted as an indicator of the main protein that is not affected by the changes in PON1 [40]. Conditions that increase oxidative stress such as diabetes, hypercholesterolemia, and cardiovascular diseases may cause low PON1 activity due to increased oxidative stress [32]. Therefore, monitoring trends in complications through PON1 may play an important role in the treatment of individuals with type 2 diabetes [33]. In the group of patients with diabetic complications, levels of FBG and TG were found to be higher compared to the control group, while levels of HDL-K and PON1 were lower. It was also reported that HDL-K in individuals with complications of type 2 diabetes is positively correlated with PON1. In the present study, although there was no difference between the groups in PON1 levels (Table 2), it was observed that a one-unit increase in PON1 level only in the control group provided a 0.436-unit decrease in A1c level (Table 4). Another significant outcome of the study is that serum ARES levels were significantly higher in previously diagnosed type 2 diabetes patients compared to both the newly diagnosed and the control group (Table 2). This finding, which differs from the literature, can be interpreted as the endogenous high production of free oxygen radicals due to increased inflammation associated with the duration of diabetes to mitigate their effects [41].

Mechanisms that increase oxidative stress in diabetes include non-enzymatic glycosylation, autoxidative glycosylation, sorbitol pathway activity, hypoxia, and various changes in the antioxidant defense system. There are increases in lipid peroxidation products in the serum and tissues of individuals with diabetes [42]. Dietary antioxidants, however, can play a protective role against type 2 diabetes by increasing the formation of free radicals in type 2 diabetes and reducing radical binding systems [43]. In a study conducted with individuals with type 2 diabetes, where DTAC values were determined through TRAP, FRAP, and TEAC analyses, it was found that the DTAC values of individuals with type 2 diabetes were lower than those of the healthy group, and there was a negative correlation between DTAC values and glycemic biomarkers [44]. Similarly, in a recent cohort study by Mancini et al. [45], higher DTAC values were associated with a lower risk of type 2 diabetes. In a study conducted by Schaft et al. [46] on individuals with type 2 diabetes, it was reported that individuals with type 2 diabetes had lower FRAP values than the control group, and in addition, it was observed that the FRAP values of the group with higher diabetes age were statistically lower than the newly diagnosed group. According to the results of this study, it was observed that the DTAC values of individuals with a previous diagnosis of type 2 diabetes were at a lower level compared to both the newly diagnosed and the control group, while among these markers, TRAP and TP values were statistically significantly lower than both groups, and the FRAP2 value was statistically significantly lower than the control group only (Table 2). In light of these results, it can be stated that as the duration of diabetes increases, the depletion of total dietary antioxidant capacity also increases. Additionally, it was found that the increase in TEAC values in newly diagnosed type 2 diabetics led to a decrease in TOC and OSI levels, while in the control group, the Total ORAC values caused an increase in TAC levels, TEAC values caused an increase in PON-1 levels, and L-ORAC values increased ARES levels (Table 3). These results are consistent with results from numerous studies examining the ability of a DTAC-rich diet to regulate serum TAC status with the consumption of tea, coffee, nuts, fruits, and vegetables [47-50].

Limitations

To the best of our knowledge, this study is one of the first to evaluate the effects of DTAC values obtained from different databases on serum oxidative, glycemic, and lipid parameters between newly diagnosed and previously diagnosed type 2 diabetics and a healthy control group, which constitutes a strong aspect of the study. However, the study also has limitations that should be acknowledged. The first is that the cross-sectional design of the study prevents causal inferences, along with the small sample size. Another limitation is that although the study categorized the groups considering the age of diabetes concerning

oxidative mechanisms, the effects of complications were not evaluated.

Conclusion

In conclusion, it was found that the total DTAC values and serum TAC levels of individuals with type 2 diabetes tend to decrease with increasing diabetes age, as well as in comparison to those without diabetes. It was also determined that DTAC values have a significant effect on some oxidative parameters, while serum oxidative parameters do not affect glycemic and lipid parameters. This can be seen as a risk factor in type 2 diabetes, which is associated with increased oxidative stress. In order to provide more comprehensive recommendations on this subject, the effects of DTAC values and oxidative parameters on the complications of type 2 diabetes should be evaluated in a larger sample. However, it is recommended that a sufficient and balanced diet rich in antioxidants be adopted by both individuals with type 2 diabetes and healthy individuals to prevent the development of type 2 diabetes at the community level. Dietary recommendations should be developed in this context to be implemented in public health strategies.

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