



Evaluation of Postprandial Total Antioxidant Activity in Normal and Overweight Individuals

Normal ve Aşırı Kilolu Kişilerde Postprandiya Total Antioksidan Aktivitenin Değerlendirilmesi

Evaluation of Postprandial Total Antioxidant Activity

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Özet

Amaç: Postprandiya deęişiklikler vücutta bazı mekanizmaları akut olarak deęiştirir. Gıda alımı ve besin takviyesi sonrası kan oksidatif durumundaki deęişiklikleri gösteren pek çok çalışma vardır. Bu çalışmanın amacı, normal ve aşırı kilolu bireylerde standardize edilmiş bir yemeğin serum total antioksidan aktivite (TAA) üzerine etkilerini değerlendirmektir. **Gereç ve Yöntem:** Ondört normal kilolu ve oniki kilolu bireye bir gece aç kaldıktan sonra sabah standardize edilmiş bir yemek verildi. Her iki grupta açlık ve yemekten sonra 3. saat ve 6. saatte serum TAA, glukoz, total kolesterol, HDL kolesterol, LDL kolesterol ve trigliserid konsantrasyonları ölçüldü. **Bulgular:** Normal ve aşırı kilolu grupta TAA için bazal ve 3. saat arasındaki fark istatistiksel açıdan anlamlıydı. Aynı zamanda 3. saatte aşırı kilolu grubun TAA'si normal kilolu grubun TAA'inden anlamlı olarak daha düşüktü. Ancak lipid parametreleri ve TAA düzeyleri arasında anlamlı bir ilişki yoktu. **Tartışma:** Bu çalışma postprandiya oksidan stresin normal kilolu kişilere göre aşırı kilolu bireylerde daha belirgin meydana geldiğini göstermektedir. Postprandiya deęişiklikler akut olarak oksidatif stresi indükler ve doğal antioksidan savunma mekanizmasını bozar. Özellikle obez bireylerde çeşitli hastalıklardan ve komplikasyonlardan korunmak için antioksidan gıdaların tüketiminin yararlı olacağını belirtmek gerekir.

Anahtar Kelimeler

Postprandiya Dönem; Total Antioksidan Aktivite; Aşırı Kiloluluk

Abstract

Aim: Postprandial changes acutely alter some mechanisms in body. There are many studies showing blood oxidative status changes after food intake and supplementation. The aim of the present study was to evaluate the effects of a standardized meal on serum total antioxidant activity (TAA) in normal weight and overweight individuals. **Material and Method:** Fourteen normal weight and twelve overweight individuals were given a standardized meal in the morning after an overnight fast. Serum TAA, glucose, total cholesterol, HDL cholesterol, LDL cholesterol and triglyceride concentrations were measured at baseline, 3rd hour and 6th hour after the meal in both groups. **Results:** In both normal and overweight groups, the difference between baseline and 3rd hour was significant for TAA. The TAA of the overweight group was also significantly lower than the TAA of the normal weight group at 3rd hour. However, there was no significant correlation between lipid parameters and TAA levels. **Discussion:** The present study shows that postprandial oxidative stress occurs more prominently in overweight individuals than in normal weight individuals. Postprandial changes acutely induce oxidative stress and impair the natural antioxidant defense mechanism. It should be noted that consuming foods with antioxidants in order to avoid various diseases and complications is useful, particularly in obese subjects.

Keywords

Postprandiya Period; Total Antioxidant Activity; Overweight

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Introduction

Obesity is an important health problem with a significant impact on mortality and morbidity of people of all ages. Furthermore, obesity is considered to be a principal causative factor in the development of metabolic syndrome and has been associated with increased incidence of hypertension, dyslipidemia, hyperinsulinemia, type-2 diabetes and cardiovascular disease [1]. Oxidative stress is caused by an imbalance between the antioxidant and prooxidant processes that occur during metabolism [2]. The net result is accumulation of oxidative products and oxidative damage in tissues. There is now ample evidence indicating the importance of total antioxidant activity (TAA) in plasma and tissues and its modification during oxidative stress development. TAA shows the cumulative action of all the antioxidants and provides an integrated parameter rather than the simple sum of measurable antioxidants [3,4].

Postprandial oxidative stress is characterized by an increased susceptibility of the organism to oxidative damage after consumption of a meal rich in lipids and/or carbohydrates [5,6]. Hyperlipidemia and hyperglycemia have been associated with increased oxidative damage affecting lipoproteins and antioxidant status [7]. Importantly, there is a link between central obesity with increased cytosolic triglyceride stores and the enhanced production of oxygen free radicals in endothelial cells or vascular smooth muscle cells [8]. However, the detailed relations among hyperlipidemia, hyperglycemia, hyperinsulinemia and oxidative stress are still under research. In the studies to date, oxidant/antioxidant balance has been assessed after the consumption of food and supplements which were thought to increase the antioxidant capacity [9-12], but antioxidant activity after a standardized meal has not yet been investigated in overweight individuals.

The aim of the present study was to evaluate and compare the effects of a standardized meal, which may cause acute elevations of plasma glucose and lipid levels, on TAA in normal weight and overweight individuals.

Material and Method

Twenty-six healthy individuals aged 30.9 ± 5.9 years (means \pm SD) were recruited from voluntary participants who were the laboratory staff at our hospital. Written informed consent was obtained from each volunteer and the procedures were in accordance with the guidelines of the Helsinki Declaration on human experimentation. Subjects were assigned to one of two groups on the basis of body mass index (BMI): those of normal weight subjects with a BMI < 25 kg/m² (controls) and overweight subjects with a BMI ≥ 25 kg/m², according to the definition of Bray [13]. In this study, a standardized meal was administered to 14 (6 male, 8 female) normal weight and 12 (6 male, 6 female) overweight individuals. None of the subjects used any medication and antioxidant supplementation or had any diseases. All subjects were nonsmokers.

In all subjects, blood samples were collected at baseline (12 hour fasting) and after the meal (3rd and 6th hours) to measure TAA, which is an indicator of known and unknown antioxidants in plasma. The standardized diet included total energy of 700 kcal (59% carbohydrate, 27% lipid, and 14% protein) was given at 8.00 a.m., after a 12-hour fast. All subjects were at rest in

a seated position throughout the test. Serum total antioxidant activity (TAA), glucose, total cholesterol, HDL cholesterol, LDL cholesterol and triglyceride concentrations were measured at fasting (0th h), 3rd h and 6th h after meal in both the normal weight and overweight groups.

TAA was determined spectrophotometrically (UV-60A, Shimadzu, Kyoto, Japan). A solution of 0.1 mM DPPH (1,1-diphenyl-2-picrylhydrazin) was rapidly mixed with the sample (1/10, v/v). The decline in absorbance was recorded at 517 nm against an ethanol blank at 15th min. The decreases of absorbance corresponding to 100% radical scavenging were determined with a solution of pyrogallol in dimethyl sulfoxide (DMSO) (ca. 0.5%), which caused complete scavenging within seconds. Other measurements were assayed photometrically by clinical chemistry analyzer (AU2700 systems, Beckman Coulter Inc., Brea, CA, USA).

SPSS Statistics 20.0 (Statistical Package for Social Sciences version 20.0, USA) was used for statistical analysis. All data were expressed as median (min-max). The values of independent samples were compared using the Mann Whitney test. The same group's values were compared using the Wilcoxon test. Relationships among variables were obtained using Spearman's correlation coefficient (*r*). P values lower than 0.05 were considered significant.

Results

Subjects' physical and metabolic variables are presented in Table 1. Postprandial changes in the metabolic variables were shown in Table 2. In both the normal and overweight groups, there was a decrease in TAA at the 3rd hour in comparison with the baseline levels ($p=0.024$ and $p=0.021$, respectively). The level of TAA at 3rd hour in overweight individuals was significantly lower than normal weight individuals ($p=0.013$). For 0th and 6th hour measurements, TAA levels in normal weight individuals were higher than overweight individuals, but it was not significant (Figure 1).

Glucose showed a significant decrease at 6th hour in normal and overweight groups ($p=0.022$ and $p=0.016$, respectively). But there was no significant difference in glucose level between the normal and overweight groups at any period. Total cholesterol levels were significantly higher in overweight individuals than in normal weight individuals at all periods. HDL cholesterol level was significantly less in overweight individuals only at the 6th hour ($p=0.045$). In both groups, LDL cholesterol levels were found significantly higher in 3rd and 6th hours compared to baseline. Also, LDL cholesterol levels were significantly higher in overweight individuals than in normal weight individuals at 0th and 3rd hours ($p=0.036$ and $p=0.029$, respectively). Triglyceride levels were significantly higher in overweight individuals than normal weight individuals in all periods and it showed a significant increase at both 3rd and 6th hours compared to baseline in each group.

Table 1. Demographic characteristics of normal weight and overweight groups

	Normal weight group (n = 14) Mean \pm SD	Overweight group (n = 12) Mean \pm SD	p value
Age (Year)	30.8 \pm 5.4	30.9 \pm 6.5	0.940
BMI (kg/m ²)	21.7 \pm 2.3	28.3 \pm 1.1	<0.001

Table 2. Serum TAA, glucose, total cholesterol, HDL cholesterol, LDL cholesterol and triglyceride concentrations (median, minimum, maximum) at 0th h and 3rd h and 6th h after meal of normal weight and overweight groups

	0 th h	3 rd h	6 th h	0 th -3 rd h p value	0 th -6 th h p value
TAA (%)					
Normal weight group	28.0 (13.0-49.0)	22.5 (10.0-30.0)	24.8 (12.5-41.5)	0.024	0.379
Overweight group	22.5 (13.0-40.5)	16.0 (9.0-21.5)	21.3 (14.0-37.0)	0.021	0.477
p value	0.440	0.013	0.236		
Glucose (mg/dL)					
Normal weight group	92 (79-110)	90 (77-115)	86 (79-95)	0.271	0.022
Overweight group	95 (81-116)	93 (81-110)	89 (82-96)	0.308	0.016
p value	0.180	0.164	0.185		
Total cholesterol (mg/dL)					
Normal weight group	169 (117-219)	165 (120-225)	169 (129-199)	0.181	0.054
Overweight group	206 (156-251)	202 (155-259)	201 (162-258)	0.305	0.307
p value	0.006	0.010	0.040		
HDL cholesterol (mg/dL)					
Normal weight group	48 (29-58)	46 (29-56)	47 (28-59)	0.100	0.858
Overweight group	40 (31-60)	39 (28-56)	38 (29-58)	0.137	0.065
p value	0.071	0.066	0.045		
LDL cholesterol (mg/dL)					
Normal weight group	105 (70-132)	94 (61-116)	96 (72-129)	0.001	0.039
Overweight group	130 (80-161)	113 (80-143)	115 (84-145)	0.003	0.004
p value	0.036	0.029	0.082		
Triglyceride (mg/dL)					
Normal weight group	78 (34-150)	137 (42-227)	106 (50-290)	0.002	0.008
Overweight group	153 (45-459)	267 (79-323)	285 (73-420)	0.034	0.034
p value	0.006	0.005	0.009		

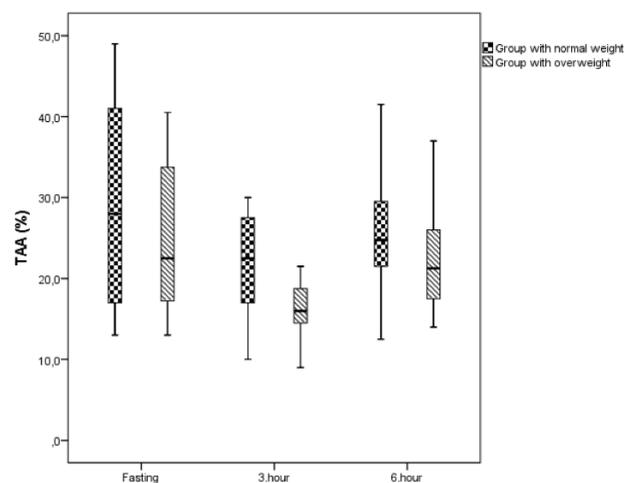


Figure 1. TAA (%) in normal weight and overweight groups according to BMI in different periods

These findings show that plasma TAA levels were elevated to fasting level within 6 hours after the meal. The organism may be exposed to meal-generated oxidative stress at the 3rd hour and the antioxidant activity restored at the 6th hour. This stress occurred more prominently in overweight subjects than in normal weight subjects. However, there was no significant correlation between lipid parameters and TAA levels in the two groups (Table 3).

Table 3. The correlations between other parameters with serum TAA

		Glucose (mg/dL)	Total cholesterol (mg/dL)	HDL cholesterol (mg/dL)	LDL cholesterol (mg/dL)	Triglyceride (mg/dL)
TAA (%) Normal weight group	p value	-0,134	-0,224	-0,053	-0,128	-0,165
	r	0,242	0,050	0,649	0,272	0,149
TAA (%) Overweight group	p value	0,264	0,820	0,524	0,704	0,872
	r	-0,197	-0,040	-0,119	-0,068	0,029

Discussion

Antioxidant molecules that scavenge free-radical species to prevent or delay oxidative damage of important macromolecules, membrane lipids and lipoproteins are prevalent in plasma and other biological fluids. The sum of endogenous and food-derived antioxidants represents the total antioxidant activity of the extracellular fluid. The levels of these antioxidants could also reflect their consumption during acute oxidative stress states [14].

The cooperation among different antioxidants provides a greater protection than any single compound alone [14]. Thus, the overall TAA may give more relevant biological information compared to that obtained by the measurement of individual parameters, because it evaluates the cumulative effect of all antioxidants present in plasma and body fluids [14-15].

Studies have shown that juvenile overweight and obesity are associated with high levels of oxidative stress [16,17]. There are several mechanisms explaining the enhanced oxidative stress observed in obese subjects including altered lipid and glucose metabolism, chronic inflammation, tissue dysfunction, hyperleptinemia and abnormal post-prandial reactive oxygen species (ROS) generation [18,19]. Obese individuals have also increased oxidizability of lipoproteins, low antioxidant defenses, and low levels of antioxidant vitamins (such as vitamins E and A) and other antioxidant substances (such as alpha and beta-carotene, lycopene etc.) [20,21]. Obese individuals may have a lower intake of foods containing antioxidants and phytochemicals, such as fruits, vegetables and legumes. On the other hand, consumption of antioxidant nutrients may be normal in obese individuals, but there is increased utilization of these molecules. Inadequate physical activity may also account for a decreased antioxidant state.

The present study shows that the postprandial TAA of overweight subjects was lower than normal weight subjects, and postprandial changes acutely induced an oxidative stress. The

mechanism of postprandial oxidative stress generation is still unknown. Hyperlipidemia and hyperglycemia have been associated with increased oxidative damage affecting lipoproteins and the antioxidant status [22]. Postprandial increases of lipid and carbohydrate levels lead to increased oxidative stress, which has been associated with increased risk for atherosclerosis and related disorders [23].

The impact of a standardized meal on the antioxidant status of overweight and normal weight subjects by measuring plasma levels of TAA was evaluated in this study. The standardized meal administered in our study included both carbohydrates and lipids. Although the TAA levels for both groups decreased at the 3rd hour, the glucose levels didn't show any significant change. The expected impact of hyperglycemia, which occurs at the 2nd hour, may be reflected at the 3rd hour in TAA. Although there was an increase in triglycerides and LDL cholesterol levels and a decrease in TAA levels at the 3rd hour, there was not a significant correlation between TAA and triglycerides and LDL cholesterol levels. The reason for decreased postprandial TAA is not only hyperlipidemia or hyperglycemia; it can also be the cumulative effect of the metabolic changes occurring after the meal. Kasuya et al. [24] found that the antioxidant capacity continued to decrease until the 3rd hour after the meal in non-obese individuals. Cao et al. [25] found a decrease at the 4th hour after breakfast in serum antioxidant capacity in overweight older women.

It may be assumed that the foods increase free-radical production due to increased oxygen metabolism, thus decreasing serum antioxidant capacity. In these studies it has been seen that antioxidant capacity after a meal is affected in similar periods. These findings support the hypothesis that postprandial changes acutely induce an oxidative stress and impair the natural antioxidant defense mechanism found in the plasma of overweight and normal weight individuals. Postprandial oxidative damage evidently occurred more prominently in overweight individuals than in normal weight individuals. In conclusion postprandial changes must be controlled to prevent various diseases and complications, particularly in overweight subjects. This control may be achieved using various strategies, especially focusing on natural approaches including weight loss, physical activity, diet, dietary supplementation and microbiota modulation rather than pharmacological treatments or surgical intervention.

Competing interests

The authors declare that they have no competing interests.

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