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A Study on the Status of Antioxidant Vitamins and Serum Lipids in Korean Adults

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Abstract

The present study was aimed at investigating whether there are sex-related differences in serum levels of lipids, retinol, α-tocopherol and ascorbic acid in Korean adults. Serum levels of antioxidant vitamins and lipids were determined along with anthropometric measurements in 53 healthy male subjects with mean age 42.7±12.2 years and 44 female subjects with mean age 46.7±10.3 years, from Taegu in Korea. BMI and W/H ratio of the men were 22.66±2.47, 0.88±0.04 and those of the women were 23.36±3.44, 0.84±0.05 respectively. Systolic and diastolic blood pressures were 121.67±14.8, 79.3±12.4 in the men and 123.1±17.5, 78.8±10.9 in the women respectively. Average serum levels of total cholesterol, LDL- and HDL-cholesterol and triglyceride were 165.8±36.4, 102.6±31.2, 41.1±10.5, and 110.2±57.8 mg/dl in the men and 169.1±39.1, 113.7±36.2, 38.1±8.6, and 85.2±37.7 mg/dl in the women respectively. Thirteen percent of men and thirtyeight percent of women had LDL-cholesterol over 130 mg/dl. Serum levels of retinol, α-tocopherol, and ascorbic acid were 43.25±15.51, 17.93±7.07, 115.24±63,25 μg/dl in the men and 31.80±15.39, 17.41±6.12, and 144.99±89.87 μg/dl in the women respectively. Serum vitamin E showed positive correlations with total cholesterol and LDL-cholesterol and triglyceride.

Key words: Korean adults, serum lipids, antioxidant vitamins

INTRODUCTION

Short-and/or long-term disturbance of pro-/antioxidant balance resulting in adverse effects is either due to impaired antioxidation or to favored prooxidation (1). Prooxidant factors are all those contributing to an increased formation of free radicals or other reactive oxygen compounds. Both cellular mechanisms (defects in mitochondrial respiration, specific enzymes) and exogenous factors (smoking with inhalation, drugs) may contribute to this. The main aim in achieving oxidative balance is to reduce the prooxidant factors, especially smoking, and the secondary aim is appropriate intake of antioxidants. Antioxidant factors are on the one hand, endogenous protective systems such as enzymes, specific proteins and thiols such as glutathione and, on the other hand, exogenous substances taken into the body (vitamins E and C, carotenoids, flavonoids, polyphenols and possibly other dietary constituents, and others) and which reduce the effect of prooxidant factors. Whereas the activity of endogenous antioxidant factors (for example enzymes) can be altered only to a limited extent, the activity of exogenous antioxidant systems depends largely on intake.

Both systems ensure the individually necessary protection from oxidative stress. A large number of epidemiological studies (2-8) has shown that high consumption of fruit and vegetables is associated with a lower risk of cancer and cardiovascular diseases. There is at present no causal explanation for this epidemiological finding. However, it is likely that an important part is played by the high content of antioxidant nutrients in many plant-derived foods, especially vitamins C and E, and the carotenoids. Many questions about the mechanism of action and, in particular, the optimal intake of antioxidant nutrients have yet to be answered.

A large number of studies have shown that antioxidant vitamins are able in appropriate concentrations to reduce oxidative damage (3,9). *In vitro* studies have shown both a direct diminution in oxidative damage to membranes or subcellular and extracellular compartments, for example lipoproteins (10), and prevention of secondary effects induced by oxidative change, such as disturbances of cell functions and membrane permeability, and mutagenic effects.

Antioxidant substances reduce the level of oxidative stress. In this way they help to maintain physiological functions of cells. The modes of action of the antioxidant substances are many and various and have not yet been entirely clarified. Although specific mechanisms of action are known for each of the antioxidant nutrients and dietary constituents, their complex interaction is ensured only when they are consumed together in the appropriate ratio. Further research is urgently needed to explain the wide variety of effects and interactions of antioxidant vitamins with one another and with other dietary constituents.

Plasma concentrations which are regarded as a measure of primary prevention in healthy adults essentially presuppose adequate intake, which should to be possible to achieve by diet.

There is a relation between high plasma levels and lower

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morbidity for coronary heart disease and certain neoplastic disorders. It can be assumed that plasma concentration sufficiently reflect the individual status necessary to prevent these disorders: plasma levels 25 to 30 percent below the threshold levels for prevention are associated with a statistical doubling of the risk (3-5).

Currently, the plasma concentrations are mainly used as an indicator of increased risk of disease. However, indicators of a disease-specific oxidative imbalance have yet to be established.

When assessing the intake of vitamin E, it must also be taken into account that determination of the plasma levels by chromatographic methods can show marked differences between laboratories. An additional factor is that the required lipid correction (relation of vitamin E to plasma lipids or cholesterol) is often not carried out. Further studies are therefore necessary to determine the distribution of plasma levels in the population with adequate reliability in order to establish the relation to the intake of vitamin E with greater accuracy.

Statements about the intake necessary to reach the preventive plasma level apply to the majority of healthy adults who are not subject to special oxidative stress (nonsmokers) up to 65 years of age. This can be achieved by a specific diet. There are groups with increased requirements (people with greater stress, smokers, pregnant women, and nursing mothers).

The present study was aimed to investigate whether there are sex-related differences in serum levels of lipids, retinol, α-tocopherol and ascorbic acid in Korean adults and if the differences are held even after adjustment for serum lipid.

MATERIALS AND METHODS

All blood specimens were obtained from blood donors from biannual health examinations held in Kyemyung University Hospital from April 22 to June 23, 1997. Questionnaires on general characteristics of the subjects were provided during the examination. Fasting blood was withdrawn and serum was prepared by centrifugation of blood at 3,000 rpm for 10 minutes and kept frozen at -70°C until analysis.

Serum total cholesterol was determined spectrophotometrically by the enzymatic method (11) using a kit (Asan, Korea). The HDL fraction was separated using the heparin-$MnCl_2$ precipitation method (12), and serum total and HDL-cholesterol were measured using the Asan-set cholesterol kit and serum triglyceride using the Alan-set triglyceride kit.

Retinol and α-tocopherol were determined simultaneously by high pressure liquid chromatography according to the method of Bieri et al. (13). A total lipid extract from 0.1 ml serum containing internal standards of α-tocopheryl acetate and retinyl acetate was injected into a micro-Bondpack C_{18} column (Waters) developed with methanol-water (97 : 3). Vitamins were detected at UV 292 nm.

Measurements of retinol, α-tocopherol, and ascorbic acid were done using an extraction from plasma samples. Methods used were modified from several references.

The retinol standard, retinyl acetate as internal standard, and α-tocopherol standard were dissolved in ethanol.

For retinol and α-tocopherol, standard curves were generated from the quantity of compounds vs. the peak height ratio using the internal standard, retinyl acetate.

Samples were extracted from serum using absolute ethanol and petroleum ether. The absolute ethanol contained ascorbic acid as an antioxidant, and retinyl acetate was used as the internal standard for retinol and α-tocopherol. The samples were shaken and centrifuged, after the addition of extractant. A sample of the petroleum ether layer was measured, dried and stored under nitrogen.

The residue was dissolved in ethanol before injection into HPLC. A portion of each sample was used first, for the measurement of retinol and α-tocopherol; the remaining portion, for the measurement of ascorbic acid.

A Perkin-Elmer HPLC system was used for the first and second steps. The system consisted of a Perkin-Elmer series 3B solvent delivery system, an injector, a stainless steel 3.9 mm × 30 cm micron particle size, a Perkin-Elmer *LC75* Spectrometer detector and a Perkin-Elmer 023 chart recorder.

For the measurements of retinol and α-tocopherol, a portion of the samples were injected into the HPLC system using a Hamilton syringe and then monitored at a wave length of 290 nm using methanol as a eluant. The height of each peak identification was confirmed by comparing plasma peak retention times to those of known standards. To calculate the concentration of unknown samples, peak height ratio with the internal standards were applied for each standard curve for retinol and α-tocopherol.

Participants completed a questionnaire about general characteristics, smoking, drinking, exercise, stress, food habit and nutrient intakes. Cigarette smoking and alcohol drinking habits of the subjects were divided into number of cigarettes smoked per day and frequency of drinking per week. Degree of exercise and stress were divided into three exercise groups (no, light, regular) and five groups of stress (no, light , moderate, severe, and very severe). Anthropometric measurements were carried out measuring weight, height, waist/hip ratio, and triceps skinfold thickness.

All data were entered and stored with the Data Base Management System. The Statistical Package for Social Science (SPSS) was utilized for analysis. For the first step of analysis, frequencies of all variables were computed. All outliers, coding errors and missing values were checked from frequency tables. Several random records were printed and checked against original data.

Two basic statistical methods were used. First, the group comparison and second, examination of different correlation coefficients, were used. The group comparisons were used for comparison among different groups. To examine relationships between the various groups, correlation coefficients were examined.

Group comparisons were done using the Student t-test, and analysis of variance (ANOVA). The Student t-test was used to test for differences in mean between two groups. The results reported as significant were based on a probability less than 0.05.

Pearson's correlation coefficients were computed to examine relations between anthropometric variables, lipid measurements, dietary intake level, and other variables and serum level of retinol, α-tocopherol, and ascorbic acid.

RESULTS AND DISCUSSION

Table 1 shows anthropometric measurements of all subjects. Mean age of the men was 42.7 years and that of the women was 46.7 years. BMI and percent of body fat in men and in women were 22.6, 15.3% and 23.4, 22.6% respectively. Table 2 shows general characteristics of the subjects. The status of cigarette smoking, alcohol drinking and degree of stress were significantly higher in males than in females. Table 3 shows blood pressure, body mass index, W/H ratio and % of fat of the subjects. 48% of men and 37% of women showed BMI over 20. 7.5% of men showed W/H ratio over 0.95, which correlates with high risk for atherosclerosis. The mean levels of lipid for men and women are summarized in Table 4. In men, mean levels of total cholesterol (TC), LDL-cholesterol, HDL-cholesterol and triglyceride (TG) were 165.8±36.4, 102.6 ±31.2, 41.1±10.5, 110.2±57.8 mg/dl respectively. In women, TC, LDL, HDL and TG were 169.1±39.1, 113.7±36.2, 38.1± 8.6, 85.2±37.7 mg/dl respectively.

Mean serum triglyceride of men was significantly higher than that of women.

In Table 4, serum cholesterol and triglyceride levels of the subjects are presented according to the risk group for atherosclerosis (14). 85% of men and 73% of women were normal; serum TC level below 200 mg/dl.

When comparing LDL-cholesterol level, 6% of men and 11% of women were in the high risk group, LDL level over 160 mg/dl. Serum triglyceride level in 11% of men were in the high risk group.

As for the protective role of HDL to atherosclerosis, several lines of evidence have suggested a role of HDL in the protection of LDL against oxidative modification. LCAT reaction can be inhibited by the presence of oxidized LDL in plasma, which inpairs HDL-mediated reverse cholesterol transport (15). HDL also has been shown to be an effective scavenger of superoxide *in vitro* (16), possibly indicating a direct effect of HDL as a reactive oxygen scavenger. Recent experimental evidence suggests that products of LDL oxidation can transfer from minimally oxidized LDL to HDL (17,18). Thus, it has been proposed that by accepting and transporting such oxidation product, HDL may protect against some of the atherogenic effects of oxidized LDL. Other workers have reported that HDL is the principal carrier of lipid hydroperoxides, and oxidized choles-

Table 1. Anthropometric indices of the subjects

Variables	Male (N=53)	Female (N=44)	P-value
Age (years)	42.7±12.2	46.7±10.3	0.085
Height (cm)	169.3± 5.8	156.3± 5.0	0.000
Weight (kg)	65.0± 8.4	57.2± 9.5	0.000
SBP (mmHg)	121.7±14.8	123.1±17.5	0.669
DBP (mmHg)	79.3±12.4	78.8±10.9	0.819
BMI (kg/m^2)	22.6± 2.5	23.4± 3.4	0.250
% of fat	15.3± 4.1	22.6± 5.4	0.000
W/H ratio	0.9± 0.0	0.8± 0.1	0.000

SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, BMI: Body Mass Index, W/H ratio: Waist/Hip ratio

Table 2. General characteristics of the subjects

	Male % (N=53)	Female % (N=44)	
Smoking (cigarettes/day)			
No	39.6 (21)	93.2 (41)	χ^2=31.658
<10	18.9 (10)	6.8 (3)	P=0.000
10~19	22.6 (12)	0	
≥20	18.9 (10)	0	
Drinking (times/week)			
No	30.2 (16)	93.2 (41)	χ^2=39.579
1~2	32.1 (17)	4.5 (2)	P=0.000
3~4	26.4 (14)	2.3 (1)	
≥5	11.3 (6)	0	
Exercise			
No	56.6 (30)	56.8 (25)	χ^2=3.247
Light	32.1 (17)	40.9 (18)	P=0.198
Regular	11.3 (6)	2.3 (1)	
Stress			
No	1.9 (1)	2.3 (1)	
Light	7.6 (4)	63.6 (28)	χ^2=38.208
Moderate	35.8 (19)	22.7 (10)	P=0.000
Severe	41.5 (22)	11.4 (5)	
Very severe	13.2 (7)	0	

Table 3. Body mass index, waist/hip ratios and % of fat of the subjects % (N)

Body mass index	<20	20~25	25~30	30>	
	M 11.3% (6)	M 67.9% (36)	M 20.8% (11)	M 0	
	F 15.9% (7)	F 56.8% (25)	F 25.0% (11)	F 2.3% (1)	
Waist/Hip ratio	<0.88	0.89~0.94	0.95≥		
	M 58.5% (31)	M 34.0% (18)	M 7.5% (4)		
	F 81.8% (36)	F 18.2% (8)	F 0		
% of fat	≤8	9~15	16~19	20~23	24~29
	M 3.8% (20)	M 47.1% (25)	M 34.0% (18)	M 13.2% (7)	M 1.9% (1)
	F 2.3% (1)	F 2.3% (1)	F 22.7% (10)	F 31.8% (14)	F 40.9% (18)

Table 4. Serum cholesterol and triglyceride levels of the subjects

Total cholesterol (mg/dl)	<200	200~239	≥240
	M 45 (84.9)[1)]	6 (11.3)	2 (3.8)
	F 32 (72.7)	11 (25.0)	1 (2.3)
HDL-cholesterol (mg/dl)	≥40	35~39	35
	M 25 (47.2)	11 (20.8)	17 (32.1)
	F 18 (40.9)	12 (27.3)	14 (31.8)
LDL-cholesterol (mg/dl)	<130	130~159	≥160
	M 46 (86.8)	4 (7.5)	3 (5.7)
	F 30 (68.2)	9 (20.5)	5 (11.4)
Triglyceride (mg/dl)	<160	160~209	≥210
	M 45 (84.9)	2 (3.8)	6 (11.3)
	F 41 (93.2)	3 (6.8)	0

[1)]Subjects number (%)

teryl esters in plasma HDL are taken up to a greater extent by liver cells than unoxidized cholesteryl esters (19).

In Table 5, mean serum retinol, α-tocopherol and ascorbic acid for men and women are presented. In men, mean serum retinol, α-tocopherol and ascorbic acid were 43.25±15.51 μg/dl, 17.93±7.07 μg/ml, 115.2±63.2 μg/ml respectively. In women, mean serum retinol, α-tocopherol and ascorbic acid were 31.80 ±15.39 μg/dl, 17.41±6.12 μg/ml, 144.99±89.87 μg/dl.

There is increasing evidence that initiation of atherosclerosis is related to lipid peroxidation and oxidative modification of LDL (20-23). The propagated oxidation process includes breakdown of essential memberane lipids, and peroxidation of unsaturated fatty acids derived from triglycerides, phospholipids and cholesterol in lipoproteins (23). The resistance of lipoproteins to oxidations is partly determined by their content of antioxidants. Alpha-tocopherol is the most active compound of vitamin E (24). Plasma level of vitamin E is the strongest predictive factor for cross-cultural mortality from ischemic heart disease among all essential antioxidants, including vitamin A, C, E, carotenoid and selenium (25). Vitamin E also is the major antioxidant in LDL and is among the first antioxidant to be consumed during LDL-oxidation (26,27).

Table 5. Biochemical measurements of the subjects

Variables	Male (N=53)	Female (N=44)	P-value
Total cholesterol (mg/dl)	165.80±36.40	169.10±39.10	0.671
HDL-cholesterol (mg/dl)	102.60±31.20	113.70±36.20	0.109
LDL-cholesterol (mg/dl)	41.10±10.50	38.10± 8.60	0.145
Triglyceride (mg/dl)	110.20±57.80	85.20±37.70	0.016
Atherogenic index	3.15± 1.16	3.59± 1.38	0.088
Vitamin A (μg/dl)	43.25±15.51	31.80±15.39	0.000
Vitamin E (μg/ml)	17.93± 7.07	17.41± 6.12	0.704
Vitamin C (μg/ml)	115.24±63.25	144.99±89.87	0.059

Values are mean±SD.

Serum retinol concentrations of women were significantly lower than that of men (31.8 vs 43.3 μg/dl). Serum α-tocopherol concentration did not differ between men and women (17.9 vs 17.4 μg/ml). Serum vitamin A concentrations ≥30 μg/dl are considered indicative of adequate vitamin A status in the US population (28). Serum retinol levels reflect vitamin A status only when liver vitamin A stores are severely depleted (below 20 μg/g liver) or excessively high (above 300 μg/g liver). When liver vitamin A concentrations are between these limits, serum retinol concentrations are homeostatically controlled and remain relatively constant.

Mean serum ascorbic acid level of women were slightly higher than that of men (144.9 vs 115.2 μg/dl). According to the work of Stryker et al. (29) men had 19% higher plasma retinol level than women. Higher levels of lipid-adjusted serum vitamin A in males than females and higher levels of serum carotene in females than in males have been reported in a study done by Comstock et al. (30). On the other hand, Herbeth et al. (31) reported that sex differences in serum levels of vitamin A and E became insignificant after adjustment for alcohol and cigarette smoking. We did not find significant differences in serum vitamin A, α-tocopherol and ascorbic acid between smokers and nonsmokers. We did not find significant differences in serum vitamin A, α-tocopherol and ascorbic acid among the drinking groups (Table 6, Table 7). Choi et al. (32) noticed that α-tocopherol adjusted for triglyceride was lower in heavy smokers than in moderate smokers, and α-tocopherol was higher in subjects who drank more than 5 times per week (32). Stryker et al. (29) noticed that plasma α-tocopherol was positively associated with daily alcohol intake in women but not in men. The findings on the relationship between smoking and serum levels of antioxidant vitamins have not always been consistent (33). Serum ascorbic acid concentrations are the most frequently used and practical index of vitamin C status in humans. Levels are influenced by any re-

Table 6. Comparison of the results according to smoking habits in the subjects

Smoking (cigarettes/day)	T-chol (mg/dl)	HDL-chol (mg/dl)	LDL-chol (mg/dl)	AI	TG (mg/dl)	sVtA (μg/dl)	sVtE (mg/L)	sVtC (μg/dl)	SBP (mmHg)	DBP (mmHg)	sVtE/TC (%)	sVtE/TG (%)
Male												
<10(31)[1)]	166.93 ±41.53	39.57 ±10.31	105.03 ±34.17	3.28 ±1.31	111.23 ±56.92	43.57 ±15.92	18.25 ±7.82	123.43 ±64.15	121.71 ±16.27	79.26 ±14.21	0.11 ±0.05	0.19 ±0.09
≥10(22)	164.21 ±28.60	43.15 ±10.68	99.23 ±26.92	2.97 ±0.94	108.81 ±60.38	42.80 ±15.28	17.48 ±6.02	103.71 ±61.57	121.59 ±12.94	79.41 ±9.71	0.11 ±0.03	0.18 ±0.06
P-value	0.792	0.226	0.510	0.347	0.882	0.859	0.700	0.267	0.977	0.966	0.664	0.621

[1)]Numbers of cigarettes smoked (Numbers of subjects)

T-chol: total cholesterol, HDL-chol: HDL-cholesterol, LDL-chol: LDL-cholesterol, AI: atherogenic index, sVtA: serum vitamin A, sVtE: serum vitamin E, sVtC: serum vitamin C, SBP: systolic blood pressure, DBP: diastolic blood pressure, sVtE/TC: serum vitamin E/total cholesterol, sVtE/TG: serum vitamin E/triglyceride

Table 7. Comparison of the results according to drinking habits in the subjects

Drinking (Numbers/week)	T-chol (ml/dl)	HDL-chol (ml/dl)	LDL-chol (ml/dl)	AI	TG (ml/dl)	sVtA (μg/dl)	sVtE (mg/L)	sVtC (μg/dl)
Male								
No (16)[1)]	150.69±26.64	37.96± 9.99	93.55±22.95	3.19±1.16	95.50±56.19	38.66±15.44	17.11±9.21	134.05±72.51
1~2 (17)	176.64±40.98	44.02±10.91	110.19±37.79	2.97±1.20	111.77±34.42	44.19±15.97	18.09±5.34	107.26±43.93
3~4 (14)	169.07±34.47	40.81± 9.40	103.27±30.23	3.33±1.33	124.57±74.84	47.69±16.72	18.59±7.64	125.81±73.85
≥5 (6)	167.73±45.21	41.50±13.40	103.85±34.34	3.15±0.90	111.65±75.21	42.45±10.83	17.97±4.43	63.07±23.55
P-value	0.225	0.442	0.512	0.873	0.602	0.463	0.960	0.101

[1)]Frequency of drinking/week (Numbers of subjects)
T-chol: total cholesterol, HDL-chol: HDL-cholesterol, LDL-chol: LDL-cholesterol, AI: atherogenic index, sVtA: serum vitamin A, sVtE: serum vitamin E, sVtC: serum vitamin C

cent intake of the vitamin, especially when intakes are high, making fasting blood samples essential. Serum ascorbic acid concentrations increase with dietary intakes until a serum concentration of approximately 1.4 mg/dl is reached, but serum concentrations rarely exceed this threshold concentration of 1.4 mg/dl despite very large doses, because the renal clearance of the vitamin rises sharply with daily intakes greater than 100 mg. In the present study, serum level of ascorbic acid in males was lower than that of Choi et al.'s study (33).

An important aspect of biochemical assessment of populations is the establishment of reference intervals for the vitamins. Because of the lack of a standardized methodology, it is not surprising that there has been some disagreement in regard to normal values. It is easy to overlook the fact that reference intervals not only are dependent on the metholology chosen but are also influenced by preanalytical as well as analytical sources of variation. Preanalytical sources of variation evolve from secular, clinical, and intrinsic differences within the population being examined and from blood sample collection and handling techniques. Analytical sources of error relate primarily to the accuracy and the precision performance of the laboratory.

Secular sources of variation result from differences in lifestyle and dietary practices within the study population, such as alcohol consumption, smoking habits, and physical activity. Clinical sources of variation include those resulting from use of medications by subjects and from metabolic states altered by acute or chronic illness.

Blood sampling and handling techniques, such as posture during sample collection, differences in capillary-venous samples, use of anticoagulants, and fasting state of subjects, all should be controlled as much as possible to reduce variation from these soures. Sample storage conditions also must be carefully monitored if analyses are delayed for any period of time.

Finally, analytical sources of variation depend on the accuracy and precision performance of the laboratory. Poor accuracy and precision may result from the absence of an acceptable quality-assurance program. One factor affecting analytical error is the nature of the substance being analyzed. For example, if serum ascorbic acid levels are to be measured by the dichlorophenolindophenol method, the sample must be treated soon after collection. Ascorbic acid is very labile and is separated from whole blood. If the plasma is not treated within 10 min with m-phosphoric acid, the results obtained will be biased on the low side. It is also imperative that individuals be in a fasting state (≥12 h) in order for serum ascorbic acid levels to be free of influence from recent intakes of vitamin C. After a meal containing ascorbic acid, blood levels of ascorbic acid peak at ~3 h and can remain elevated ≤12 h before returning to a steady-state level (34). If this tendency is not realized, values from analyses may be spuriously high and, therefore, misleading.

The total of these preanalytical and analytical sources of variation accounts for the range of laboratory results observed in any population study (35).

Vitamin supplementation is another important factor affecting serum vitamin levels (36). Unfortunately, we do not have reliable information on vitamin supplementation. Therefore, further study should be carried on serum levels of antioxidant vitamins including vitamin supplementation and lifestyles. But we should know that an increase in the antioxidant vitamin intake cannot, however, compensate for unhealthy lifestyles. Moreover, an increase in the intake of antioxidants is worthwhile only if the oxidative balance cannot be restored in spite of reducing prooxidant factors.

Table 8 shows overall associations of retinol and α-tocopherol with total cholesterol, LDL-cholesterol and triglyceride. α-tocopherol was positively correlated with total cholesterol both in males and females. Retinol was positively correlated with triglyceride both in males and females. α-Tocopherol in males was positively correlated with LDL-tocopherol. α-Tocopherol in females was positively correlated with trigly-

Table 8. Correlations of serum retinol and α-tocopherol by serum total cholesterol and triglyceride

	Male		Female	
	Retinol	α-Tocopherol	Retinol	α-Tocopherol
Total-cholesterol	0.200	0.411**	-0.072	0.303*
LDL-cholesterol	-0.032	0.273*	-0.194	0.238
Triglyceride	0.530**	0.623**	0.576**	0.442**
Vt. E/TC	0.406**	0.783**	0.326*	0.762**
Vt. E/TG	-0.177	0.151	-0.252	0.327*

*p<0.05, **p<0.01
TC: total cholesterol, TG: triglyceride

ceride. In this study we found a strong correlation of serum retinol and α-tocopherol with triglyceride in men and women ($p<0.01$).

As shown in Table 9, cholesterol-adjusted levels of retinol (0.27 vs 0.2 μg/ml, $p<0.01$) were significantly higher in males than in females. Triglyceride-adjusted level of α-tocopherol (18.70 vs 23.18 μg/ml, $p<0.05$) were significantly higher in females than in males. Total lipid-adjusted level of retinol (82.19 vs 54.64 μg/ml, $p<0.01$) were significantly higher in males than in females.

Table 10 and 11 show the correlation coefficients in male and female subjects. As shown in Table 10 and 11, the status of vitamin C and AI (atherogenic index) in males was significantly correlated at $p<0.05$. The status of vitamin E and HDL in females was significantly correlated at $p<0.01$. And the status of vitamin C and AI and triglyceride in females were significantly correlated at $p<0.05$.

Table 9. Sex-related differences in serum retinol and α-tocopherol without and with adjustment for the level of serum total cholesterol

	Male	Female	P-value
Unadjusted levels			
Retinol	43.25±15.51	31.80±15.39	0.000
α-Tocopherol	17.93± 7.07	17.41± 6.12	0.704
Cholesterol-adjusted levels			
Retinol/TC	0.27± 0.11	0.2 ± 0.12	0.005
α-Tocopherol/TC	10.98± 4.17	10.61± 3.76	0.652
Triglyceride-adjusted levels			
Retinol/TG	0.46± 0.22	0.41± 0.18	0.212
α-Tocopherol/TG	18.7 ± 7.99	23.18±10.09	0.016
Total lipid-adjusted levels			
Retinol/TL	82.19±40.45	54.64±26.05	0.01
α-Tocopherol/TL	3.2 ± 1.64	2.95± 1.06	0.456

Mean±SD, NS: not significant, TC: total cholseterol, TG: triglyceride, TL: total lipid

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Table 10. Correlation coefficients in male subjects

	Age	Wt	BMI	Body Fat	WHR	SBP	DBP	TC	LDL	HDL	AI	TG	sVtA	sVtE	sVtC	sVtE/TG	sVtE/TC
Age																	
Wt	-.031																
BMI	.174	.855**															
Body Fat	-.239	.547**	.502**														
WHR	.529**	.334*	.597**	.141													
SBP	.287*	.288*	.440**	.081	.314*												
DBP	.311*	.270	.396**	-.016	.323*	.861**											
TC	-.013	.138	.215	.190	.173	.475**	.354**										
LDL	.032	.123	.168	.139	.106	.445**	.333*	.919**									
HDL	-.271*	-.116	-.138	.135	-.269	-.089	-.092	.348*	.120								
AI	.206	.294*	.343*	.117	.361**	.382**	.288*	.485**	.585**	.545**							
TG	.119	.209	.350*	.101	.503**	.375**	.302*	.352**	.087	-.138	.444**						
sVtA	.035	-.017	.114	.196	.313*	.283*	.226	.200	-.032	.204	.038	.530**					
sVtE	.377**	.334*	.461**	.171	.575**	.421**	.447**	.411**	.273*	-.072	.460**	.623**	.516**				
sVtC	-.023	-.213	-.234	-.006	-.201	-.108	-.013	-.065	-.018	-.044	.043	-.117	.183	-.004			
sVtE/TG	.235	-.028	-.079	.034	-.116	-.164	-.102	-.071	.075	.188	-.173	-.599**	-.177	.151	.073		
sVtE/TC	.458**	.207	.300*	.040	.513**	.102	.192	-.201	-.284*	-.278*	.160	.387**	.406**	.783**	-.030	.269	

BMI: Body Mass Index, WH: Waist/Hip ratio, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, TC: total cholesterol, LDL: LDL-cholesterol, HDL: HDL-cholesterol, AI: atherogenic index, TG: triglyceride, sVtA: Serum Vitamin A, sVtE: Serum Vitamin E, sVtC: Serum Vitamin C, sVtE/TC: Serum Vitamin E/total cholesterol, sVtE/TG: Serum Vitamin E/triglyceride *$p<0.05$, **$p<0.001$

Table 11. Correlation coefficients in female subjects

	Age	Wt	BMI	Body Fat	WHR	SBP	DBP	TC	LDL	HDL	AI	TG	sVtA	sVtE	sVtC	sVtE/TG	sVtE/TC
Age																	
Wt	.300*																
BMI	.406**	.925**															
Body Fat	.212	.811**	.768**														
WHR	.619**	.586*	.685**	.498**													
SBP	.385**	.281	.344*	.223	.286												
DBP	.292	.353*	.423**	.256	.252	.802**											
TC	.390**	.198	.247	.104	.206	.206	.229										
LDL	.416**	.188	.242	.106	.229	.148	.185	.959**									
HDL	-.097	-.044	-.049	-.039	-.119	.063	.004	.415**	.218								
AI	.430**	.291	.311*	.229	.342*	.114	.175	.530**	.673**	.497**							
TG	.162	.216	.216	.119	.133	.296	.321*	.149	-.039	-.023	.110						
sVtA	.169	.072	.092	.035	.032	.166	.182	-.072	-.194	-.018	-.056	.576**					
sVtE	.202	.242	.273	.209	.272	.118	.195	.303*	.238	.010	.310*	.442**	.326*				
sVtC	.172	-.179	-.118	-.115	-.117	-.081	.085	-.111	-.068	-.219	.048	.017	-.036	.020			
sVtE/TG	-.005	-.004	.026	.0144	.073	-.215	-.192	.098	.213	.107	.069	-.620**	-.252	.327*	.069		
sVtE/TC	-.012	.092	.0112	.126	.174	.029	.080	-.347*	-.378*	-.260	-.054	.310*	.326*	.762**	.175	.284	

BMI: Body Mass Index, WH: Waist/Hip ratio, SBP: Systolic Blood Pressure, DBP: Diastolic Blood Pressure, TC: total cholesterol, LDL: LDL-cholesterol, HDL: HDL-cholesterol, AI: atherogenic index, TG: triglyceride, sVtA: Serum Vitamin A, sVtE: Serum Vitamin E, sVtC: Serum Vitamin C, sVtE/TC: Serum Vitamin E/total cholesterol, sVtE/TG: Serum Vitamin E/triglyceride *$p<0.05$, **$p<0.001$

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