Original Article

The relationships between anthropometric measurements, serum vitamin A and E concentrations and lipid profiles in overweight and obese subjects

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The weight, height, body mass index (BMI), waist/hip ratio, serum retinol and α -tocopherol and lipid profiles of 16 overweight (BMI \geq 25.0 kg/m²) Thai males and 56 overweight females, compared with 14 males and 58 females in a control group (BMI 18.5-24.9 kg/m²), were investigated. Subjects for the study were those persons who turned up regularly for physical check-up at the Outpatient Department, General Practice Section of Rajvithi Hospital, Bangkok. The study was conducted between December 2000-March 2001. Higher levels of cholesterol, LDL-C, LDL-C/HDL-C ratio were found in the overweight compared with the control subjects. Statistically significant higher triglyceride levels were found in the overweight compared with the control subjects. The median serum retinol concentration in overweight subjects was 2.80 µmol/L (range 0.53-4.62 µmol/L) compared with 2.97 µmol/L (range 1.21-4.12 μ mol/L) in control subjects (p=0.0736). The median serum α -tocopherol concentration in overweight subjects was 17.30 µmol/L (range 6.29-28.65 µmol/L) compared with 18.75 µmol/L (range 5.30-30.28 µmol/L) in control subjects (P < 0.05). The median values of retinol and α -tocopherol serum concentrations in the overweight and obese males were lower than those of the overweight and obese females. A total of 6.3% (1 out of 16) and 12.5% (2 out of 16) of the overweight/obese males had decreased retinol and α -tocopherol levels, while the overweight/obese females had decreased retinol and α -tocopherol level of 1.8% (1 out of 56) and 10.7% (6 out of 56), respectively. A total of 12.5% and 39.3% of the overweight/obese males and females had cholesterol concentrations of ≥ 6.48 mmol/l. However, the prevalence of low HDL-C (HDL-C ≤ 0.91 mmol/l) was found to be 50% in the overweight and obese males and 10.7% in the overweight and obese females. Statistically significant associations were found between age, cholesterol, LDL-C, and serum α -tocopherol in the overweight and obese male and female subjects. A negative correlation was found between weight, BMI, AC, MAMC, hip circumference and serum retinol in both the overweight and obese subjects. A negative correlation was found between weight, BMI, MAMC, waist, hip circumferences and serum α tocopherol in both the overweight and obese subjects.

Key Words: anthropometric measurements, serum retinol, serum a-tocopherol, lipid profiles, obesity

Introduction

Obesity is a complex syndrome with a multifactorial origin and may be explained by monogenic mutations, but in most cases it appears as a polygenic condition, which may be affected by a myriad of environmental influences.¹ Obesity currently affects approximately one-third of middle aged Americans.² The fact that Thailand is rapidly approaching the status of an industrialised country is well reflected in some demographic and economic indicators.³ A total of 23.6% of Thai female construction site workers were reported to be obese.⁴ Obesity is found in 11% of Thai elderly.⁵ Moderate to severe obesity is increasingly found and might be associated with clear health risks, including hypertension, diabetes and dyslipidaemia.⁶ Diseases related to obesity, such as hypertension and diabetes mellitus, have increased in rural Thai people.⁷ Physical signs of obesity are hypertension, coronary heart disease, predisposition to diabetes, hyperlipidaemia,

Correspondence address: Dr Duangkamol Viroonudomphol, Department of Tropical Nutrition and Food Science, Faculty of Tropical Medicine, Mahidol University, 420/6 Rajvithi Road, Rajthevee, Bangkok 10400, Thailand. Tel: 66 2 248 5748; Fax: 66 2 248 5748 Email: v_duangkamol@hotmail.com Accepted 11 June 2002 metabolic abnormalities, increased risk of gallbladder disease, gout, some types of cancer, and development of osteoarthritis of the weight-bearing joints. Age, gender and race are modifiers of obesity.

It has been recently suggested that oxidative stress induced by reactive oxygen species (ROS) are capable of reacting with unsaturated lipids and of initiating chain reactions of lipid peroxidation in the membrane. In the last decade, evidence has accumulated that a crucial and causative role in the pathogenesis of atherosclerosis (a physical sign of obesity) is played by the free radical process known as lipid peroxidation. It is currently believed that lipid peroxidation is involved in the oxidative modification of low density lipoprotein. This ultimately results in the formation of atherosclerotic lesions. This hypothesis is supported by data coming from biochemical studies, experimental animal studies and from epidemiological investigations.⁸⁻¹²

It may also be very significant that oxidized low density lipoprotein contains highly cytotoxic lipid peroxidation products such as certain aldehydes, which can be considered diffusible toxins. The LDL particle contains a total of approximately 2200 cholesterol molecules (free and esterified) and the total number of fatty acids bound in the different lipid classes is on average 2600, roughly half of which are polyunsaturated fatty acids (PUFA).

LDL is, therefore, not only rich in cholesterol but also in PUFA known to be highly susceptible to lipid peroxidation, which are protected by the presence of several antioxidants.^{13,14} In particular vitamins E, C and carotenoids, are capable of protecting against oxidative damage to cell and tissue. The major antioxidant is α -tocopherol; on average about seven molecules of this antioxidant are present in each LDL particle. Other potential antioxidants in LDL are γ tocopherol, β -carotene, α -carotene, lycopene, cryptoxanthine, cantaxanthin, lutein, zeaxanthin, phytofluene, retinoids and ubiquinol-10. However, these are present in amounts 20-300 fold lower than α -tocopherol.⁹

Interim analysis of 16 of 33 populations constituting the World Health Organization/ Monitoring of Trends and Determinations in Cardiovascular Disease core study suggested a strong inverse cross-cultural correlation between plasma lipid-standardized vitamin E concentrations and coronary artery disease (CAD) mortality.¹⁰

The aim of this study was to examine the association between anthropometric measurements, antioxidant vitamins and lipid profiles.

Materials and methods

Study population

The study population comprised 16 male and 56 female (total = 72) overweight and obese Thai volunteers (BMI \ge 25.0 kg/m²) and 14 male and 58 female (total = 72) normal (BMI=18.5-24.9 kg/m²) Thai volunteers who attended the Outpatient Department, General Practice Section, Rajvithi Hospital, Bangkok, between December 2000-March 2001. The individuals investigated generally belonged to the middle

class, attending a health check-up facility in the urban centre of Bangkok. The volunteers with diabetes mellitus, anaemia, liver, lung, kidney, hypertension and CAD diseases were excluded from further analysis as they were found to have abnormal biochemical parameters. None of the subjects had any major complaints of ill health and, judging from their appearance and general check up they seemed to be healthy. The check up included x-ray diagnostics, electrocardiogram, temperature and blood deter-minations. Overweight and normal subjects were matched for age and sex. Informed consent was obtained from the subjects before their blood specimens were drawn. The age, marital status, place of origin, drinking and smoking habits were assessed through standardized questionnaires. Physical examinations were conducted by the same medical doctor throughout the study.

Analytical methods

The nutritional status of all subjects investigated was assessed by means of anthropometric measurements. The body weight of each individual dressed in light clothing was measured using a carefully calibrated beam balance (Detecto®). The height of each individual was measured using a verticalmeasuring rod. The BMI or Quetelet Index was conventionally calculated as weight in kg/height (in meters²). The classifications of BMI employed were those used by the WHO Expert Committee 1995:¹⁵

Overweight grade I BMI = 25.00-29.99; Obese grade II: BMI = 30.00-39.99; Obese grade III: BMI \ge 40 kg/m².

Waist and hip circumferences were also measured to calculate waist/hip ratio; normal value for female <0.77, male <0.90.^{16,17} Mid arm muscle circumference (MAMC) was calculated by arm circumference (AC) – [0.3412 × tricep skinfolds thickness (TSF)]. In the morning, about 15 ml of venous blood was drawn from the study subjects, after their overnight fast. The serum samples were separated and stored at 2-5°C for not more than 24 hours prior to lipid profile determination. A serum aliquot was stored frozen at -70°C for serum retinol and α -tocopherol then analyzed within 1 month of collection to ensure the stability of the compounds.

Laboratory techniques

A commercially available Boehringer Mannheim (Germany) test kit was used to determine cholesterol, high density lipoprotein-cholesterol (HDL-C), low density lipoprotein-cholesterol (LDL-C) and triglycerides (TG). The values of \geq 5.18 mmol/l and 6.48 mmol/l of cholesterol, \geq 3.98 mmol/l of LDL-C, \leq 0.91 mmol/l of HDL-C and \geq 2.26 mmol/l of TG were taken as cut-off points. Serum retinol and α -tocopherol were extracted and determined by using a reverse-phase high pressure liquid chromatography (HPLC) apparatus. HPLC analysis was carried out as described elsewhere.²⁰⁻²⁴ Briefly, the chromatographic system consisted of Aphasil column (250×4.6mm). The mobile phase was methanol:water, 95:5 (by vol). Detection of compounds was carried out using a UV detector (Model 486, Waters Associates, Melford, MA, USA) set at 290 nm.

Statistical analysis

Standard statistical methods provided by the Minitab statistical computer programme were used to analyze the data.²³ The median, range and 95% confidence interval (CI) were calculated. The Mann Whitney U-Wilcoxon Rank Sum W test (two tailed) was used to calculate statistical differences between groups.

Results

Median, range and 95% confidence interval (CI) of age, anthropometric variables, serum retinol, α -tocopherol concentration and lipid profiles in overweight and control subjects are shown in Table 1. All of the anthropometric variables, except the height of the overweight group, were significantly higher than those of the normal subjects. Statistically higher levels of LDL-C/HDL-C ratio and triglycerides were observed in the overweight than in the control subjects, whereas HDL-C and serum α -tocopherol were lower in the overweight/obese than in the control subjects. The medians of retinol and α -tocopherol did not differ significantly between those of the overweight/obese females. Because of the relations between vitamin E and cholesterol and triglycerols in serum and LDL, the results for α -tocopherol were also lipid-adjusted [vitamin E/(cholesterol + triglycerides), µmol/mmol].^{27,28} Lipid adjusted and serum vitamin E concentrations were significantly lower in the overweight/obese group than the control group in both genders (Tables 1 and 2).

Table 1. The medians, range and 95% CI of age,	anthropometric variables	s, lipid profiles, retinol	l and α -tocopherol in overweight
and control subjects between male and female			

Parameter		М	ale		Female					
Overweight (N=16)		t (N=16)	Control (N=14)		<i>P</i> -value	Overweight (N=56)		Control (N=58)		<i>P</i> -value ^a
	Median (range)	95%CI	Median (range)	95%CI		Median (range)	95%CI	Median (range)	95%CI	
Age	35.5	25.76-40.24	41.0	39.0-46.0	0.0810	42	39.67-46.00	40	36.05-45.00	0.1265
Weight	(21.0-49.0) 84.75 (67.2-127.0)	76.0-98.4	(25.0-53.0) 62.15 (52.0-72.0)	57.47-68.34	0.0000	(20.0-60.0) 73 (55.8-153.0)	68.3-74.66	(16.0-60.0) 52.5 (42.5-78.0)	51.21-54.29	0.0000
Height	1.66	1.64-1.74	1.68	1.63-1.78	0.8353	1.55	1.55-1.57	1.56	1.54-1.57	0.9977
BMI	(1.51-1.86) 30.78 (25.12-39.98)	28.56-34.18	(1.57-1.80) 22.3 (18.52-24.5)	19.89-24.05	0.0000	(1.47-1.70) 29.72 (25.08-56.00)	28.89-31.00	(1.43-1.78) 21.85 (18.52-24.97)	21.39-22.67	0.0000
SST	28.40	19.33-34.26	17.70	12.55-22.16	0.0050	31.65	28.46-34.26	20.0	16.72-21.30	0.0000
AC	33.5	32.76-35.24	(24.5, 27.5) 28.0	25.84-28.37	0.0000	33.5	33.0-34.50	27.0	26.00-27.17	0.0000
TSF	(28.0-40.0) 23.15 (13.7-39.1)	16.56-31.50	(24.3-29.3) 17.0 (9.9-27.9)	12.18-21.99	0.0540	(27.0-49.0) 29.35 (20.4-50.0)	26.47-31.26	(22.1-33.3) 23.30 (9.9-32.10)	21.92-25.09	0.0000
MAMC	27.37 (19.73-30.47)	23.83-28.20	22.14 (17.43-25.89)	20.83-24.31	0.0015	23.82 (17.29-36.99)	23.28-25.12	19.52 (14.17-24.68)	18.78-19.99	0.0000
Waist	98.5	92.00-106.9	82.0 82.0	77.05-83.00	0.0000	88.5 ((0.0.142.0)	86.00-91.80	72.0	70.05-74.95	0.0000
Hip	(87.0-119.0) 108.5 (91.0-129.0)	99.90-115.3	(65.0-94.0) 91.5 (85.0-103.0)	90.00-97.16	0.0005	(68.0-142.0) 107.0 (87.0-169.0)	104.2-109.0	(61.0-99.0) 92.0 (82 0-109 0)	90.05-93.00	0.0000
W/H ratio	0.926 (0.834-0.982)	0.906-0.950	0.879	0.819-0.907	0.0025	0.822	0.810-0.842	0.780	0.766-0.798	0.0003
Cholest	(0.034-0.982) 5.47 (2.22.7.21)	4.20-5.79	(0.705-0.940) 5.96 (4.34,7.01)	5.01-6.46	0.1190	5.91	5.64-6.53	5.33	5.04-5.74	0.0111
HDL-C	0.92	0.79-1.16	(4.34-7.01) 1.29	1.16-1.41	0.0017	1.28	1.17-1.32	(3.44-7.39) 1.71	1.42-1.68	0.0003
LDL-C	(0.02-1.55) 3.61 (1.22, 5, 17)	2.75-3.93	(0.62-1.71) 3.84 (2.51, 5.07)	3.05-4.37	0.2798	(0.75-2.09) 3.96	3.61-4.28	(0.75-2.53) 3.44 (2.02.5.25)	3.13-3.88	0.0127
Trigly-	(1.32-5.17) 3.76 (1.27, 11, 28)	3.50-5.74	(2.51-5.07) 2.62 (0.82, 6.12)	2.14-3.31	0.0222	(2.09-6.36) 2.87 (1.24, 7.87)	2.44-3.57	(2.02-5.25) 1.76 (0.01, 4.01)	1.50-1.89	0.0000
Retinol	(1.27-11.58) 2.79	2.51-3.49	(0.85-0.15) 3.00	2.80-3.57	0.2530	(1.54-7.87) 2.81	2.61-2.90	(0.91-4.01) 2.96	2.82-3.12	0.2306
α-	(0.53-4.62) 16.03	13.65-19.26	(2.51-4.02) 18.73	16.40-22.68	0.0235	(0.62-4.59) 17.36	15.22-19.38	(1.20-4.12) 18.53	17.25-19.81	0.2630
α -T/	(6.29-27.48) 3.28	2.76-3.94	(2.62-28.30) 3.52	3.08-3.86	0.3805	(7.95-28.65) 3.10	2.55-3.53	(5.30-30.28) 3.41	2.97-3.95	0.0059
Cholesterol α-T/ Choles+TG	$(1.00-4.77) \\ 1.93 \\ (0.64-3.40)$	1.42-2.05	$\begin{array}{c} (2.73-4.15) \\ 2.29 \\ (1.98-3.15) \end{array}$	2.03-2.63	0.0044	$(1.54-5.15) \\ 2.03 \\ (0.97-3.69)$	1.65-2.31	$\begin{array}{c} (0.81-7.78) \\ 2.56 \\ (0.63-5.20) \end{array}$	2.132-2.91	0.0000

SST = subscapular skinfold thickness; AC = arm circumference; TSF = tricep skinfold thickness; MAMC= mid arm muscle circumference;

W/H=waist/hip ratio; HDL-C=high density lipoprotein cholesterol; LDL-C=low density lipoprotein cholesterol; ^a Mann-Whitney U-Wilcoxon Rank Sum

Decreased retinol and α -tocopherol levels were found in 6.3% (1 out of 16) and 12.5% (2 out of 16) of the overweight and obese males, while 1.8% (1 out of 56) and 10.7% (6 out of 56) were found in overweight and obese females (Table 2). Significant associations were found between age, cholesterol, LDL-C and serum α -tocopherol in both the overweight males and females. A negative correlation was found between weight, BMI, AC, MAMC, hip circumference and serum retinol in both the overweight and obese subjects. A negative correlation was found between weight, hip circumferences and serum α -tocopherol in both the overweight and obese subjects. A negative correlation was found between weight, BMI, MAMC, waist, hip circumferences and serum α -tocopherol in both the overweight and obese subjects. Serum α -tocopherol was significantly affected by age (p<0.05) and also serum cholesterol was significantly affected by age (p<0.001).

When the cut-off points for retinol (normal range 0.7-2.8 μ mol/l) and α -tocopherol (normal range 16.2-46.0 μ mol/l) concentrations suggested by Maiani *et al.*1993; Morrissey *et al.*1993; Olmedilla *et al.*1997²⁴⁻²⁶ were used, serum retinol and α -tocopherol concentrations fell within the accepted normal ranges. Retinol and α -tocopherol deficiencies were found in 6.3% (1 out of 16) and 12.5% (2 out of 16) of the overweight and obese males and 1.8% (1 out of 56) and 10.7% (6 out of 56) in the overweight and obese females. A cholesterol concentration of \geq 6.48 mmol/l was found in 12.5% and 39.3% of the male and female overweight and obese subjects, respectively. However, the prevalence of low HDL-C (HDL-C \leq 0.91 mmol/l) was found to be 50% in the overweight and obese males and 10.7% in the overweight and obese males and 10.7% in the overweight and obese females.

Table 3 shows the correlation coefficients between various parameters in the overweight males and females. Significant associations were found between weight, BMI, AC, MAMC, hip circumference and serum retinol in both overweight males and females. Significant associations were found between age, weight, BMI, MAMC, waist, hip circumferences, cholesterol, LDL-C and serum α -tocopherol in both overweight males and females. A significant positive correlation between age, cholesterol, LDL-C with α -tocopherol, was observed in the overweight male and female subjects.

Discussion

In this study, lower serum retinol and α -tocopherol concentrations were found in the Thai overweight and obese subjects (BMI $\geq 25.0 \text{ kg/m}^2$), when compared with the control subjects. In the subjects studied here, age appeared to influence serum α -tocopherol concentrations in an independent way. Data from the literature show that plasma α -tocopherol appears to increase with age in most longitudinal^{29,30} and cross sectional studies.³¹⁻³⁴

Most studies have found no difference between the genders for plasma retinol in healthy people.³³⁻³⁴ Regarding α -tocopherol, Woo *et al.* found a higher level in women,³⁵ whereas no difference was reported by Hallfrisch *et al.*^{32,36} The sex differences observed in this study may simply be a product of the relatively small sample size and of the likelihood that males were less willing to volunteer.³⁷ In addition, obesity is more common among women than men.³⁸ These factors may be limitations of the study.

Higher levels of LDL-C, LDL-C/HDL-C and triglycerides were present in overweight/obese (Table 1). A prevalence of dyslipidemia (36.1% cholesterol \geq 5.18 mmol/l, 48.6% LDL-C \geq 3.89mmol/l, 33.3% triglyceride) and of serum vitamin deficiencies (2.8% of retinol < 0.7 µmol/l and 11.1% of α -tocopherol <16.2 µmol/l) (Table 2) were observed in obese

Table 2. Number and percentage of individuals with overweight, abnormal vitamin A & E, dyslipidaemia in overweight subjects

Parameter		Male			Female			Total				
	Overwe	eight	Contr	rol	Overw	eight	Cont	rol	Overwe	eight	Con	trol
Grading of overweight by BMI	N/Total	%	N/Total	%	N/Total	%	N/Total	%	N/Total	%	N/Total	%
(kg/m^2)												
Grade I (BMI =25.00-29.99)	7/16	43.8			32/56	57.0			39/72	54.1		
Grade II (BMI =30.00-39.99)	9/16	56.2			22/56	39.2			31/72	43.0		
Grade I (BMI ≥40.00)	-	0.0			3/56	5.3			3/72	4.0		
Vitamin deficiency												
Retinol < 0.7µmol/l*	1/16	6.3	-	0.0	1/56	1.8	-	0.0	2/72	2.8	-	0.0
α -Tocopherol < 16.2 μ mol/l*	2/16	12.5	3/14	21.4	6/56	10.7	15/58	25.8	8/72	11.1	18/72	25.0
Dyslipidemia												
Cholesterol ≥5.18 mmol/l	7/16	43.8	5/14	35.7	19/56	33.9	10/58	17.2	26/72	36.1	15/72	20.8
Cholesterol ≥6.48 mmol/l	2/16	12.5	4/14	28.6	22/56	39.3	13/58	22.4	24/72	33.3	17/72	23.6
HLD-C $\leq 0.91 \text{ mmol/l}$	8/16	50.0	-	0.0	6/56	10.7	3/58	5.2	14/72	19.4	3/72	4.2
$LDL-C \ge 3.89 \text{ mmol/l}$	6/16	37.5	7/14	50.0	29/56	51.8	23/58	39.6	35/72	48.6	30/72	41.7
LDL-C/HDL-C > 5.0	3/16	18.8	-	0.0	3/56	5.3	1/58	1.7	6/72	8.3	1/72	1.4
Triglyceride $\geq 2.26 \text{ mmol/l}$	7/16	43.7	1/14	7.1	17/56	30.3	-	0.0	24/72	33.3	1/72	1.4

*Maiani et al. 1993; Morrisey et al. 1993; Olmedilla et al. 1997.²⁴⁻²⁶

Parameter	Retinol	α-tocopherol	α -tocopherol/chol	α-tocopherol/ (chol+TG)
Age	0.072	0.204*	0.035	0.083
Weight	-0.197*	-0.184*	0.043	0.131
Height	-0.143	-0.054	0.067	0.015
BMI	-0.178*	-0.165*	-0.029	-0.027
AC	-0.232**	-0.160	0.147	0.053
MAMC	-0.273**	-0.228**	-0.066	-0.156
TSF	-0.024	0.024	0.321**	0.248*
SST	-0.086	-0.015	0.239*	0.180
Retinol	1.000	0.336**	0.455**	0.260*
α-tocopherol	0.336**	1.000	0.730**	0.662**
Cholesterol	-0.058	0.358**	0.279*	-0.122
HLD-C	0.018	0.090	-0.146	0.333**
LDL-C	-0.065	0.314**	-0.191	-0.004
Triglycerides	-0.015	0.072	0.089	-0.489**

Table 3. Correlation coefficients of age, anthropometric variables, rettinol, α -tocopherol and lipid profiles in both overweight males and females (BMI $\ge 25.0 \text{ kg/m}^2$)

Significant difference: *P<0.05, **P<0.01

subjects. These appear to have been due to poor dietary intake, which may increase oxidation of LDL and lead to a higher risk of coronary artery disease (CAD). However, we had insufficient dietary intake data for interpretation.

There were no significant differences between median concentrations of serum retinol for overweight/obese subjects and control subjects in both genders (Table 1), which is similar to the results from NHANES II³⁸ and III.³⁹ Serum retinol concentrations are normally maintained within a narrow range in individuals with adequate liver vitamin A stores. When liver stores are depleted, however, serum retinol concentrations decrease. Thus serum retinol is a useful indicator of vitamin A status and can be used to identify subjects with lower depleted liver vitamin A stores.⁴⁰⁻⁴¹

In the control group, women had slightly lower serum retinol concentrations than did men, which may reflect lower liver stores in women, but could also be due to differences in retinol metabolism. Serum retinol binding protein concentrations fluctuate during the menstrual cycle and increase with estrogen therapy⁴² making it unlikely that estrogen-induced differences in retinol metabolism account for the lower concentrations in women. On the other hand, overweight/obese men had slightly lower serum retinol concentrations than women. It has been suggested that dietary differences³⁹ and variability in body compartment size⁴³ are likely explanations. Moreover, men are reported to consume alcohol and smoke tobacco more than women.

The concentration of the lipid-soluble molecule vitamin E, carried by lipoproteins in the bloodstream, was previously reported to correlate closely with that of total cholesterol.^{27, 28} The medians of α -tocopherol/cholesterol were above the cut-off point of 2.2 µmol/mmol²⁵ in all groups and showed significant differences in female overweight/obese subjects and control subjects, but no significant difference between male

overweight/obese subjects and control subjects (Table 1). In male subjects, limitation factors include smoking and alcohol intake, which have been shown to be predictors of retinol, α tocopherol and the carotenoids in adults.^{44,45} In this study, after recalculation using the formula α -tocopherol/cholesterol + triglyceride⁴⁶ the α -tocopherol concentration level was shown to be lower in Thai overweight/obese subjects than normal control subjects in both genders (Table 1). A failure to adjust for lipid concentration in plasma and lipoprotein fractions may lead to an underestimation of the impact of vitamin E on nutritional status⁴⁷ and CAD.²⁷

Strongly significant differences in vitamin E concentrations between overweight/obese and control subjects were noted in the present study after lipid adjustment, as described by Riemersma *et al.*²⁷ Earlier studies showed that normolipidaemic subjects have the largest amount of plasma vitamin E in the LDL fraction, whereas subjects with elevated triacyglycerol concentrations have the largest amount of plasma vitamin E in VLDL fraction.^{28,48}

The results of the present study provide further support for the notion that low concentrations of antioxidant vitamins may be an important risk factor for CAD. The results of epidemiological studies of the relation between antioxidant vitamins and CAD have not been entirely congruent. The basis for lower serum concentrations of retinol and α tocopherol in obese people compared with non-obese people remain speculative, but it has been suggested that dietary differences and variability in body compartment size are likely explanations. Furthermore, the overweight and obese group had been informed about a lipid lowering diet at the time of the investigation. The diet instructions aimed at increasing the intake of polyunsaturated fatty acids and hence vitamin E, which may have decreased any existing differences in vitamin E concentrations between overweight/ obese and control subjects. Prospective randomized controlled trials are clearly needed to answer the question of a truly beneficial role for antioxidants in preventing atherosclerosis and a better understanding of the highly complex process.

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