Correlation between vitamin A, E, coenzyme Q<sub>10</sub> and degree of insulin resistance in obese and non-obese subjects

Idris Mehmetoglu, F. Hümeyra Yerlikaya* and Sevil Kurban

University of Selcuk, Meram Faculty of Medicine, Biyokimya AD, 42080, Department of Biochemistry, Konya, Turkey

(Received 11 January, 2011; Accepted 6 February, 2011; Published online 5 October, 2011)

The aim of the present study was to investigate correlation between plasma vitamin A, vitamin E, serum coenzyme Q<sub>10</sub> levels and degree of insulin resistance in obese and normal weight people. The study was performed on 98 (21 Male, 77 Female) obese people and 78 (20 Male, 58 Female) control subjects. Vitamin A, E and coenzyme Q<sub>10</sub> levels were adjusted to the lipid levels. Adjusted vitamin A and E and coenzyme Q<sub>10</sub> levels of the obese female group were significantly lower than those of the control female group. Adjusted vitamin A and coenzyme Q<sub>10</sub> levels of the obese male group were significantly lower than those of the control male group. Insulin resistance level of the obese female and male groups were significantly higher than that of the control female and male groups. There were no significant correlations between serum coenzyme Q<sub>10</sub>, plasma vitamin A and E levels and insulin resistance in obese and control subjects. Our findings show that it is essential to use the lipid adjusted levels of lipid soluble nutrients in obesity. Also, we have found no association between insulin resistance and vitamin A, vitamin E and coenzyme Q<sub>10</sub> levels in obese subjects.

Key Words: coenzyme Q<sub>10</sub>, vitamin A, vitamin E, insulin resistance, obesity

The prevalence of obesity has increased in world with a major impact on public health because of its role as risk factor for many diseases including cardiovascular disease and cancer, with a consequent reduction in life expectancy. Obesity has been associated with low-grade inflammation, playing a role in the pathogenesis of insulin resistance and cardiovascular risk and with oxidative stress, an imbalance between the production of reactive oxygen species and antioxidant defences that is also involved in the pathogenesis of cardiovascular disease and cancer.

The antioxidants α-tocopherol, the dominant component of vitamin E, vitamin A and coenzyme Q<sub>10</sub> (CoQ<sub>10</sub>) contribute to the body’s defense against reactive oxygen species. Epidemiologic studies have consistently shown that higher intakes of vitamin A, vitamin E and CoQ<sub>10</sub> are associated with reduced risk of several chronic diseases, including heart failure, age-related macular degeneration and some cancers. These micronutrients exhibit multiple biological actions that may protect against disease. For example, vitamin E is a chain breaking antioxidant that protect cell membranes from damage caused by lipid peroxidation and also inhibits cell proliferation and platelet adhesion. Vitamin A is essential for differentiation of epithelial cells and maintenance of cell signaling and communication. CoQ<sub>10</sub> is well known for its role as an electron transporter in the mitochondrial respiratory chain. Also, CoQ<sub>10</sub> is a powerful antioxidant even stronger than α-tocopherol.

The aim of the present study was to investigate correlation between vitamin A, vitamin E, CoQ<sub>10</sub> levels and homeostasis model assessment (HOMA-IR) in obese and normal weight subjects. Also, we examined associations of these parameters with body mass index (BMI), waist circumference and waist-to-hip ratio in the subjects.

Materials and Methods

Patients. This study was performed on 98 (21M, 77F) obese people aged 18–65 years and 78 (20M, 58F) control subjects aged 18–65 years. There were no complaints and symptoms of the obese subjects other than obesity. BMI was used as an obesity criteria. BMI of the obese subjects was more than 30 kg/m<sup>2</sup> and that of healthy controls was less than 25 kg/m<sup>2</sup>. Diatery patterns of the subjects were estimated using a food questionnaire. Less than 5% of the obese and healthy control participants reported tobacco use. None of the subjects were taking a HMG-CoA reductase inhibitor (statin) treatment and vitamin supplementation. The study was approved by Hospital Ethics Committee, and informed consent was obtained from all participants. Blood samples were obtained from the subjects between 8 A.M. and 9 A.M. after a 12- h overnight fast on plain and EDTA tubes. Plasma and sera of the samples were separated and frozen at −80°C until the day of study and thawed only once.

Anthropometric assessment. All anthropometric measurement were made with participants wearing light clothing and no shoes. BMI was measured in all participants. BMI was calculated as weight (in kilograms) divided by height (in meters) squared, and participants waists were measured with a soft tape midway between the lowest rib and the iliac crest. The hip circumferences was measured at the widest part of the gluteal region.

Sample analyses. Serum CoQ<sub>10</sub> levels were measured using the ImmunoChrom reagent kit for high-performance liquid chromatography (HPLC) technique with UV-detector. Plasma (in EDTA-blood) vitamin A and vitamin E levels were measured using the Chromsystems (Munich, Germany) reagent kit for HPLC technique with UV-detector. Serum total cholesterol, triglycerides, high density lipoprotein (HDL) cholesterol and blood glucose was measured by commercially available kits based by routine methods on the Synchron LX System (Beckman Coulter, Fullerton CA). Low density lipoprotein (LDL) cholesterol was calculated using the formula by Friedewald et al. Insulin levels were determined by routine chemiluminesans method on E170 analyzer (Roche Diagnostics).

Calculations. HOMA-IR was used to detect the degree of insulin resistance. The resistance can be assessed from the fasting
blood glucose and insulin concentrations by the formulae:

\[ \text{HOMA-IR} = \text{insulin (mU/l)} \times \text{glucose (mmol/l)} / 22.5 \]

HOMA-IR scores greater than \( \geq 2.5 \) shows low insulin sensitivity (insulin resistance).\(^{14,15}\) Adjusted vitamin A and vitamin E were calculated as vitamin A/(total cholesterol + triglycerides) (\( \mu \text{mol/mmol} \)) and vitamin E/(total cholesterol + triglycerides) (\( \mu \text{mol/mmol} \)).

**Statistical analysis.** All data are expressed as mean ± standard deviation (SD). Statistical analyses were done using SPSS ver. 17.0 (SPSS Inc., IL). The normal distribution of variables were examined with Independent-Samples t test, and non-normally distributed variables were examined by Mann-Whitney U test. The correlations between variables were performed by Pearson’s Correlation test. Differences were considered significant at a probability level of \( p < 0.05 \).

**Results**

Clinical and analytical characteristics of the obese and control subjects are presented in Table 1. As seen, BMI, waist circumference, waist-to-hip ratio, systolic and diastolic blood pressure, total cholesterol, LDL-cholesterol, triglycerides, fasting blood glucose, fasting blood insulin and HOMA-IR values of the obese female and male subjects were significantly higher than those of the controls (\( p < 0.001 \)). Total-cholesterol and LDL-cholesterol of the obese female subjects were significantly higher than those of the controls (\( p < 0.001 \) for total-cholesterol and \( p < 0.01 \) for LDL-cholesterol) whereas HDL-cholesterol level of obese female group was lower than that of the control group (\( p < 0.001 \)). There were no significant differences between total-cholesterol, LDL-cholesterol and HDL-cholesterol levels of the male groups.

Vitamins and \( \text{CoQ} \) concentrations of the obese and the control subjects are presented in Table 2. As seen, adjusted vitamin A (\( p < 0.001 \)), adjusted vitamin E (\( p < 0.05 \)), \( \text{CoQ} \)/total cholesterol (\( p < 0.05 \)), \( \text{CoQ} \)/LDL (\( p < 0.05 \)), \( \text{CoQ} \)/(triglycerides) (\( p < 0.001 \)) and \( \text{CoQ} \)/(total cholesterol + triglycerides) (\( p < 0.001 \)) values of the obese female subjects were significantly lower than those of the control group. Adjusted vitamin A (\( p < 0.05 \)) and \( \text{CoQ} \)/(triglycerides) (\( p < 0.05 \)) values of the obese male subjects were significantly lower than those of the control group.

Simple correlation analysis were performed to investigate the association of plasma vitamins (adjusted and unadjusted) and \( \text{CoQ} \) measures with BMI, waist circumference, waist-to-hip ratio, age and HOMA-IR. As shown in Table 3, vitamin A (\( r = -0.222, \)

---

**Table 1. Clinical and analytical characteristics of the control and the obese subjects**

|                      | Control (mean ± SD) | Obese (mean ± SD) |
|----------------------|---------------------|-------------------|
|                      | Female (n = 58)     | Male (n = 20)     | Female (n = 77) | Male (n = 21) |
| Age (years)          | 36.15 ± 10.1        | 39.20 ± 14.4      | 37.65 ± 12.4   | 39.47 ± 14.1  |
| BMI (kg/m²)          | 20.66 ± 2.4         | 22.10 ± 2.2       | 43.61 ± 6.4**  | 39.85 ± 6.0**  |
| Waist circumference (cm) | 71.15 ± 7.4        | 84.70 ± 5.3       | 117.85 ± 11.5**| 123.85 ± 11.4**|
| Waist-to-hip ratio   | 0.74 ± 0.05         | 0.84 ± 0.03       | 0.84 ± 0.06**  | 0.95 ± 0.07**  |
| Systolic blood pressure (mmHg) | 110.13 ± 4.1      | 117.1 ± 2.8       | 130.13 ± 3.4** | 137.13 ± 3.4** |
| Diastolic blood pressure (mmHg) | 70.1 ± 2.5         | 73.4 ± 1.0        | 82.1 ± 3.0**   | 84.2 ± 2.4**   |
| Total cholesterol (mmol/l) | 4.62 ± 0.9         | 4.91 ± 0.9        | 5.14 ± 1.1**   | 5.0 ± 1.0      |
| Triglycerides (mmol/l) | 0.80 ± 0.4          | 1.23 ± 0.5        | 1.77 ± 1.0**   | 1.98 ± 0.9**   |
| HDL-cholesterol (mmol/l) | 1.39 ± 0.3          | 1.01 ± 0.2        | 1.13 ± 0.2**   | 0.92 ± 0.2     |
| LDL-cholesterol (mmol/l) | 2.86 ± 0.8          | 3.15 ± 1.0        | 3.19 ± 0.9*    | 3.34 ± 0.8     |
| Fasting blood glucose (mg/dl) | 84.11 ± 7.4        | 83.8 ± 10.6       | 99.81 ± 12.9** | 100.1 ± 11.3**|
| Fasting blood insulin (μU/ml) | 5.65 ± 2.7         | 6.26 ± 3.9        | 15.94 ± 8.4**  | 22.07 ± 11.7** |
| HOMA-IR              | 1.16 ± 0.6          | 1.31 ± 0.9        | 3.75 ± 3.2**   | 4.64 ± 2.9**   |

\(^1\) standard deviation. BMI, body mass index. HDL-cholesterol, high density lipoprotein-cholesterol. LDL-cholesterol, low density lipoprotein-cholesterol. HOMA-IR, homeostasis model assessment. \(^*\)\( p < 0.05 \), \(^**\)\( p < 0.01 \).

**Table 2. Vitamins and \( \text{CoQ} \) concentrations in the control and the obese subjects**

|                      | Control (mean ± SD) | Obese (mean ± SD) |
|----------------------|---------------------|-------------------|
|                      | Female (n = 58)     | Male (n = 20)     | Female (n = 77) | Male (n = 21) |
| Vitamin A (retinol) (µmol/l) | 1.84 ± 0.6         | 2.33 ± 0.5        | 2.01 ± 0.5      | 2.25 ± 0.5     |
| Adjusted Vitamin A (µmol/mmol) (vitamin A/(total cholesterol + triglycerides)) | 0.34 ± 0.1         | 0.39 ± 0.1        | 0.29 ± 0.08**  | 0.32 ± 0.09*   |
| Vitamin E (α-tocopherol) (µmol/l) | 30.46 ± 8.5        | 32.55 ± 8.3       | 33.47 ± 8.8    | 29.75 ± 8.1    |
| Adjusted Vitamin E (µmol/mmol) (vitamin E/(total cholesterol + triglycerides)) | 5.53 ± 1.1         | 4.86 ± 0.9        | 5.07 ± 1.2*    | 4.67 ± 1.2     |
| \( \text{CoQ} \) (µmol/l) | 1.58 ± 0.7         | 1.46 ± 0.5        | 1.49 ± 0.6     | 1.38 ± 0.4     |
| \( \text{CoQ} \)/total cholesterol (µmol/mmol) | 0.34 ± 0.1         | 0.29 ± 0.1        | 0.29 ± 0.1*    | 0.27 ± 0.07    |
| \( \text{CoQ} \)/LDL (µmol/mmol) | 0.58 ± 0.3         | 0.44 ± 0.1        | 0.48 ± 0.2*    | 0.47 ± 0.1     |
| \( \text{CoQ} \)/(triglycerides) (µmol/mmol) | 2.50 ± 2.0         | 1.28 ± 0.5        | 1.05 ± 0.6**   | 0.89 ± 0.5*    |
| \( \text{CoQ} \)/(total cholesterol + triglycerides) (µmol/mmol) | 0.30 ± 0.1         | 0.024 ± 0.08      | 0.22 ± 0.08**  | 0.20 ± 0.05    |

\(^1\) standard deviation. \( \text{CoQ} \), coenzyme Q. LDL, low density lipoprotein-cholesterol. \(^*\)\( p < 0.05 \), \(^**\)\( p < 0.01 \).
p<0.05) and adjusted vitamin A \((r = -0.269, p<0.01)\) levels were negatively correlated with BMI and adjusted vitamin A was negatively correlated with waist circumference \((r = -0.202, p<0.05)\) in obese group. Vitamin A was positively correlated with age \((r = 0.258, p<0.05)\) in obese group.

Adjusted vitamin E was negatively correlated with BMI \((r = -0.237, p<0.05)\), waist circumference \((r = -0.380, p<0.01)\) and waist-to-hip ratio \((r = -0.271, p<0.05)\) in control group (Table 1). On the other hand, adjusted vitamin E was negatively correlated with waist circumference \((r = -0.233, p<0.05)\), waist-to-hip ratio \((r = -0.273, p<0.01)\) in obese group. Furthermore, vitamin E \((r = 0.246, p<0.05)\) was positively correlated with age in obese group.

CoQ\textsubscript{10} was positively correlated with age \((r = 0.240, p<0.05)\) in obese group. CoQ\textsubscript{10}/total cholesterol ratio was negatively correlated with age \((r = -0.228, p<0.05)\) in control group (Table 5). CoQ\textsubscript{10}/LDL ratio was negatively correlated with waist circumference \((r = -0.337, p<0.01)\), waist-to-hip ratio \((r = -0.285, p<0.05)\) and age \((r = -0.295, p<0.01)\) in the control group. CoQ\textsubscript{10}/triglyceride ratio was negatively correlated with waist circumference \((r = -0.387, p<0.01)\), waist-to-hip ratio \((r = -0.285, p<0.05)\) in the control group. CoQ\textsubscript{10}/total cholesterol + triglyceride ratio was negatively correlated with waist circumference \((r = -0.281, p<0.01)\), waist-to-hip ratio \((r = -0.250, p<0.05)\) and age \((r = -0.233, p<0.05)\) in the control group.

CoQ\textsubscript{10} was positively correlated with vitamin E in obese group \((r = 0.272, p<0.01)\) (data not shown). On the other hand, there were no correlations between HOMA-IR values and adjusted and unadjusted vitamin E, vitamin A and CoQ\textsubscript{10} levels in the obese and the control subjects.

### Discussion

In the present study, adjusted serum vitamin E concentration was significantly higher in the obese female group than in the control group. However, there was no significant difference between unadjusted vitamin E levels of the groups. Also, there was no correlation between unadjusted vitamin E levels and BMI while adjusted vitamin E level was negatively correlated with BMI in the control group and waist circumference and waist-to-hip ratio in the obese and the control group. Since vitamin E is transported in lipoprotein particles we believe that it is more accurate to use the lipid-adjusted vitamin E levels to evaluate vitamin E status of the obese subjects. Indeed, it has been reported that the strongest and most consistent predictor of all serum fat-soluble nutrients were serum cholesterol\(^{16,16,17}\).

In the literature, there are conflicting findings about the correlation of vitamin E levels with obesity. For example, adjusted\(^{18}\) and unadjusted\(^{15,19,20}\) plasma vitamin E level was found to be negatively associated with BMI. In contrast, adjusted vitamin E level was not associated in one study\(^{21}\) and positively associated with BMI in another study\(^{5}\).

We believe that the reason for the differences between these findings is the usage of the adjusted and unadjusted levels of

---

**Table 3. Correlations of plasma vitamin A and adjusted vitamin A levels with clinical and biochemical variables of the control and the obese subjects**

| Variable          | Control \((n = 78)\) | Obese \((n = 98)\) |
|-------------------|----------------------|-------------------|
|                   | Vitamin A | Adjusted vitamin A | Vitamin A | Adjusted vitamin A |
| BMI               | 0.132     | -0.085             | -0.222*   | -0.269**           |
| Waist circumference | 0.336     | 0.060              | -0.161    | -0.202*            |
| Waist-to-hip ratio | 0.320     | 0.082              | -0.019    | -0.059             |
| HOMA-IR           | 0.148     | 0.135              | 0.073     | 0.092              |

BMI, body mass index. HOMA-IR, homeostasis model assessment. *\(p<0.05\), **\(p<0.01\).

**Table 4. Correlations of plasma vitamin E and adjusted vitamin E levels with clinical and biochemical variables of the control and the obese subjects**

| Variable          | Control \((n = 78)\) | Obese \((n = 98)\) |
|-------------------|----------------------|-------------------|
|                   | Vitamin E | Adjusted vitamin E | Vitamin E | Adjusted vitamin E |
| BMI               | 0.068     | -0.237*            | 0.095     | -0.044             |
| Waist circumference | 0.060     | -0.380**           | 0.047     | -0.233*            |
| Waist-to-hip ratio | 0.080     | -0.271*            | -0.121    | -0.273**           |
| HOMA-IR           | -0.082    | -0.130             | 0.134     | -0.003             |

BMI, body mass index. HOMA-IR, homeostasis model assessment. *\(p<0.05\), **\(p<0.01\).

**Table 5. Correlations of serum CoQ\textsubscript{10} levels with clinical and biochemical variables of the control and the obese subjects**

| Variable          | Control | Obese |
|-------------------|---------|-------|
|                   | BMI     | Waist circumference | Age | HOMA-IR | BMI     | Waist circumference | Age | HOMA-IR |
| CoQ\textsubscript{10} | 0.110   | -0.065           | -0.509 | 0.065  | 0.140  | 0.085           | 0.101 | 0.240* |
| CoQ\textsubscript{10}/total cholesterol | -0.026 | -0.217           | -0.185 | -0.228* | 0.054  | 0.142  | 0.067           | 0.095  | 0.126  | 0.063 |
| CoQ\textsubscript{10}/LDL | -0.147 | -0.337**         | -0.285* | -0.295** | 0.057  | 0.168  | 0.083           | 0.084  | 0.111  | -0.050 |
| CoQ\textsubscript{10}/triglyceride | -0.155 | -0.387**         | -0.285* | -0.211  | -0.060 | -0.097 | -0.089           | -0.022  | 0.111  | -0.055 |
| CoQ\textsubscript{10}/total cholesterol + triglyceride | -0.059 | -0.281*         | -0.250* | -0.233*  | 0.017  | 0.064  | 0.003           | 0.049  | 0.108  | -0.047 |

BMI, body mass index. LDL-cholesterol, low density lipoprotein-cholesterol. HOMA-IR, homeostasis model assessment. CoQ\textsubscript{10}, coenzyme Q\textsubscript{10}. *\(p<0.05\), **\(p<0.01\).
the vitamin. Another reason might be the differences between the degree of obesity, age and dietary habituate of the investigated subjects.

In our study there was no significant difference between unadjusted plasma vitamin A levels of the obese and control subjects. However, adjusted vitamin A level of the obese female and male subjects was significantly lower than that of the control group. Also, adjusted vitamin A level was negatively correlated with BMI and waist circumference in the obese group. Since retinol is considered an antioxidant and anti-inflammatory agent(22,23) our results suggest that obesity is associated with a lesser capacity of anti-inflammatory and antioxidant potential. Although there are some conflicting findings concerning plasma vitamin A levels of obese subjects, generally the findings of the other investigators are in consisting with our findings. For example, Viroonudomphol et al. (19) have found a negative correlation between serum unadjusted retinol and BMI, waist-hip circumferences in both overweight and obese subjects. Botella-Carretero et al. (5) have found a negative correlation of serum unadjusted retinol with BMI in morbid obese people. Neuhouser et al. (5) have found that obesity was associated with low serum unadjusted retinol concentration. Switzer et al. (21) have reported that plasma adjusted retinol concentrations was not associated with BMI in older African American women.

The present study demonstrated that there was no difference between unadjusted plasma CoQ_{10} levels of the obese female and male subjects and the control subjects. However, lipid adjusted CoQ_{10} levels were significantly lower in the obese female and male subjects than in the controls. Since, CoQ_{10} is a powerful antioxidant, our lipid adjusted CoQ_{10} levels show that there is an antioxidant deficiency in obese subjects.

Mancini et al. (15) have found no difference between unadjusted plasma CoQ_{10} levels of obese and control subjects. Also, Menke et al. (14) have found that unadjusted plasma CoQ_{10} levels of obese and normal weight children were not different. Butler et al. (20) have reported that unadjusted plasma CoQ_{10} level was significantly lower in obese subjects than in control subjects. Since, CoQ_{10} is transported in lipoproteins, it is clear that lipid-corrected levels are more informative. Therefore, we believe that our results provide more accurate information about CoQ status of the obese subjects.

On the other hand, CoQ_{10} originates from endogenous synthesis as well as from food intake. (14) Therefore, the ethnic difference in lifestyle and nutritional patterns may result in differences in plasma CoQ_{10} levels found in different studies. (25)

In this study, both unadjusted plasma CoQ_{10} and unadjusted vitamin E levels positively correlated to cholesterol, triglycerides and HDL concentrations in the obese subjects. Also, unadjusted plasma CoQ_{10} levels were positively correlated to unadjusted vitamin E in obese people (data not shown). This may indicate that vitamin E and endogenous CoQ_{10} synthesis may compensate the greater demand of lipophilic antioxidants in the plasma in situations like obesity where plasma lipids are increased. (26-28)

Both vitamin E and CoQ_{10} possess distinct lipoprotective antioxidant properties in biological membranes. Their combined antioxidant activity, however, is markedly synergistic when both are present together. (29) These data provide direct evidence for an interactive effect between vitamin E and CoQ_{10} in obese people.

In our study, we have found no correlation between HOMA-IR, CoQ_{10}, vitamin A and E values in both the obese and the control subjects. Botella-Carretero et al. (5) have found no correlation between HOMA-IR and unadjusted plasma vitamin A and E levels in obese subjects, a finding which supports our findings. We have found no study investigating an association between (adjusted/ unadjusted) CoQ_{10} levels and HOMA-IR values in obese subjects. Therefore, our study is the first one investigating such a correlation.

As seen from our data (Table 2), the findings of the obese female and the control female are different from those of the findings of the obese male and the control male. Although the underlying mechanism of these differences are not known we believe that this may be due to metabolic differences between male and female subjects and the lower number of male subjects in our study.

In conclusion, our findings show that lipid-adjusted vitamin A, E and CoQ_{10} levels of the obese subjects were significantly lower than those of the controls. Also, we have found no association between HOMA-IR and vitamin A, E and CoQ_{10} levels in the obese subjects.

Abbreviations

| Abbreviation | Description |
|--------------|-------------|
| BMI          | body mass index |
| CoQ_{10}     | coenzyme Q_{10} |
| HDL          | high density lipoprotein |
| HOMA-IR      | homeostasis model assessment |
| HPLC         | high-performance liquid chromatography |
| LDL          | low density lipoprotein |
| SD           | standard deviation |

References

1. Calle EE, Rodriguez C, Walker-Thurmond K, Thun MJ. Overweight, obesity, and mortality from cancer in a prospectively studied cohort of U.S. adults. N Engl J Med 2003; 348: 1625–1638.
2. Ikeoka D, Mader JK, Pieber TR. Adipose tissue, inflammation and cardio-vascular disease. Rev Assoc Med Bras 2010; 56: 116–121.
3. Kahn BB, Flier JS. Obesity and insulin resistance. J Clin Invest 2000; 106: 473–481.
4. Wilson PW, D’Agostino RB, Sullivan L, Parise H, Kannel WB. Overweight and obesity as determinants of cardiovascular risk: the Framingham experience. Arch Intern Med 2002; 162: 1867–1872.
5. Botella-Carretero JJ, Balsa JA, Vázquez C, Peromingo R, Díaz-Enríquez M, Escobar-Morreale HF. Retinol and α-tocopherol in morbid obesity and non-alcoholic fatty liver disease. Obes Surg 2010; 20: 69–76.
6. Wallström P, Wirfält E, Lahmann P, Gullberg B, Janzon L, Berglund G. Serum concentrations of β-carotene and α-tocopherol are associated with diet, smoking, and general and central adiposity. Am J Clin Nutr 2001; 73: 777–785.
7. Crane FL. Biochemical Functions of Coenzyme Q_{10}. J Am Coll Nutr 2001; 20: 591–598.
8. Sohal RS, Forster MJ. Coenzyme Q, oxidative stress and aging. Mitochondrion 2007; 7: S103–S111.
9. Neuhouser ML, Rock CL, Eldridge AL, et al. Serum concentrations of retinol, α-tocopherol and the carotenoids are influenced by diet, race and obesity in a sample of healthy adolescents. J Nutr 2001; 131: 2184–2191.
10. Bonakdar RA, Guarnieri E. Coenzyme Q_{10}. Am Fam Physician 2005; 72: 1065–1070.
11. Plat J, Mensink RP. Effect of diets enriched with two different plant stanol ester mixtures on plasma ubiquinol-10 and fat-soluble antioxidant concentrations. Metabolism 2001; 50: 520–529.
12. Quinzii CM, DiMauro S, Hirano M. Human Coenzyme Q_{10} Deficiency. Neurochem Res 2007; 32: 723–727.
13. Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. Clin Chem 1972; 18: 499–502.
14. Menke T, Niklozwitz P, de Sousa, Reinehr T, Andler W. Comparison of coenzyme Q_{10} plasma levels in obese and normal weight children. Clin Chim Acta 2004; 349: 121–127.
15. Mancini A, Leone E, Festa R, et al. Evaluation of antioxidant systems (coenzyme Q_{10} and total antioxidant capacity) in morbid obesity before and after biliopancreatic diversion. Metabolism 2008; 57: 1384–1389.
16. Morinobu T, Murata T, Takaya R, Tamai H. Nutritional status of beta-carotene, alpha-tocopherol and retinol in obese children. Int J Vitam Nutr Res

©2011 JCBN
17 Zwirska-Korczala K, Jagodzińska J, Wielkoszyński T, et al. Plasma oxysterols and vitamin E concentrations and lipid profile in morbidly obese women. Pol Arch Med Wewn 2001; 106: 909–915.

18 Aasheim ET, Hofso D, Hjelmesæth J, Birkeland KI, Bøhmer T. Vitamin status in morbidly obese patients: a cross-sectional study. Am J Clin Nutr 2008; 87: 362–369.

19 Viroonudomphol D, Pongpaew P, Tungtrongchitr R, et al. The relationships between anthropometric measurements, serum vitamin A and E concentrations and lipid profiles in overweight and obese subjects. Asia Pac J Clin Nutr 2003; 12: 73–79.

20 White E, Kristal AR, Shikany JM, et al. Correlates of serum alpha- and gamma-tocopherol in the Women’s Health Initiative. Ann Epidemiol 2001; 11: 136–144.

21 Switzer BR, Atwood JR, Stark AH, et al. Plasma carotenoid and vitamins A and E concentrations in older African American women after wheat bran supplementation: effect of age, body mass and smoking history. J Am Coll Nutr 2005; 24: 217–226.

22 Reifen R. Vitamin A as an anti-inflammatory agent. Proc Nutr Soc 2002; 61: 397–400.

23 Papas A, Stacewicz-Sapuntzakis M, Lagiou P, Bamia C, Chloptsis Y, Trichopoulou A. Plasma retinol and tocopherol levels in relation to demographic, lifestyle and nutritional factors of plant origin in Greece. Br J Nutr 2003; 89: 83–87.

24 Butler MG, Dasouki M, Bittel D, Hunter S, Naini A, DiMauro S. Coenzyme Q10 levels in Prader-Willi syndrome: comparison with obese and non-obese subjects. Am J Med Genet A 2003; 119A: 168–171.

25 Hughes K, Lee BL, Feng X, Lee J, Ong CN. Coenzyme Q10 and differences in coronary heart disease risk in Asian Indians and Chinese. Free Radic Biol Med 2002; 32: 132–138.

26 Kagan VE, Fabisiak JP, Quinn PJ. Coenzyme Q10 and vitamin E need each as antioxidants. Protoplasma 2000; 214: 11–18.

27 Ohrvall M, Tengblad S, Vessby B. Lower tocopherol serum levels in subjects with abdominal adiposity. J Intern Med 1993; 234: 53–60.

28 Knekt P, Seppänen R, Aaran RK. Determinants of serum alpha-tocoferol in Finnish adult. Prev Med 1998; 17: 725–735.