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A dietary pattern characterized by high consumption of whole-grain cereals and low-fat dairy products and low consumption of refined cereals is positively associated with plasma adiponectin levels in healthy women

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Abstract

In light of the potential beneficial effects of adiponectin on insulin resistance, metabolic syndrome, and cardiovascular risk, it is becoming increasingly important to identify all modifiable factors, including dietary patterns, that may affect circulating adiponectin concentrations. The aim of the present study was to explore potential associations between dietary patterns and plasma adiponectin levels using principal component analysis (PCA) in a sample of apparently healthy adult Mediterranean women. Two hundred twenty women were enrolled in this study. Anthropometric and body composition measurements were performed in all subjects. Blood samples were taken, and adiponectin concentrations were measured. Food intake was evaluated by 3-day food diaries, and PCA was used for the identification of the participants' dietary patterns. The PCA identified 10 dietary components explaining 82% of the total variance in food intake. Bivariate correlation between circulating adiponectin levels and dietary components revealed a positive significant association only with the first component that was characterized by high intake of whole-grain cereals and low-fat dairy products as well as low intake of refined cereals ($P = .04$). This association remained unchanged after controlling for potential confounders (standardized β coefficient = 0.18, $P = .03$). A dietary pattern characteristic of consumption of alcoholic beverages was found to be marginally related to adiponectin levels in the multivariate model (standardized β coefficient = 0.14, $P = .10$). Our data indicate that a dietary pattern characterized by a high consumption of whole-grain cereals and low-fat dairy products is modestly positively associated with adiponectin concentrations.

1. Introduction

It has become increasingly evident that adipokines participate actively in a number of physiological processes to mediate the effects of obesity and lifestyle factors in modulating metabolism, inflammation, and cardiovascular risk [1]. Adiponectin, in particular, has been shown to improve insulin action, as well as glucose and lipid metabolism [2,3]. Cross-sectional studies in both diabetic and nondiabetic populations have indicated a positive association between plasma adiponectin levels and favorable lipid profile, glycemic control, and reduced concentrations of inflammatory markers [4-8]. Furthermore, increased blood adiponectin concentrations have been prospectively associated with reduced risk of diabetes [9,10]. In light of the potential beneficial effect of adiponectin on endocrine-related disorders and cardiovascular risk, it is becoming important to identify modifiable lifestyle factors that may affect adiponectin blood concentrations, including diet-related parameters.

The effect of energy restriction on adiponectin levels has been investigated with ambiguous results. Weight loss has been associated with an increase [11–14], no change [15,16],

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or a decrease in adipokine levels [17]. The diversity of the findings reported can be attributed, in part, to differences in the duration of energy restriction, rate of weight loss, type of adipose tissue lost (visceral vs subcutaneous fat), or types of dietary interventions, as well as the obesity status of the study participants [17,18].

With regard to the dietary variables, no statistically significant association has been observed between serum adiponectin concentrations and total energy or macronutrient intake [19]. In contrast to macronutrients, significant associations between circulating adiponectin and specific food groups have been recently reported. In particular, high intakes of dietary cereal fiber or whole-grain cereals have been associated with higher levels of plasma adiponectin concentrations [20-22]. This is consistent with previous findings that diets with low glycemic load or index have a favorable effect in both healthy and diabetic individuals [20-23]. Finally, several trials have shown that moderate alcohol consumption (40 g/d) either in the form of whisky, red wine, or beer can induce increases in adiponectin levels in the context of short-term interventions [24-26].

Dietary pattern analysis, through consideration of nutrients and foods, their interactions, intercorrelations, and cumulative effects, has been used as an alternative approach to studying overall eating. Importantly, dietary patterns reflect real-world situations where foods are consumed in combination [27]. The methodology for defining dietary patterns consists of 3 main approaches: analysis dietary indices, cluster analysis, and factor analysis, including principal component analysis. Dietary indices, based on scientific information and dietary recommendations or guidelines, reflect diet quality and, more specifically, the degree of adherence to a particular diet. On the other hand, the exploratory approach, that is, factor or cluster analysis, identifies dietary patterns representing actual eating practices based on the dietary information of the population under investigation. In this regard, using a Mediterranean diet score it has been shown that adherence to a Mediterranean-type diet is positively related to plasma adiponectin concentrations in diabetic women with no history of heart disease, even after controlling for potential confounders [22].

To our knowledge, no prior study has evaluated associations between plasma adiponectin levels and diet assessed by principal component analysis [28]. The aims of the present study were to explore potential associations between plasma adiponectin concentrations, overall diet, and eating patterns using principal component analysis in a sample of apparently healthy adult Mediterranean women.

2. Subjects and methods

2.1. Subjects

Two hundred twenty Greek women without a known history of diabetes, cardiovascular disease, or cancer were consecutively enrolled in this study in response to an advertisement in a local magazine. The study protocol was approved by the Ethics Committee of Harokopio University. After being informed on the purpose and procedures of the study and signing a consent form, subjects provided a fasting blood sample and underwent anthropometric and body composition measurements. Demographic, medical, and lifestyle information was collected from study participants by the same well-trained researcher. Women (mean age, 48.3 ± 12.3 years; age range, 18-84 years) were classified as premenopausal if they had regular menses, perimenopausal if they were having irregular menses, and postmenopausal if they had ceased menstruating for at least 12 months. Duration and intensity of smoking were also recorded; never smokers and former smokers were categorized as nonsmokers and the rest as current smokers.

Although study subjects reported no known history of diabetes, we found 2 subjects with fasting glucose levels higher than 126 mg/dL, who were excluded from the analysis. We also excluded 22 subjects who either were taking medication (corticosteroids, lipid-lowering, or other drugs) or were on a weight-reducing diet. Thus, our analysis was restricted to 196 apparently healthy women taking no medications and not reporting to actively lose weight. None of the study participants had been exposed to hormone replacement therapy for menopause, and only 3 received oral contraceptives.

2.2. Body composition

Anthropometric and body composition measurements were performed with the subject wearing light clothing and without shoes. Body weight and height were measured on a leveled platform scale with a beam, movable weights, and a wall-mounted stadiometer to the nearest 0.5 kg and 0.5 cm, respectively. Body mass index (BMI) was computed as weight (in kilograms) divided by height (in meters squared). *Obesity* was defined as BMI >29.9 kg/m^2. Waist circumference (in centimeters) was measured in the middle between the 12th rib and the iliac crest; and hip circumference (in centimeters) around the buttocks, at the level of the maximum extension. The waist-to-hip ratio was then calculated. Dual-energy x-ray absorptiometry was used for the assessment of body composition (Model DPX+, Lunar, Madison, WI).

2.3. Dietary assessment

Dietary intake was assessed using 3-day food records. Subjects were asked to record the type and amount of food and beverage consumed for 2 specific consecutive weekdays and 1 weekend day. Clear instructions were given to them on how to record the quantity of food eaten using standard household and other measures. The frequency of several food groups' consumption was then approximately quantified in terms of number of servings per day. The food groups assessed were the core food groups of the traditional Greek

Table 1
Description of the food groups and items evaluated

	Food items included
Whole-grain cereals	Whole-grain breakfast cereals, whole-grain bread, brown rice, barley rusks
Refined cereals	Refined breakfast cereals, white bread, pasta, white rice, pastries, crisp bread
Potatoes	Baked, fried, or boiled potatoes
Vegetables	All kinds of vegetables (leafy, cruciferous, root vegetables, tomatoes): fresh, boiled, or cooked
Fruits	Fresh fruits or freshly squeezed juices
Nuts	Nuts as whole or part of composite dishes (eg, sweets)
Legumes	All types of legumes
Fish	Small or big fishes, crustaceans, and other seafood: fresh, boiled, fried, or otherwise cooked
Poultry	All parts of chicken and turkey, including processed poultry meat
Red meat and products	Beef, veal, pork, lamb, goat meat, as well as all kinds of red meat products
Low-fat dairy	Low-fat milk, yogurt, and cheese
Full-fat dairy	Full-fat milk, yogurt, and cheese
Olive oil	Olive oil in salads or mixed dishes, including sweets
Alcoholic beverages	Wine, beer, and all other alcoholic beverages
Coffee	All types of coffee

diet, as this was depicted in the Mediterranean diet pyramid and the dietary guidelines for the Greek population [29,30], namely, dairy products (low-fat and full-fat dairy), fruits, vegetables, cereals (refined and whole-grain), potatoes, legumes, red meat and products, poultry, fish, and nuts. For the definition of the size of a serving for each food group, we used the definitions provided in the guidelines for the Greek population [29]. Frequency of olive oil use was also recorded, as well as the daily consumption of alcoholic beverages and coffee. Nonalcoholic beverages and coffee consumptions were recorded as milliliters per day, whereas for alcoholic beverages, alcohol consumption was measured in terms of daily ethanol intake (eg, one 100-mL wine glass as 12% ethanol concentration).

For the assessment of low-energy reporting, the ratio of the energy intake to the basal metabolic rate (EI/BMR) was determined for each individual. The BMR was estimated using the Schofield equations for the prediction of BMR [31], adopted by the 2004 Food and Agriculture Organization of the United Nations/World Health Organization/United Nations University report [32]. Participants with EI/BMR <1.04 were classified as "low-energy reporters" based on the cutoff limits developed by Goldberg et al [31]. "Normal-energy reporters" or non–low-energy reporters were participants with EI/BMR ≥1.04.

2.4. Physical activity assessment

Assessment of physical activity was performed through a brief self-reported questionnaire (the Harokopio Physical Activity Questionnaire), which collects the previous week's self-reported physical activity [33]. The Harokopio Physical Activity Questionnaire examines the time spent in light-, moderate-, and high-intensity activities and also requires sleeping to be recorded. The questionnaire is based on the metabolic equivalents of all activities of the previous week, including activities at work, leisure time, and rest or sleep, thus allowing the prediction of mean physical activity level.

2.5. Assessment of plasma adiponectin concentrations

Blood samples were drawn after a 12-hour fast between 8:30 AM and 10:30 AM, and plasma was immediately frozen in −80°C until biochemical analysis. Adiponectin concentrations (in μg/mL) were measured by commercially available radioimmunoassay kits (Linco Research, St Louis, MO). The sensitivity of the assay was 2 ng/mL. The interassay and the intraassay coefficients of variation were less than 9% for adiponectin.

2.6. Statistical analysis

Continuous variables are presented as mean values ± standard deviations; and categorical variables, as absolute frequencies. The Kolmogorov-Smirnov test was used to test for the normality of distributions. To obtain food patterns, the principal components analysis was used [34,35]. The food groups or beverages used in all analyses are presented in Table 1. The orthogonal rotation (varimax option) was used to derive optimal, noncorrelated components (food patterns). To decide the number of components retained, the proportion of the variance in consumption explained by the components was used. In particular, 10 of the 15 components were retained because they explained 82% of the variance in consumption. Based on the principle that the component scores are interpreted similarly to correlation coefficients (thus, higher absolute values indicate that a given food variable contributes more to the construction of the component), the food components (patterns) were named according to scores of the foods that correlated most with the component (scores >0.3). Multiple regression analysis

Table 2
Descriptive characteristics of the study population (n = 196)

Variable	Mean ± SD or frequencies
Age (y)	47.2 ± 12.1
Current smokers (%)	43.9
Menstrual status (%)[a]	
Premenopausal	51.0
Perimenopausal	8.2
Postmenopausal	40.8
BMI (kg/m^2)	27.6 ± 5.3
Percentage of body fat	39.5 ± 7.5
Obesity (%)	28.1
Waist-to-hip ratio	0.78 ± 0.06
Plasma adiponectin (μg/mL)	16.8 ± 6.8
Physical activity level	1.47 ± 0.20

[a] Age range for premenopausal women was 18 to 53 years; for perimenopausal, 43 to 57 years; and for postmenopausal, 46 to 74 years.

was applied to evaluate the explanatory ability of the principal components extracted in relation to adiponectin levels after adjusting for potential confounders. The results from the regression models are presented as standardized β coefficients. All reported P values were based on 2-tailed tests and compared with a significance level of 5%. Statistical Package for Social Sciences software (version 13.0; SPSS 2003, Chicago, IL) was used for all the statistical calculations.

3. Results

The descriptive characteristics of the study participants are presented in Table 2. Mean BMI was 27.6 ± 5.2 kg/m^2; percentage of body fat, 39.5 ± 7.5; and adiponectin levels, 16.8 ± 6.8 μg/mL. To recognize food patterns for the whole group, principal component analysis was performed and 10 components were extracted, explaining 82% of the total variance in food intake. Table 3 represents the food groups or items with score coefficients ≥0.3 for each component. In particular, we identified a pattern characterized by high consumption of whole-grain cereals and low-fat dairy products and low consumption of refined cereals (PC1); a pattern high in vegetables, fruits, and olive oil (PC2); a pattern low in low-fat dairy products and high in coffee (PC3); a pattern loaded with full-fat dairy products (PC4); a pattern high in poultry and low in red meat and its products (PC5); a pattern high in fish and low in red meat and its products (PC6); a pattern representing a high consumption of alcoholic beverages (PC7); a pattern high in nuts and low in fruits (PC8); a pattern characteristic of legumes intake (PC9); and one characteristic of potatoes intake (PC10). The first food component (PC1) was the most dominant food pattern, explaining 13.8% of the variance of food intake.

Table 3
Principal components and respective scoring coefficients for the food groups or foods recorded to be consumed by the study participants

			Explained variance (%)
PC1	Whole-grain cereals (0.86) Low-fat dairy (0.32)	Refined cereals (−0.81)	13.8
PC2	Vegetables (0.87) Fruits (0.38) Olive oil (0.68)		11.5
PC3	Coffee (0.78)	Low-fat dairy (−0.73)	10.0
PC4	Full-fat dairy (0.90)		7.9
PC5	Poultry (0.87)	Red meat and products (−0.55)	7.7
PC6	Fish (0.92)	Red meat and products (−0.45)	7.2
PC7	Alcoholic beverages (0.93)		6.9
PC8	Nuts (0.88)	Fruits (−0.52)	6.4
PC9	Legumes (0.95)		5.4
PC10	Potatoes (0.99)		5.0

Table 4
Results of the multiple regression model evaluating associations between participant characteristics and circulating adiponectin levels

Dependent variable: circulating adiponectin levels	Standardized β coefficient	P
Age (y)	.35	.01
Waist (cm)	−.46	.03
Current smoker (yes/no)	−.21	.02
PC1 [a]	.18	.03

Potential confounders, such as BMI, menopausal status, physical activity level, and being low-energy reporter, as well as all the other components extracted from the principal component analysis, were also included in the model; but they did not significantly influence associations with circulating adiponectin levels.

[a] PC1 was characterized by high intake of nonrefined cereals and low-fat dairy products, as well as low intake of refined cereals.

Bivariate analysis with plasma adiponectin levels and all the extracted components revealed a positive significant association only with PC1 (r = 0.15, P = .04). To evaluate potential associations between adiponectin levels and the food components, after controlling for potential confounders (age, BMI, waist circumference, smoking status, physical activity level, menopausal status, and low-energy reporting), multiple regression analysis was performed. Additional multiple regression analyses were performed by including all the 10 principal components in the model. Results are presented in Table 4. Age, waist circumference, and smoking status were significant predictors of adiponectin levels. The positive association between PC1 and plasma adiponectin levels detected in the bivariate analysis remained statistically significant after adjusting for the aforementioned confounders (standardized β coefficient = 0.18, P = .03). Furthermore, a marginally significant association was also found with PC7, a pattern characteristic of alcoholic beverages consumption, in the multivariate model including the above-mentioned confounders and the other principal components (standardized β coefficient = 0.14, P = .10). The results did not change when data were stratified by obesity status.

4. Discussion

Ten food components were identified herein using the principal component analysis for the identification of dietary patterns in this group of apparently healthy adult Mediterranean women. The first food component (PC1), representing the high consumption of whole-grain cereals and low-fat dairy products as well as the low consumption of refined cereals, was found to be positively associated with plasma adiponectin levels. The observed association remained significant after controlling for potential confounders, including age, anthropometric and potential lifestyle confounders, such as smoking status, physical activity, and low-energy reporting.

Our data indicate that a dietary pattern characterized by high consumption of whole-grain cereals was modestly

positively, yet significantly, related to circulating adiponectin concentrations. These findings are in line with those previously reported for adiponectin levels and cereal fiber, whole-grain cereals, or glycemic index [20-22]. Prior epidemiologic studies have demonstrated that whole-grain cereals have been inversely associated with biomarkers of risk for diabetes and cardiovascular disease, namely, insulin sensitivity, fasting insulin, and C-peptide levels [36-39]. Compared with refined cereals, whole-grain cereals contain a variety of nutritive and nonnutritive compounds, including fiber, minerals (magnesium, potassium, zinc, iron, manganese, selenium), vitamins (B complex vitamins and E), phenolic compounds, and phytoestrogens. Their beneficial role in health outcomes has thus been attributed to the biological roles of one or more of these compounds. For example, most of the association between whole-grain cereals and insulin sensitivity has been explained by their dietary fiber and magnesium content [36]. On the basis of the data presented herein, we could hypothesize that the beneficial effect of whole-grain cereals on risk for diabetes may be mediated by their effect on circulating adiponectin levels. Furthermore, potential beneficial effect on adiponectin levels could induce health benefits not only on insulin and glucose metabolism, but also on changes on inflammatory markers [40] or other adipokines not studied herein.

Interestingly, PC1 is also characterized by high intake of low-fat dairy products. Although no evidence exists so far with regard to adiponectin and low-fat dairy intake, available literature supports a negative association between dairy consumption and insulin resistance and prevalence of metabolic syndrome [41-43] and/or risk for type 2 diabetes mellitus [44,45]. These findings have been attributed to the nutrient content of dairy products. Ma et al [46] found that calcium and magnesium were negatively associated with insulin resistance, whereas Liu et al [44] reported that the inverse association between dairy products and diabetes could mainly be attributed to low-fat dairy products, even after controlling for calcium and magnesium intake. They also speculated that dairy products could possibly influence glucose tolerance primarily through their insulinotropic effect, rather than their relatively low glycemic index. They further commented that saturated fat content may mitigate this potential benefit, a hypothesis that may possibly explain the null association with high-fat dairy foods in their study or the discriminant inclusion of only low-fat dairy products in the beneficial PC1 of the present study.

Finally, we found that another pattern representing mainly alcoholic beverage consumption is marginally associated with adiponectin levels after adjustment for potential confounders. Similar results have also been reported in a prospective and cross-sectional evaluation of 987 diabetic women from the Nurses Health Study [22], where a trend toward significance for a quadratic association between alcohol and plasma adiponectin levels was observed. In our sample, similarly to other female populations [22], alcoholic beverage intake was small to moderate, well below the daily alcohol consumption of 40 g that has been reported to induce increases in adiponectin levels in short-term intervention trials [24-26].

Our study has both strengths and limitations. The strengths of the study include the blood sample collection just after the completion of dietary records, use of state-of-the-art methodology to measure adiponectin, and statistical control for potential confounders. Diet records, as a nutritional assessment method, have the potential to provide quantitatively accurate information on food consumed during the recording period. They are often regarded as "gold standard" or "reference method" against which other dietary assessment tools have been validated [47]. Among their advantages is that they rely to a lesser extent on memory recalls, so that the problem of omission is eliminated, and that they are more likely to provide a detailed description of both the type and the portions of the foods eaten. For the present analysis, low-energy reporting was evaluated and considered as a potential confounder of food intake; in multivariate models, however, it was not found to be significantly related to adiponectin levels, suggesting that no bias related to low-energy reporting was likely to be present. Finally, the focus on healthy individuals offers the advantage of minimizing the potential effect of disease and/or related treatments on dietary intake.

Among the limitations of this work is its cross-sectional, observational design, which cannot establish a cause-effect relationship and/or elucidate underlying mechanisms. Although we carefully adjusted for known and potential confounders, residual confounding remains a possibility. Given the relatively small study size and the cross-sectional study design, the hypotheses described herein need to be assessed through future prospective clinical studies. Furthermore, the use of principal component analysis, a multivariate statistical technique for identifying patterns of food consumption, has been criticized for the subjective nature of food intake classification before the analysis, the choice of the number of factors to be extracted, and the labeling of the factors [48]. Similar to most data reduction analyses, some arbitrary decisions must be inevitably made [49]. McCann et al [50], however, found that similar patterns could be obtained regardless of food intake categories, although the classification scheme did affect the percentage of variance in food intake explained by the extracted factors. We have conducted the analysis using a standard methodology [35]. Despite the fact that the goal of principal component analysis should be to explain 80% to 90% of the variance in food intake, in most of the studies so far, interpretable identified factors explained a total variance of no more than 37% [50]. We decided to present all factors explaining approximately 80% of the variance in dietary intake to account for as much of the variability as possible (comprehensiveness of the data). All individuals had a loading on every factor, the level of which was further explored for potential associations with adiponectin levels.

In conclusion, a dietary pattern characterized by the high consumption of nonrefined cereals and low-fat dairy products as well as the low consumption of refined cereals was found to be modestly positively associated with adiponectin levels in a group of apparently healthy adult Mediterranean women. Future studies are needed to elucidate underlying mechanisms as well as whether this favorable association could be further translated into reduced risk for diabetes or other chronic diseases.

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References

[1] Lago F, Dieguez C, Gomez-Reino J, et al. The emerging role of adipokines as mediators of inflammation and immune responses. Cytokine Growth Factor Rev 2007;18:313-25.
[2] Yamauchi T, Kamon J, Minokoshi Y, et al. Adiponectin stimulates glucose utilization and fatty-acid oxidation by activating AMP-activated protein kinase. Nat Med 2002;8:1288-95.
[3] Ouchi N, Kihara S, Arita Y, et al. Adipocyte-derived plasma protein, adiponectin, suppresses lipid accumulation and class A scavenger receptor expression in human monocyte-derived macrophages. Circulation 2001;103:1057-63.
[4] Mantzoros CS, Li T, Manson JE, et al. Circulating adiponectin levels are associated with better glycemic control, more favorable lipid profile, and reduced inflammation in women with type 2 diabetes. J Clin Endocrinol Metab 2005;90:4542-8.
[5] Schulze MB, Rimm EB, Shai I, et al. Relationship between adiponectin and glycemic control, blood lipids, and inflammatory markers in men with type 2 diabetes. Diabetes Care 2004;27:1680-7.
[6] Cnop M, Havel PJ, Utzschneider KM, et al. Relationship of adiponectin to body fat distribution, insulin sensitivity and plasma lipoproteins: evidence for independent roles of age and sex. Diabetologia 2003;46:459-69.
[7] Matsubara M, Maruoka S, Katayose S. Decreased plasma adiponectin concentrations in women with dyslipidemia. J Clin Endocrinol Metab 2002;87:2764-9.
[8] Ryo M, Nakamura T, Kihara S, et al. Adiponectin as a biomarker of the metabolic syndrome. Circ J 2004;68:975-81.
[9] Spranger J, Kroke A, Mohlig M, et al. Adiponectin and protection against type 2 diabetes mellitus. Lancet 2003;361:226-8.
[10] Choi KM, Lee J, Lee KW, et al. Serum adiponectin concentrations predict the developments of type 2 diabetes and the metabolic syndrome in elderly Koreans. Clin Endocrinol (Oxf) 2004;61:75-80.
[11] Polak J, Kovacova Z, Jacek M, et al. An increase in plasma adiponectin multimeric complexes follows hypocaloric diet-induced weight loss in obese and overweight pre-menopausal women. Clin Sci (Lond) 2007; 112:557-65.
[12] Behre CJ, Gummesson A, Jernas M, et al. Dissociation between adipose tissue expression and serum levels of adiponectin during and after diet-induced weight loss in obese subjects with and without the metabolic syndrome. Metabolism 2007;56:1022-8.
[13] Yang WS, Lee WJ, Funahashi T, et al. Weight reduction increases plasma levels of an adipose-derived anti-inflammatory protein, adiponectin. J Clin Endocrinol Metab 2001;86:3815-9.
[14] Faraj M, Havel PJ, Phelis S, et al. Plasma acylation-stimulating protein, adiponectin, leptin, and ghrelin before and after weight loss induced by gastric bypass surgery in morbidly obese subjects. J Clin Endocrinol Metab 2003;88:1594-602.
[15] Mazzali G, Di Francesco V, Zoico E, et al. Interrelations between fat distribution, muscle lipid content, adipocytokines, and insulin resistance: effect of moderate weight loss in older women. Am J Clin Nutr 2006;84:1193-9.
[16] Ryan AS, Nicklas BJ, Berman DM, et al. Adiponectin levels do not change with moderate dietary induced weight loss and exercise in obese postmenopausal women. Int J Obes Relat Metab Disord 2003; 27:1066-71.
[17] Wolfe BE, Jimerson DC, Orlova C, et al. Effect of dieting on plasma leptin, soluble leptin receptor, adiponectin and resistin levels in healthy volunteers. Clin Endocrinol (Oxf) 2004;61:332-8.
[18] de Luis DA, Aller R, Izaola O, et al. Effects of a low-fat versus a low-carbohydrate diet on adipocytokines in obese adults. Horm Res 2007; 67:296-300.
[19] Yannakoulia M, Yiannakouris N, Bluher S, et al. Body fat mass and macronutrient intake in relation to circulating soluble leptin receptor, free leptin index, adiponectin, and resistin concentrations in healthy humans. J Clin Endocrinol Metab 2003;88:1730-6.
[20] Qi L, Meigs JB, Liu S, et al. Dietary fibers and glycemic load, obesity, and plasma adiponectin levels in women with type 2 diabetes. Diabetes Care 2006;29:1501-5.
[21] Qi L, Rimm E, Liu S, et al. Dietary glycemic index, glycemic load, cereal fiber, and plasma adiponectin concentration in diabetic men. Diabetes Care 2005;28:1022-8.
[22] Mantzoros CS, Williams CJ, Manson JE, et al. Adherence to the Mediterranean dietary pattern is positively associated with plasma adiponectin concentrations in diabetic women. Am J Clin Nutr 2006; 84:328-35.
[23] Pischon T, Girman CJ, Rifai N, et al. Association between dietary factors and plasma adiponectin concentrations in men. Am J Clin Nutr 2005;81:780-6.
[24] Sierksma A, Patel H, Ouchi N, et al. Effect of moderate alcohol consumption on adiponectin, tumor necrosis factor–alpha, and insulin sensitivity. Diabetes Care 2004;27:184-9.
[25] Beulens JW, de Zoete EC, Kok FJ, et al. Effect of moderate alcohol consumption on adipokines and insulin sensitivity in lean and overweight men: a diet intervention study. Eur J Clin Nutr 2007.
[26] Beulens JW, van Beers RM, Stolk RP, et al. The effect of moderate alcohol consumption on fat distribution and adipocytokines. Obesity (Silver Spring) 2006;14:60-6.
[27] Hu FB. Dietary pattern analysis: a new direction in nutritional epidemiology. Curr Opin Lipidol 2002;13:3-9.
[28] Willett WC. Nutritional epidemiology. New York: Oxford University Press; 1990.
[29] Supreme Scientific Health Council, Ministry of Health and Welfare. Dietary guidelines for adults in Greece. Archives of Hellenic Medicine 1999;16:516-24.
[30] Willett WC, Sacks F, Trichopoulou A, et al. Mediterranean diet pyramid: a cultural model for healthy eating. Am J Clin Nutr 1995;61: 1402S-6S.
[31] Goldberg GR, Black AE, Jebb SA, et al. Critical evaluation of energy intake data using fundamental principles of energy physiology: 1. Derivation of cut-off limits to identify under-recording. Eur J Clin Nutr 1991;45:569-81.
[32] FAO/WHO/UNU. Human energy requirements: report of a joint FAO/ WHO/UNU Expert Consultation. Rome: FAO; 2004.
[33] Kollia M, Kavouras SA, Gioxari A, et al. Development, validity and reliability of the Harokopio Physical Activity Questionnaire in Greek adults. 8th Panhellenic Congress on Nutrition and Dietetics. Abstract Book. Athens; 2006. p. 130-1.
[34] Mardia KV, Kent JT, Bibby JM. Multivariate analysis. 1st ed. New York, NY: Academic Press; 1979.
[35] Cureton E, D'Agostino R. Factor analysis: an applied approach. Hillside, NJ: Lawrence Erlbaum Associates; 1983.
[36] Liese AD, Roach AK, Sparks KC, et al. Whole-grain intake and insulin sensitivity: the Insulin Resistance Atherosclerosis Study. Am J Clin Nutr 2003;78:965-71.

[37] Jensen MK, Koh-Banerjee P, Franz M, et al. Whole grains, bran, and germ in relation to homocysteine and markers of glycemic control, lipids, and inflammation 1. Am J Clin Nutr 2006;83: 275-83.
[38] McKeown NM, Meigs JB, Liu S, et al. Whole-grain intake is favorably associated with metabolic risk factors for type 2 diabetes and cardiovascular disease in the Framingham Offspring Study. Am J Clin Nutr 2002;76:390-8.
[39] Pereira MA, Jacobs Jr DR, Pins JJ, et al. Effect of whole grains on insulin sensitivity in overweight hyperinsulinemic adults. Am J Clin Nutr 2002;75:848-55.
[40] Shetty GK, Economides PA, Horton ES, et al. Circulating adiponectin and resistin levels in relation to metabolic factors, inflammatory markers, and vascular reactivity in diabetic patients and subjects at risk for diabetes. Diabetes Care 2004;27:2450-7.
[41] Pereira MA, Jacobs Jr DR, Van Horn L, et al. Dairy consumption, obesity, and the insulin resistance syndrome in young adults: the CARDIA Study. Jama 2002;287:2081-9.
[42] Elwood PC, Pickering JE, Fehily AM. Milk and dairy consumption, diabetes and the metabolic syndrome: the Caerphilly prospective study. J Epidemiol Community Health 2007;61:695-8.
[43] Mennen LI, Lafay L, Feskens EJM, et al. Possible protective effect of bread and dairy products on the risk of the metabolic syndrome. Nutr Res 2000;20:335-47.
[44] Liu S, Choi HK, Ford E, et al. A prospective study of dairy intake and the risk of type 2 diabetes in women. Diabetes Care 2006;29:1579-84.
[45] Choi HK, Willett WC, Stampfer MJ, et al. Dairy consumption and risk of type 2 diabetes mellitus in men: a prospective study. Arch Intern Med 2005;165:997-1003.
[46] Ma B, Lawson AB, Liese AD, et al. Dairy, magnesium, and calcium intake in relation to insulin sensitivity: approaches to modeling a dose-dependent association. Am J Epidemiol 2006;164:449-58.
[47] Thompson FE, Byers T. Dietary assessment resource manual. J Nutr 1994;124:2245-317.
[48] Martinez ME, Marshall JR, Sechrest L. Invited commentary: factor analysis and the search for objectivity. Am J Epidemiol 1998;148:17-9.
[49] Slattery ML, Boucher KM. The senior authors' response: factor analysis as a tool for evaluating eating patterns. Am J Epidemiol 1998;148:20-1.
[50] McCann SE, Marshall JR, Brasure JR, et al. Analysis of patterns of food intake in nutritional epidemiology: food classification in principal components analysis and the subsequent impact on estimates for endometrial cancer. Public Health Nutr 2001;4:989-97.