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Original article

# Modifications of serum levels of omentin-1 and other cardiovascular risk factors following weight loss secondary to a Mediterranean hypocaloric diet

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SUMMARY

*Background & aims:* Omentin-1 might play a role on insulin resistance, dyslipidemia and obesity. The aim of this investigation was to evaluate the influence of weight loss on omentin-1 concentrations after a hypocaloric diet with Mediterranean pattern.

*Methods:* A Caucasian sample of 67 obese patients was analyzed before and after 3 months on a hypocaloric diet. Anthropometric parameters, blood pressure, fasting blood glucose, C-reactive protein (CRP), fasting insulin, insulin resistance (HOMA-IR), lipid concentrations and omentin-1 were measured.

*Results:* Sixty-seven obese subjects were enrolled in the study. The mean age was 48.3 ± 8.0 years (range: 25–66) and the mean BMI 34.5 ± 4.8 kg/m$^2$ (range: 30.2–40.8). Gender distribution was 50 females (74.6%) and 17 males (25.4%). After dietary intervention and in males and females; body mass index, weight, fat mass, waist circumference, blood pressure, glucose, LDL cholesterol, insulin and HOMA-IR decreased. Omentin-1 levels increase after dietary intervention (males vs females) (delta basal vs 3 months: 10.0 ± 3.8 ng/dl: p = 0.01 vs 9.9 ± 4.1 ng/dl; p = 0.03). In the multiple regression analysis adjusted for age and sex; BMI and insulin remained independently associated with baseline and post-treatment levels of omentin-1.

*Conclusions:* Our investigation showed a significant increase in omentin-1 levels after weight loss secondary to a hypocaloric diet with a Mediterranean pattern.



## 1. Introduction

Obesity is a major global health problem with massive proportions [1]. This disease affects all areas of the body leading to a metabolic dysfunction. The adipose tissue is an energy storage organ, but the role of this tissue as an endocrine organ has emerged [2]. All the cells of the adipose tissue such as adipocytes, macrophages, mast cell and stromal vascular cells are known to secrete a large number of proteins with many functions called adipokines. The adipokines are implied in the pathogenesis of insulin resistance, hypertension, dyslipidemia and cardiovascular disease [3]. Omentin is one of these adipokines. This molecule is a 40 kDa fat depot-specific adipokine, which has been isolated from visceral omental adipocytes by Yang et al. [17]. There are two homologous isoforms of omentin, omentin-1 and omentin-2; moreover, omentin-1 is the major circulating form. Circulating omentin-1 correlated negatively with anthropometric parameters such as weight, body mass index (BMI), waist circumference and biochemical parameters (fasting insulin, HOMA-IR and leptin) and positively with high density lipoprotein cholesterol (HDL-C) and adiponectin levels [5].

Therefore, some strategies have been investigated in order to reduce body weight secondary to a negative energy balance, such as a reduction of energy intake, drugs or surgical treatments [6,7]. This weight loss is considered the main intervention to reduce the levels of pro-inflammatory adipokines and to increase circulating anti-inflammatory adipokines [8]. Some studies have evaluated the effect of bariatric surgery [9,10] and dietary interventions [11,12] on serum concentrations of omentin-1. Contradictory results have been reported in the literature on omentin-1 changes after weight loss with these strategies.

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As far as we know, there are no studies evaluating the effect of a hypocaloric diet with a Mediterranean pattern on omentin-1 levels after a weight reduction intervention. The aim of our investigation was to evaluate the influence of weight loss on omentin-1 concentrations and other parameters after a hypocaloric diet with Mediterranean pattern.

## 2. Materials and methods

### 2.1. Subjects and procedure

All participants were Caucasians females and males with obesity, who accepted the lifestyle modification programs for weight loss in our Institution. The recruitment of patients was a consecutive method of sampling among subjects send from Primary Care Physicians with obesity. A total of 67 patients were recruited and all participants provided informed consent. This protocol was conducted according to the guidelines laid down in the Declaration of Helsinki, the local ethics committee (HCUVA) approved all procedures involving patients and patient data were codified to guarantee anonymity.

Participants were included for the following standards; body mass index ranged from 30 kg/m$^2$ to 45 kg/m$^2$, absence of a diet during the 6 months prior to the study and an adult age ranged from 20 to 65 years. Exclusion criteria included without the history of cardiovascular disease, psychiatric disorders, diabetes mellitus, pregnancy, use of a weight-loss medication, as well as the use of drugs such as glucocorticoids, angiotensin receptor blockers, angiotensin converting enzyme inhibitors, statins and other lipid drugs.

### 2.2. Procedure

All biochemical and anthropometric dates were obtained within the start of the trial and repeated after 3 months of intervention. Anthropometric parameters (weight, height, and total fat mass by bioimpedance) and blood pressure were measured in both times. Venous blood specimens (10 ml) were collected in EDTA-treated tubes after a 12 h fast. Fasting glucose, C-reactive protein (CRP), insulin, insulin resistance as homeostasis model assessment (HOMA-IR), lipid profile determined by total cholesterol, LDL-cholesterol, HDL-cholesterol, plasma triglycerides concentration and omentin-1 were measured.

### 2.3. Dietary intervention

Diet was designed to provide about 500 kcal/day less than individually estimated total energy expenditure during 12 weeks. The target macronutrient composition with a Mediterranean pattern (fresh vegetables, fruits, grains, dairy, fish and olive oil) was; 53% of carbohydrates, 26% of lipids and 21% of proteins. Distribution of dietary fats was: 50.5% of monounsaturated fats, 38.6% of saturated fats and 11.9% of polyunsaturated fats. The distribution of caloric intake was 20% in breakfast, 30% in lunch, 30% in dinner, 10% in mid-morning and 10% in snack. All participants had an individual session (60 min with diet sheets and example menu plans) with the dietitian at the start of the trail to explain the diet. A dietitian assessed the adherence of this diet each 14 days with a telephone call in order to improve compliment of the calorie restriction and macronutrient distribution. All enrolled subjects received instruction to record their daily dietary intake for three days including a weekend day. Records were analyzed with a computer-based data evaluation system (Dietosource®; Geneva Switzerland). National composition food tables were used [13].

### 2.4. Biochemical assays

Plasma glucose levels were measured by using an automated glucose oxidase method (Glucose analyser 2, Beckman Instruments, Fullerton, California). Insulin was determined by RIA (RIA Diagnostic Corporation, Los Angeles, CA) with a sensitivity of 0.5 mUI/L (normal range 0.5–30 mUI/L) [14] and the homeostasis model assessment for insulin resistance (HOMA-IR) was calculated using this formula (fasting insulin × fasting glucose concentrations/22.5) [15]. Serum total cholesterol and triglyceride concentrations were measured by enzymatic colorimetric assay (Technicon Instruments, Ltd., New York, N.Y., USA), while HDL cholesterol was measured enzymatically in the supernatant after precipitation of other lipoproteins with dextran sulfate–magnesium. LDL cholesterol was calculated using Friedewald formula [16].

CRP was determined by immunoturbimetry (Roche Diagnostics GmbH, Mannheim, Germany), with a normal range of (0–7 mg/dl) and analytical sensitivity 0.5 mg/dl. Omentin-1 was determined by ELISA (Biovendor Laboratory, Inc., Brno, Czech Republic) (RD191100200R) with a sensitivity of 0.5 ng/ml, a normal range of 50–250 ng/ml [17] and a CV% 3.2%.

### 2.5. Anthropometric measurements and blood pressure

Body weight was assessed to an accuracy of 50 g and body mass index computed as body weight in kg/(height in m$^2$). Waist and hip circumferences to derive waist-to hip ratio (WHR) were measured, too. Bioimpedance was used to determine total body fat mass with an accuracy of 50 g [18] (Akern, EFG, It). Blood pressure was determined three times after a 5 min rest with a random zero mercury sphygmomanometer, and averaged (Omrom, LA, CA).

### 2.6. Statistical analysis

Sample size was calculated to detect differences over 5 ng/ml in omentin levels after weight loss with 90% power and 5% significance (n = 65). The Shapiro–Wilk test was used to determine normality. The results were expressed as mean ± standard deviation. Numerical variables with normal distribution were studied with a two-tailed Student's t-test. Non-parametric variables were evaluated with the Mann–Whitney U test. Categorical variables were analyzed with the chi-square test, with Yates correction as necessary, and Fisher's test. The statistical analysis to evaluate the omentin-1 and diet interaction was an univariate ANCOVA. Correlation analysis was realized with Pearson and Spearmen test as needed. Multiple regression analysis (stepwise method) was used to analyze relationship of omentin-1 concentrations as dependent variable. A p-value <0.05 was considered significant. SPSS version 15.0 has been used to realize statistical analysis.

## 3. Results

Sixty-seven obese subjects were enrolled in the study. The mean age was 48.3 ± 8.0 years (range: 25–66) and the mean body mass index (BMI) 34.5 ± 4.8 kg/m$^2$ (range: 30.2–40.8). Sex distribution was 50 females (74.6%) and 17 males (25.4%). Age was similar in males; 47.8 ± 8.1 years than females 48.6 ± 13.8 years. BMI was similar in males 34.9 ± 4.3 kg/m$^2$ than females 34.4 ± 5.1 kg/m$^2$, too.

After 3 months of dietary intervention, all patients achieved dietary recommendations in both gender groups. Basal and final caloric intakes were higher in male than female; (male: basal 2019.2 ± 418.9 vs 3 months 1516.2 ± 313.1 kcal/day vs female: basal 1799.9 ± 323.0 vs 3 months 1320.1 ± 282.9 kcal/day; p = 0.01). Final percentage of macronutrient intakes were similar in both gender

groups; from carbohydrates (male: basal 52.1% vs 3 months 50.5%: p = 0.53) and (female: basal 52.3% vs 3 months 50.8%: p = 0.12). Percentage of dietary fats was similar (male: basal 24.8% vs 3 months 25.1%: p = 077) and (female: basal 24.3% vs 3 months 24.9%: p = 0.33). Finally, percentage of proteins did not show differences (male: basal 23.1% vs 24.4%: p = 056) and (female: 24.4% vs 24.3%: p = 0.27).

After dietary intervention BMI, weight, fat mass, waist circumference and systolic blood pressure decreased in a statistical way from basal values (Table 1). After dietary intervention, fasting glucose, total cholesterol, LDL cholesterol, insulin levels and HOMA-IR decreased in a statistical way from basal values (Table 2). Omentin-1 levels increase in males and females, too.

Correlation analysis showed an association of omentin-1 basal levels with (age, r = 0.265; p = 0.009), (BMI, r = −0.275; p = 0.01) and (insulin, r = −0.431; p = 0.001). These association were observed in each gender; (male age, r = 0.257; p = 0.01 and female age, r = 0.369; p = 0.007), (male BMI, r = −0.257; p = 0.04 and female BMI, r = −0.391; p = 0.01) and (male insulin, r = −0.403; p = 0.01 and female insulin, r = −0.513; p = 0.007).

Post weight loss omentin-1 levels showed a significate correlation with (age, r = 0.212; p = 0.03), (BMI, r = −0.290; p = 0.02) and (insulin, r = −0.369; p = 0.01). These association were observed in each gender; (male age, r = 0.191; p = 0.01 and female age, r = 0.269; p = 0.009), (male BMI, r = −0.334; p = 0.01 and female BMI, r = −0.201; p = 0.04) and (male insulin, r = −0.313; p = 0.01 and female insulin, r = −0.453; p = 0.02). A correlation analysis between changes of omentin-1 levels and changes of other parameters was realized; (Delta BMI, r = −0.280; p = 0.03) and (Delta insulin, r = −0.377; p = 0.01). These association were observed in each gender; (male Delta BMI, r = −0.301; p = 0.01 and female Delta BMI, r = −0.286; p = 0.03) and (male Delta insulin, r = −0.309; p = 0.02 and female Delta insulin, r = −0.478; p = 0.01).

In the multiple regression analysis adjusted by age and sex; BMI kg/m$^2$ (Beta −0.307: CI95% −3.51 to 0.16) and insulin UI/L (Beta −0.26: CI95% −4.31 to 0.06) remained associated with basal omentin-1 levels as dependent variable. The second regression model with post-treatment omentin-1 levels as dependent variable showed a statistical association with BMI kg/m$^2$ (Beta −0.117: CI95% −4.61 to 0.03) and insulin UI/L (Beta −0.21: CI95% −4.49 to 0.07).

## 4. Discussion

Our study shows a significant increase in circulating omentin-1 levels, after weight loss due to a hypocaloric diet with Mediterranean patterns and its negative relationship with body mass index and insulin levels. We decided to use a hypocaloric Mediterranean diet because we work in an area in which it is the dietary pattern usually used and this fact ensures a greater follow-up of the diets by the patients studied.

In our study, circulating omentin-1 was correlated with body mass index in males and females, and no relationship was detected with either waist circumference or fat mass. In some studies [11], a correlation with waist circumference in males and no correlation in females was found. We didn't detect either correlation with visceral fat or sexual dimorphism. Some authors [19] found higher omentin-1 concentration in females than males. Moreover, Moreno-Navarete et al. [11] reported higher omentin-1 levels in males than females. Perhaps the differences found in the literature are due to different hormonal status of the evaluated females [20]. The exact factors contributing to inverse relationship of omentin-1 levels and adiposity remain to be determined. Perhaps, increased insulin levels found in subjects with obesity [21] might be an important contributor preceding decreased omentin-1 levels as described in patients with anorexia nervosa [22].

To our best knowledge, this is the first study to evaluate the effect of a hypocaloric diet with a Mediterranean pattern on omentin-1 levels, a dietary pattern that has shown multiple health benefits. In a previous study, Moreno-Navarrete et al. [11] reported that omentin-1 levels increase after weight loss-induced by prescription of a standard hypocaloric diet providing a daily energy deficit of 500–1000 kcal per day during four months. The hypocaloric diet of this study supplied 16%, 30% and 54% of energy requirements in the form of protein, fat and carbohydrates, respectively. In this previous study [11] the effect on insulin levels was similar to ours, with greater weight loss.

In a second study [10], a very low caloric diet during 2 weeks had not significant effect on omentin-1 levels. Moreover, omentin-1 levels increased 6 months after laparoscopy sleeve gastrectomy and a maintained increase of these levels remained during 2-year follow up [10]. These differences with our results can be due to a longer duration of dietary intervention in our study. In this previous study [10], levels of omentin-1 were inversely correlated with BMI, serum insulin, LDL-cholesterol, CRP and triglycerides. Moreover, multiple regression analysis performed in above-mentioned study didn't identify a statistical significant independent predictor of serum omentin-1 levels as our study (insulin and body mass index). Lesna et al. [12] reported that omentin-1 levels were stable during an intervention with a hypocaloric diet (one month). Nevertheless, a significant increase in omentin-1 levels were detected for a further diabetic diet, during 11 months.

Perhaps these conflicting results found in the literature may be due to different types of intervention to lose weight and the final amount of weight loss. Thus, in a study [9] evaluating the effect of biliopancreatic derivation on circulating omentin-1 levels, an elevation of omentin-1 levels was detected at 24 h of surgery, which was maintained throughout the follow-up period one year (59% of patients). Moreover, another group of patients after the initial elevation of omentin-1 levels at 24 h of surgery, showed a decrease that was maintained at one year of follow up (18% of patients). In a recent study [23], omentin-1 remained unchanged after

**Table 1**
Changes in anthropometric parameters and blood pressure (Average ± SD).

| | Total group n = 67 | | | Males n = 17 | | | Females n = 50 | | |
|---|---|---|---|---|---|---|---|---|---|
| | Basal | 3 months | Delta | Basal | 3 months | Delta | Basal | 3 months | Delta |
| BMI | 34.5 ± 2.6 | 33.0 ± 2.1* | −1.4 ± 0.9″ | 34.6 ± 8.1 | 33.2 ± 5.0* | −1.2 ± 1.0 | 34.5 ± 6.4 | 33.3 ± 5.0* | −1.4 ± 1.0″ |
| Weight (kg) | 87.2 ± 15.6 | 84.9 ± 10.8* | −3.4 ± 1.1″ | 95.8 ± 12.1$ | 91.7 ± 16.1*$ | −4.1 ± 3.0 | 85.3 ± 15.1$ | 83.1 ± 8.1*$ | −3.2 ± 3.0″ |
| Fat mass (kg) | 36.1 ± 7.2 | 35.0 ± 6.1* | −1.1 ± 1.0″ | 28.3 ± 3.2 $ | 27.3 ± 4.0*$ | −1.0 ± 1.1 | 39.7 ± 4.1$ | 37.8 ± 3.0*$ | −1.1 ± 0.9″ |
| WC (cm) | 106.7 ± 6.0 | 103.5 ± 4.0* | −3.2 ± 1.8″ | 109.9 ± 9.1$ | 105.2 ± 5.2*$ | −4.7 ± 2.9 | 105.7 ± 4.1 $ | 103.0 ± 4.1*$ | −2.7 ± 2.3″ |
| SBP (mmHg) | 129.9 ± 12.8 | 125.3 ± 3.0* | −4.6 ± 1.0″ | 131.7 ± 5.6 | 125.1 ± 7.1* | −6.6 ± 2.1 | 130.3 ± 9.8 | 126.0 ± 5.0* | −4.1 ± 3.1″ |
| DBP (mmHg) | 81.7 ± 5.0 | 81.2 ± 4.3 | −0.5 ± 1.1 | 82.9 ± 5.0 | 80.9 ± 5.1 | −1.0 ± 0.8 | 82.8 ± 5.8 | 81.5 ± 7.1 | −1.3 ± 1.1 |

BMI: body mass index; DBP, diastolic blood pressure; SBP, systolic blood pressure; WC, waist circumference; *$P < 0.05$, in each group with basal value. $$P < 0.05$, between males and females. ″$P < 0.05$ Delta differences.

**Table 2**
Changes in biochemical parameters (Average ± SD).

| | All group n = 67 | | | Males n = 17 | | | Females n = 50 | | |
|---|---|---|---|---|---|---|---|---|---|
| | Basal | 3 months | Delta | Basal | 3 months | Delta | Basal | 3 months | Delta |
| Glucose (mg/dl) | 101.4 ± 14.0 | 98.1 ± 14.0* | −3.1 ± 2.6″ | 97.3 ± 9.1 | 94.1 ± 8.1* | 3.2 ± 2.8″ | 102.7 ± 8.0 | 99.6 ± 9.1* | 3.1 ± 2.1″ |
| Cholesterol total (mg/dl) | 215.5 ± 20.8 | 200.1 ± 30.1* | 15.3 ± 6.1″ | 215.8 ± 17.2 | 200.4 ± 15.2 | −15.4 ± 7.8″ | 217.9 ± 10.1 | 202.6 ± 13.1* | −15.3 ± 5.1″ |
| LDL-cholesterol (mg/dl) | 135.8 ± 34.1 | 122.0 ± 30.1* | 14.0 ± 5.8″ | 135.8 ± 7.1 | 121.8 ± 7.4* | −14.0 ± 5.8″ | 136.0 ± 12.1 | 122.1 ± 14.3* | −13.9 ± 6.1″ |
| HDL-cholesterol (mg/dl) | 54.5 ± 3.0 | 53.4 ± 2.4 | −1.1 ± 0.5 | 47.3 ± 7.0 | 49.3 ± 8.0 | 2.0 ± 0.9 | 56.1 ± 3.0 | 54.4 ± 4.1 | −1.7 ± 0.8 |
| Triglycerides (mg/dl) | 123.4 ± 34.1 | 119.9 ± 23.4 | −4.5 ± 5.8 | 139.3 ± 17.9 | 143.4 ± 17.1 | −4.1 ± 3.4 | 118.9 ± 22.1 | 112.7 ± 23.4 | −6.2 ± 5.7 |
| CRP (ng/dl) | 5.8 ± 1.8 | 6.1 ± 1.5 | 0.3 ± 0.1 | 5.1 ± 1.1 | 5.3 ± 1.2 | 0.2 ± 0.1 | 6.5 ± 2.1 | 6.7 ± 2.2 | 0.2 ± 0.09 |
| Insulin (mUI/l) | 14.7 ± 5.1 | 13.2 ± 4.8* | −1.4 ± 0.9″ | 16.4 ± 4.1 | 15.1 ± 6.0* | −1.3 ± 1.8″ | 13.5 ± 6.0 | 12.1 ± 4.6* | −1.4 ± 2.1″ |
| HOMA-IR | 3.4 ± 1.4 | 3.2 ± 1.3* | −0.2 ± 0.9″ | 3.9 ± 1.4 | 3.6 ± 1.1* | −0.3 ± 1.1″ | 3.3 ± 2.1 | 3.1 ± 1.7* | −0.2 ± 1.0″ |
| Omentin-1 ng/ml | 357.8 ± 55.4 | 366.7 ± 65.1* | 9.9 ± 4.0″ | 342.6 ± 66.1 | 352.6 ± 49.9* | 10.0 ± 3.8″ | 360.5 ± 61.4 | 370.4 ± 57.1* | 9.9 ± 4.1″ |

CRP: C reactive protein. HOMA-IR (homeostasis model assessment).; *$P < 0.05$, in each group with basal value. $$P < 0.05$, between males and females. ″$P < 0.05$ Delta differences.

54 weeks. Finally, Sdralis et al. [24] have evaluated the impact of omentectomy performed in patients with morbid obesity undergoing sleeve gastrectomy (SG). After 1 year postoperatively, omentin-1 levels increased significantly in the control group only with SG and decreased in the omentectomy group.

Other types of treatment have also been evaluated in the literature. Other intervention study has demonstrated the lack of effect of leptin infusion at pharmacological doses [25]. However, an increase in omentin-1 levels has been demonstrated in obese women with polycystic ovarian syndrome when treated with an 850 mg daily dose of metformin for 3 months [26].

The limitations of our study are that sample size was small and it was a heterogeneous sample in BMI and age, too. Second, the current correlations results cannot be interpreted as causative. Thirdly, the short time follow up may restricted the possibility to detect other long-term differences. Finally, our sample were a sample of adult obese patients and the age and the body mass index range could modulate the reported data. This sample was no necessarily representative of the general population.

The results from our design showed a significant increase in serum omentin-1 levels after weight loss secondary to a hypocaloric diet with a Mediterranean pattern. This increase may be beneficial for obese patients, improving cardiovascular parameters and it is necessary to evaluate the longer-term effects.

## Conflict of interest

The authors declare no conflicts of interest.
D.A de Luis designed the study an wrote the article.
R Aller wrote the article and realized statistical analysis.
O Izaola realized nutritional evaluation.
D Primo realized biochemical analysis.

## References


[1] Ng M, Fleming T, Robinson M, Thomson B, Graetz N, Margono C, et al. Global, regional, and national prevalence of overweight and obesity in children and adults during 1980–2013: a systematic analysis for the global burden of disease study 2013. Lancet 2014;384:766–81.
[2] Hamdy O. The role of adipose tissue as an endocrine gland. Curr Diab Rep 2005;5:317–9.
[3] Maury E, Brichard SM. Adipokine dysregulation, adipose tissue inflammation and metabolic syndrome. Mol Cell Endocrinol 2010;314:1–16.
[5] Ouchi N, Parker JL, Lugus JJ, Walsh K. Adipokines in inflammation and metabolic disease. Nat Rev Immunol 2011;11:85–97.
[6] Kushner RF. Weight loss strategies for treatment of obesity. Prog Cardiovasc Dis 2014;56:465–72.
[7] Abete I, Astrup A, Martinez JA, Thorsdottir I, Zulet MA. Obesity and the metabolic syndrome: role of different dietary macronutrient distribution patterns and specific nutritional components on weight loss and maintenance. Nutr Rev 2010;68:214–31.
[8] de Luis DA, Izaola O, Aller R, de la Fuente B, Bachiller R, Romero E. Effects of a high-protein/low carbohydrate versus a standard hypocaloric diet on adipocytokine levels and insulin resistance in obese patients along 9 months. J Diabetes Complicat 2015;29(7):950–4.
[9] Lapointe M, Poirier P, Martin J, Bastien M, Auclair A, Cianflone K. Omentin changes following bariatric surgery and predictive links with biomarkers for risk of cardiovascular disease. Cardiovasc Diabetol 2014 Aug 21;13:124.
[10] Urbanová M, Dostálová I, Trachta P, Drápalová J, Kaválková P, Haluzíková D, et al. Serum concentrations and subcutaneous adipose tissue mRNA expression of omentin in morbid obesity and type 2 diabetes mellitus the effect of very-low-calorie diet, physical activity and laparoscopic sleeve gastrectomy. Physiol Res 2014;63(2):207–18.
[11] Moreno-Navarrete JM, Catalán V, Ortega F, Gómez-Ambrosi J, Ricart W, Frühbeck G, et al. Circulating omentin concentration increases after weight loss. Nutr Metab (Lond) 2010 Apr 9;7:27.
[12] Lesná J, Tichá A, Hyšpler R, Musil F, Bláha V, Sobotka L, et al. Omentin-1 plasma levels and cholesterol metabolism in obese patients with diabetes mellitus type 1: impact of weight reduction. Nutr Diabetes 2015;5:e183.
[13] Mataix J, Mañas M. Tablas de composición de alimentos españoles. University of Granada; 2003.
[14] Duart MJ, Arroyo CO, Moreno JL. Validation of an insulin model for the reactions in RIA. Clin Chem Lab Med 2002;40:1161–7.
[15] Mathews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF. Homeostasis model assessment: insulin resistance and beta cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia 1985;28:412–4.
[16] Friedewald WT, Levy RJ, 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.
[17] Yang RZ, Lee MJ, Hu H, Pray J, Wu HB, Hansen BC, et al. Identification of omentin as a novel depot-specific adipokine in Human adipose tissue: possible role in modulating insulin action. Am J Physiol Endocrinol Metab 2006;290:E1253–61.
[18] Lukaski H, Johson PE. Assessment of fat-free mass using bioelectrical impedance measurements of the human body. Am J Clin Nutr 1985;41:810–7.
[19] Souza Batista CM, Yang RZ, Lee MJ, Glynn NM, Yu DZ, Pray J, et al. Omentin plasma levels and gene expression are decreased in obesity. Diabetes 2007;56:1655–61.
[20] Tan BK, Adya R, Farhatullah S, Lewandowski KC, O'Hare P, Lehnert H, et al. Omentin-1, a novel adipokine, is decreased in overweight insulin-resistant women with polycystic ovary syndrome. Ex vivo and in vivo regulation of omentin-1 by insulin and glucose. Diabetes 2008;57:801–8.
[21] Choi JH, Rhee EJ, Kim KH, Woo HY, Lee WY, Sung KC. Plasma omentin-1 levels are reduced in non-obese women with normal glucose tolerance and polycystic ovary syndrome. Eur J Endocrinol 2011;165:789–96.
[22] Guo LJ, Jiang TJ, Liao L, Liu H, He HB. Relationship between serum omentin-1 level and bone mineral density in girls with anorexia nervosa. J Endocrinol Investig 2013;36:190–4.
[23] Wroblewski E, Swidnicka-Siergiejko A, Hady HR, Luba M, Konopko M, Kurek K, et al. Variation in blood levels of hormones in obese patients following weight reduction induced by endoscopic and surgical bariatric therapies. Cytokine 2016 Jan;77:56–62.
[24] Sdralis E, Argentou M, Mead N, Kehagias I, Alexandridis T, Kalfarentzos F. A prospective randomized study comparing patients with morbid obesity submitted to sleeve gastrectomy with or without omentectomy. Obes Surg 2013 Jul;23(7):965–71.
[25] Hamnvik OP, Thakkar B, Chamberland J, Aronis K, Schneider B, Mantzoros CS. Omentin-1 levels are reduced by pharmacologic doses of leptin, but remain unaffected by energy deprivation and display no day-night variation. Int J Obes (Lond) 2015;39:260–4.
[26] Shaker M, Mashhadani ZI, Mehdi AA. Effect of treatment with metformin on omentin-1, ghrelin and other biochemical, clinical features in PCOS patients. Oman Med J 2010;25:289–93.