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Modest weight loss does not increase plasma adiponectin levels: effects of weight loss on C-reactive protein and DNA damage

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Abstract

Adiponectin has been reported to have antiatherogenic, antidiabetic properties and was proposed as an important mediator of insulin action and glucose metabolism. This study was conducted to determine weight loss effects on the plasma adiponectin levels and clinical parameters including oxidative DNA damage and C-reactive protein (CRP) in overweight-obese subjects. A total of 184 overweight-obese volunteers underwent a clinical intervention study consisting of −1255 kJ/d for 12 weeks. Anthropometic parameters, blood lipid profiles, glucose, insulin, CRP, and baseline DNA damage using comet assay and adiponectin concentrations were determined at baseline and at 12 weeks of intervention. The treatment induced modest weight loss averaged 4.75% of initial body weight. C-reactive protein ($P < .005$); baseline DNA damages as measured by tail length (micrometers, $P < .05$), tail DNA (percentage, $P < .001$), and tail moment on DNA ($P < .001$); and the insulin resistance–related variables ($P < .01$ for glucose; $P < .05$ for homeostasis model assessment of insulin resistance) showed significant changes toward clinical improvement at the end of the intervention. However, no significant changes in plasma adiponectin levels were found after 12 weeks. When subjects were divided into 3 groups according to the degree of weight change, significant increases in plasma adiponectin levels were found only in the subjects with the greatest weight change.In conclusion, modest weight loss improved metabolic parameters including blood lipids, insulin resistance, CRP, and DNA damage, but did not increase plasma levels of adiponectin in overweight-obese subjects. However, the increases in plasma adiponectin after 12 weeks were observed in the subjects with a higher degree of weight loss.

Keywords: Weight loss; Obese subjects; Adiponectin; C-reactive protein; DNA damage

1. Introduction

It is well known that modest weight loss through dietary changes and exercise is an effective approach for preventing and managing obesity-associated disorders [1-3]. Several intervention studies have shown that intentional weight loss through dietary change reduces oxidative stress and markers for inflammation such as C-reactive protein (CRP), tumor necrosis factor α, and interleukin 6 (IL-6) in obese subjects [4,5], which results in beneficial effect on the improvement of obesity-related complications.

Adiponectin, one of the most abundant adipose tissue-specific adipokines, is associated with obesity, metabolic syndrome, and coronary artery disease [6,7]. This protein is,

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ironically, reduced in obesity, the existence of insulin resistance, and type 2 diabetes mellitus [8]. Adiponectin has been reported to have antiatherogenic, antidiabetic properties and was proposed as an important mediator of insulin action and glucose metabolism [9]. Furthermore, recent weight loss studies in humans have reported that weight loss achieved by low-energy diets increases plasma adiponectin in both nondiabetic and diabetic patients [10] and this increase may mediate improvement in insulin action and carbohydrate metabolism observed during weight loss [11-13]. There is also evidence that adiponectin administration improved insulin action accompanied by weight loss in mice [14]. However, the weight loss effect on adiponectin concentration is different in terms of the degree of weight change and duration of weight loss intervention.

Given these observations, the present study was designed to determine the effect of weight loss on the plasma adiponectin levels in addition to clinical parameters including baseline DNA damage, CRP, and insulin resistance observed during a 12-week weight loss period in obese subjects.

2. Methods and materials

2.1. Subjects

One hundred eighty-four obese subjects with a body mass index of 23 kg/m^2 or higher based on Asia-Pacific guideline [15] participated in a 12-week weight loss program conducted by the Clinical Nutrition Research Team at Yonsei University. The subjects were all healthy, and those subjects with type 2 diabetes mellitus, thyroid disorders, coronary heart disease, or who were pregnant were excluded. In addition, the subjects who were receiving any medications known to influence the variables studied were excluded. This study was approved by the institutional review board of the Yonsei University, and all subjects gave their written informed consent to participate in this study.

2.2. Protocol for weight loss

The subjects followed a 12-week weight loss program consisting of at least 1255-kJ energy restrictions of their usual intakes. At baseline, usual dietary intakes of subjects were assessed using a semiquantitative food frequency questionnaire and 24-hour recall method. Based on the reported intake of each subject, an individualized and nutritionally balanced diet plan was developed by nutritionists to achieve the goal for a minimum of 3% weight loss. The instructions included changes in food choice, cooking methods, reduction in frequency of snack consumption, exchange of high-energy with low-energy foods, low-fat foods, and limitation of simple sugar consumption. To evaluate and reinforce the subjects' compliance during the intervention period, the nutritionist interviewed them biweekly by telephone interview and the subjects were asked to report the 3-day food records at each visit. Nutrient intakes were determined and calculated as mean values from a 3-day food record using CAN pro (Korean Nutrition Society, Seoul, Korea) based on food composition tables from the National Rural Living Science Institute (7th ed., 2000) in Korea.

2.3. Serum lipid profiles

A blood sample was drawn after a 12-hour fasting period to determine serum lipid profiles (total cholesterol, triacylglycerol [TG], low-density lipoprotein cholesterol [LDL-C], high-density lipoprotein cholesterol) at baseline and at 12 weeks. Serum cholesterol and HDL-C were measured with commercially available kits (Choongwae, Seoul, Korea) by enzymatic methods. Serum TGs were analyzed using a total glycerol test kit (Roche, Basel, Switzerland). Low-density lipoprotein cholesterol was estimated indirectly using the Friedewald formula [16], that is, LDL-C = total cholesterol − [HDL-C + (TG/5)], for subjects with serum TG levels of less than 400 mg/dL and directly measured for subjects with serum TG levels of more than 400 mg/dL. All measurements were made on a Hitachi 747 autoanalyzer (Hitachi Ltd, Tokyo, Japan).

2.4. Serum glucose, insulin, and homeostasis model assessment

Glucose was measured by a glucose oxidase method using a Beckman Glucose Analyzer (Beckman Instruments, Irvine, Calif). Insulin was measured by radioimmunoassay with a commercial kit obtained from Immuno Nucleo Corporation (Stillwater, Minn). Insulin resistance was determined using the homeostasis model assessment in which insulin resistance (HOMA-IR) = fasting serum insulin (pmol/L) × fasting serum glucose (mmol/L)/135.

2.5. Alkaline comet assay for DNA damage

DNA damage was analyzed as described by Green et al [17]. For the comet assay, 120 μL of whole blood was mixed with 900 μL of phosphate-buffered saline and was poured gently over 150 μL of lymphocyte separation solution

Table 1
The baseline characteristics of the subjects

	Total subjects (N = 184)
Male/female	40/144
Age (y)	40.5 ± 1.09
BMI (kg/m^2)	26.9 ± 0.23
Waist (cm)	90.6 ± 0.61
Waist-to-hip ratio	0.89 ± 0.01
Blood pressure	
Systolic blood pressure (mm Hg)	123.5 ± 1.33
Diastolic blood pressure (mm Hg)	76.9 ± 0.84
Tobacco consumption (cigarettes per day)	3.55 ± 0.69
Alcohol intake (g/d)	5.75 ± 1.03
Total energy intakes	10050 ± 102
% Protein	18.6 ± 0.85
% Carbohydrate	58.7 ± 0.99
% Total fat	22.7 ± 0.50

Data are expressed as mean ± SE. BMI indicates body mass index.