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SHORT REPORT

Weight loss improves serum mediators and metabolic syndrome features in android obese subjects

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Summary Serum anti-/pro-inflammatory molecules such as adiponectin, IL-6, IL-10, and TNF-α, and metabolic syndrome (Met Syn) features in 15 android obese (6 Met Syn and 9 non-Met Syn) subjects were assessed during an 8-week weight control program. The results showed that the body mass index, weight, lean body mass, triglyceride, total cholesterol/high density lipoprotein cholesterol ratio, and TNF-α in Met Syn subjects were significantly ($P < 0.05$) improved. This study suggests that weight reduction in android obese subjects may be beneficial in reducing cardiovascular diseases via improving serum IL-6 and TNF-α levels, as well as Met Syn features.

Obesity causes physiological changes in the body, accompanied with Met Syn features, although obesity is caused by multiple factors. Obese subjects deposit excessive adipose cells in the body. Unfortunately, these adipose cells may further secret pro-inflammatory cytokines, such as tumor necrosis factor (TNF)-α and interleukin (IL)-6 [1]. Secreted pro-inflammatory cytokines further induce a macrophage accumulation, resulting in the over-production of TNF-α, IL-6 and monocyte chemo-attractant protein-1 (MCP-1) by the recruited macrophages, causing chronic diseases due to inflammation damage [2]. In addition, obese subjects are generally accompanied with glucose intolerance, suggesting that pro-inflammatory cytokines may cause insulin resistance and cardiovascular diseases (CVD).

Adipose fat in visceral obesity is a stronger risk factor for CVD than subcutaneous fat [3]. It has

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been found that visceral obese subjects, regardless of a normal waist circumference (WC), have a higher carotid artery far-wall intima-media thickness compared with those with increased WC, but less visceral fat, suggesting that a direct estimation for visceral fat may be necessary in assessing atherosclerotic burden in men with type 2 diabetes [4]. In contrast to pro-inflammatory cytokines, adipose tissues also secret a protein hormone, adiponectin, that plays an anti-inflammatory role via inhibiting pro-inflammatory cytokines TNF-α and IL-6, but increasing an anti-inflammatory cytokine IL-10 productions by macrophages *in vitro* [5]. Adiponectin exclusively secreted from adipose tissue into the bloodstream modulates metabolic processes including glucose regulation and fatty acid catabolism [6]. Adiponectin is negatively associated with Met Syn in middle aged and elderly Chinese independently of BMI, physical activity and life habits [7]. Undoubtedly, hypoadiponectinemia is a risk factor for Met Syn [8], and is associated with indices of subclinical atherosclerosis, such as intima media thickness and arterial compliance in obese patients [9]. Therefore, a pernicious balance between pro-inflammatory cytokines and adiponectin secreted by adipose tissues in obese subjects deteriorate metabolic syndrome features.

Android obesity plays an important role in Met Syn. Abdominal fat seems to release some activating factors that modulate metabolism *in vivo*. Android obese subjects have lower serum adiponectin and IL-10 levels compared to normal subjects [10]. Met Syn subjects seem to have lower plasma IL-10 [11], but higher IL-6 levels [12]. However, the real role of IL-6 in Met Syn has not been clarified, yet. This study tried to investigate the relationship among adiponectin, IL-10, TNF-α, IL-6 and Met Syn.

This study analyzed the effects of an 8-week weight control program on serum inflammatory markers and the risk factors for Met Syn in android obese subjects. Based on the WHO criteria (2004) for android obesity in the Asia-Pacific region [13], android obese subjects were recruited for the program at Taichung Veterans General Hospital, Taiwan, ROC. All applicable institutional and governmental regulations concerning the ethical use of human volunteers were approved by the Institutional Review Board (IRB) of Taichung Veterans General Hospital at the 61st committee meeting of the IRB. Fifteen obese subjects including four males and eleven females were qualified for this study. Anthropometric evaluation was measured using traditional method. Biochemical markers for Met Syn from participant subjects were determined using a colorimetric method. Met Syn or non-Met Syn subjects were primarily identified with the updated National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III, 2001) criteria [14], and a slight adoption by Department of Health, Taiwan, ROC. Definition of Met Syn requires the presence of at least three of the following: WC $\geq$90 cm for men, or $\geq$80 cm for women; triglycerides (TC) $\geq$150 mg/dl; high density lipoprotein (HDL) cholesterol <40 mg/dl for men or <50 mg/dl for women; blood pressure $\geq$130/85 mm Hg; fasting glucose $\geq$100 mg/dl [15]. To compare intervention effects of the 8-week weight control program on Met Syn and non-Met Syn subgroups, data collected were also subdivided into two subgroups (Met Syn versus non-Met Syn) for comparison.

The intervention of the 8-week weight control program which combined dietary guidance and aerobic exercise training was implemented. A dietician in the hospital corrected the 3-day diet record and taught the participant subjects to select low-fat, low-sugar, low-salt, high-fiber, and low-calorie diet as usual foods [16]. The subjects were also encouraged to take regular exercise to increase the weight loss. Changes in weight loss, WC, body fat, blood pressure, fasting plasma glucose level, and fasting serum lipid profiles, inflammatory cytokines including IL-6 and TNF-α, as well as anti-inflammatory cytokines including adiponectin and IL-10, were calculated as the difference between the participant subjects' baseline (week 0) and the last week of attendance (week 8). Plasma fasting glucose levels and serum lipids including TG, total cholesterol (TC), and HDL were processed by the Central Laboratory Services at Taichung Veterans General Hospital using enzymatic methods. The body fat was measured using a machine with tetra-polar segmental bioelectrical impedance analysis (SBIA) (Zeus 9.9 Jawon medical Co. Ltd., Seoul, Korea). Baseline data were collected within 1 week prior to the program. Post-program samples were obtained within 1 week after the program finished [15]. Serum adiponectin, TNF-α, IL-6, and IL-10 levels were determined using sandwich ELISA kits, respectively. The adiponectin concentration was assayed using an ELISA kit (Quantikine Human adiponectin, R&D Systems, Inc., Minneapolis, MN, USA). The TNF-α, IL-6, and IL-10 concentrations were assayed using high-sensitivity ELISA kits (Quantikine HS Human TNF-α, IL-6, IL-10, R&D Systems, Inc., Minneapolis, MN, USA). The sensitivity of adiponectin assay was about <15.6 pg/ml. The sensitivity of TNF-α, IL-6, and IL-10 assays was about <0.039 pg/ml, 0.039 pg/ml, and 0.500 pg/ml, respectively.

Values were expressed as means $\pm$ SD. Data were first analyzed using the Kolmogorov—Smirnov test to recognize a normal distribution, followed by the

Table 1 Anthropometric and biochemical characteristics of all obese subjects as well as their grouping including metabolic syndrome (Met Syn) and non-Met Syn obese subjects.

Variable	All subjects ($n = 15$)	Met Syn group ($n = 6$)	Non-Met Syn group ($n = 9$)	P-Value (Met Syn versus non-Met Syn subjects)
Age (years)	41 ± 11	44 ± 10	39 ± 11	0.346
Gender (M/F)	4/11	2/4	2/7	0.645
Waist circumference (cm)	101 ± 7.5	99 ± 7.5	103 ± 7.3	0.154
Weight (kg)	79 ± 13	78 ± 16	80 ± 11	0.480
Body-mass index (kg/m^2)	30.3 ± 3.5	29.3 ± 2.8	30.9 ± 3.9	0.596
Body fat (%)	34.1 ± 4.2	33.1 ± 3.1	34.7 ± 4.8	0.346
Subcutaneous fat (kg)	22.4 ± 4.0	21.3 ± 3.2	23.1 ± 4.5	0.556
Visceral fat (kg)	4.38 ± 1.03	4.15 ± 0.88	4.53 ± 1.14	0.515
Lean mass (kg)	47.6 ± 8.9	47.8 ± 11.8	47.4 ± 7.2	0.768
Fat mass (kg)	26.8 ± 5.0	25.5 ± 4.0	27.6 ± 5.7	0.479
Waist/Hip ratio (WHR)	0.9 ± 0.04	0.91 ± 0.05	0.90 ± 0.03	0.906
Systolic BP (mm Hg)	126 ± 13	133 ± 14	121 ± 10	0.099
Diastolic BP (mm Hg)	79 ± 12	82.2 ± 12.9	76.9 ± 11.5	0.515
Fasting glucose (mg/dl)	97.3 ± 27.2	111 ± 47	87.9 ± 6.1	0.238
HDL cholesterol (mg/dl)	47.1 ± 9.9	43 ± 6	49.8 ± 11.3	0.214
Triglycerides (mg/dl)	162 ± 89	224 ± 87	120 ± 67	0.013*
Adiponectin (μg/ml)	1.26 ± 0.52	1.32 ± 0.5	1.23 ± 0.56	0.814
IL-10 (pg/ml)	1.79 ± 1.66[a]	ND	1.79 ± 1.66	0.000*
TNF-α (pg/ml)	1.27 ± 0.59	1.48 ± 0.56	1.13 ± 0.59	0.346
IL-6 (pg/ml)	2.76 ± 1.63	1.73 ± 0.90	3.33 ± 1.75	0.053

Values are means ± SD. The measurements were, respectively, made at the first day before the weight control program. Statistical significance was calculated by Mann—Whitney U test. ND, not detectable.

[a] The IL-10 values were obtained from 5 subjects in the non-Met Syn group. The minimum detectable dose of human IL-10 ELISA kit used in this study is typically less than 0.5 pg/ml.

* $P < 0.05$.

Pearson correlation for a two-tailed test between two variables. Changes in all subjects between the beginning and post-treatment were analyzed using paired Student's t-test. Intra-changes in Met Syn or non-Met Syn subgroups between the beginning and post-treatment were analyzed using the Wilcoxon Signed-Rank test. Inter-changes between Met Syn and non-Met Syn subgroups through the program were analyzed using the Mann-Whitney U test. Differences between groups were considered statistically significant if $P < 0.05$. Statistical tests were performed using SPSS version 11.5 (SPSS, Inc., Chicago, IL, USA).

Results

To characterize participant subjects of the program, anthropometric and biochemical characteristics of all obese subjects, as well as their subgroups including Met Syn and non-Met Syn subjects were surveyed before the weight control program was administered (Table 1). There were no significant differences in anthropometric and biochemical characteristics, except TG and IL-10, between Met Syn and non-Met Syn subgroups. However, Met Syn-positive subjects had significantly higher TG levels, but slightly lower IL-6 levels in sera than those in non-Met Syn subjects. Particularly, the IL-10 level in Met Syn subjects was too low to be detectable.

To determine the correlation among serum pro-inflammatory, anti-inflammatory mediators and Met Syn features in subjects, the association among serum pro-inflammatory, anti-inflammatory mediators and body compositions of all subjects before the weight control program were analyzed (Table 2). Serum adiponectin concentrations in all subjects showed statistically ($P < 0.05$) negative correlations with BMI, body weight, lean body mass, subcutaneous fat, visceral fat, body fat, and WC. A significantly negative correlation also existed between serum TNF-α and HDL concentration, but a statistically positive correlation existed between serum TNF-α and blood pressure (BP). Serum IL-6 concentration exhibited statistically ($P < 0.05$) positive correlations with the subcutaneous fat, visceral fat, body fat and WC, suggesting that excess levels of IL-6 may result from adipose tissues in android obese subjects. However, there was no significant

Table 2 Correlations among individual inflammatory mediators in sera and body compositions of all subjects before the weight control program.

R	Adiponectin ($n = 15$)	IL-10[a] ($n = 5$)	TNF-α ($n = 15$)	IL-6[b] ($n = 14$)
BMI (kg/m^2)	−0.693*	−0.412	0.341	0.491
Body weight (kg)	−0.672**	0.092	0.463	0.499
Lean mass (kg)	−0.583*	0.700	0.501	0.304
Subcutaneous fat (kg)	−0.547*	−0.652	0.192	0.606*
Visceral fat (kg)	−0.526*	−0.584	0.205	0.658*
Body fat (%)	−0.546*	−0.641	0.196	0.620*
WHR	−0.459	0.369	0.359	0.449
Waist circumference (cm)	−0.514*	−0.423	0.326	0.637*
TG (mg/dl)	0.038	0.372	0.210	−0.329
HDL cholesterol (mg/dl)	0.416	0.105	−0.669**	−0.028
Systolic BP (mm Hg)	0.071	0.819	0.519*	0.232
Fasting glucose (mg/dl)	−0.200	−0.166	0.453	−0.137
Lean/fat mass	−0.115	0.832	0.300	−0.153
Cholesterol (mg/dl)	0.205	−0.472	−0.096	0.293
TC/HDL	−0.183	−0.316	0.388	−0.108
Adiponectin (μg/ml)	1	−0.664	−0.171	−0.354
IL-10 (pg/ml)	−0.182	1	0.273	0.443
TNF-α (pg/ml)	−0.246	0.468	1	−0.111
IL-6 (pg/ml)	−0.362	0.458	−0.111	1

All data are analyzed by Pearson correlation. BMI, body mass index; WHR, waist/hip ratio.
[a] The IL-10 values were obtained from 5 subjects in the non-Met Syn group.
[b] The IL-6 values were obtained from 8 subjects in the non-Met Syn group.
* Means significant at the level of $P < 0.05$.
** Mean significant at the level of $P < 0.01$.

association among Met Syn features and serum IL-10 levels.

The Met Syn features of all subjects, including WC, diastolic BP, and TC were significantly improved ($P < 0.05$) through the 8-week weight control program (Table 3). However, serum anti- or pro-inflammatory mediators, such as adiponectin, IL-10, IL-6 and TNF-α, did not significantly change ($P > 0.05$), although anti-inflammatory adiponectin and IL-10 slightly increased, pro-inflammatory IL-6 and TNF-α slightly decreased after the 8-week weight control program (Table 3). In non-Met Syn subjects, the weight, BMI, lean body mass (Table 4), WC and TC (Table 5) were also significantly improved. The improvement in Met Syn features, such as WC and TC levels in non-Met Syn subjects was better than those in Met Syn subjects (Table 5). Interestingly, serum TNF-α levels in non-Met Syn subjects significantly increased through the 8-week weight control program, while IL-10 also slightly increased (Table 5).

Discussion

We hypothesized that Met Syn responders themselves may hinder the remission of Met Syn. In general, Met Syn may result in an increased risk for developing type 2 diabetes, cardiovascular diseases, psychiatric comorbidity, stress, and a reduced quality of life [17]. The 8-week program indeed achieved some outcomes to both Met Syn and non-Met Syn subjects, although the weight control program was still a short-term program. Unfortunately, serum anti- or pro-inflammatory mediators, such as adiponectin, IL-10, IL-6 and TNF-α, did not markedly change, although anti-inflammatory adiponectin and IL-10 slightly increased and pro-inflammatory IL-6 and TNF-α slightly decreased after the 8-week weight control program (Table 3). We suggest that the weight loss intervention for remission of Met Syn and inflammatory mediators in obese subjects should be extended for at least 15 weeks to avoid significant decrease in lean mass [15], but to increase anti-inflammatory adiponectin and IL-10 and decrease pro-inflammatory IL-6 and TNF-α levels. Nicklas et al. (2005) reported that inflammatory mediators in obese subjects may have a significant decrease by weight loss through behavior modification for 12 months [18]. In addition, a significant weight loss via weight loss intervention is required, too. Previous studies performed in patients after gastric by-pass show a significant decrease in all these parameters after massive

Table 3 Effects of the 8-week weight control program on the body compositions, Met Syn features and inflammatory mediators of obese subjects.

Variable	($n = 15$)			
	Before	After	Change	*P*
Weight (kg)	78.8 ± 12.5	76.6 ± 12.5	−2.19 ± 1.65**	<0.001
BMI (kg/m^2)	30.3 ± 3.5	29.4 ± 3.4	−0.82 ± 0.60**	<0.001
Fat mass (kg)	26.8 ± 5.0	25.7 ± 5.2	−1.02 ± 1.49*	0.019
Body fat (%)	34.1 ± 4.2	33.7 ± 4.2	−0.43 ± 1.27	0.206
WHR	0.90 ± 0.04	0.89 ± 0.05	−0.01 ± 0.02	0.084
Subcutaneous fat (kg)	22.4 ± 4.0	21.6 ± 4.2	−0.82 ± 1.11*	0.013
Visceral fat (kg)	4.38 ± 1.03	4.18 ± 1.09	−0.20 ± 0.40	0.062
Lean mass (kg)	47.6 ± 8.9	46.5 ± 8.5	−1.07 ± 0.84**	0.001
Lean/fat mass	1.81 ± 0.37	1.85 ± 0.39	+0.04 ± 0.11	0.207
Waist circumference (cm)	101 ± 7.5	97.3 ± 7.4	−4.1 ± 2.5**	0.001
Systolic BP (mm Hg)	126 ± 13	122 ± 16.8	−3.07 ± 14.59	0.429
Diastolic BP (mm Hg)	79 ± 12	74.2 ± 11.2	−4.73 ± 7.65*	0.031
Fasting glucose (mg/dl)	97.3 ± 27.2	94.1 ± 13.8	−3.2 ± 6.6	0.108
Cholesterol (mg/dl)	204 ± 38	189 ± 30	−17.3 ± 22.2*	0.002
HDL cholesterol (mg/dl)	47.1 ± 9.9	45.1 ± 7.9	−1.93 ± 4.01	0.830
Triglycerides (mg/dl)	162 ± 89	156 ± 78	−6.27 ± 36	0.512
TC/HDL	4.49 ± 1.18	4.35 ± 1.15	−0.14 ± 0.38	0.165
Adiponectin (μg/ml) ($n = 15$)	1.26 ± 0.52	1.37 ± 0.71	+0.11 ± 0.44	0.350
IL-10 (pg/ml) ($n = 5$)	1.79 ± 1.66	1.89 ± 1.21	+0.08 ± 2.79	0.276
IL-6 (pg/ml) ($n = 14$)	2.76 ± 1.63	2.65 ± 1.7	−0.10 ± 1.39	0.950
TNF-α (pg/ml) ($n = 15$)	1.27 ± 0.59	1.16 ± 0.64	−0.11 ± 0.67	0.540

Values are means ± SD. Statistical significance was analyzed by paired Student's *t*-test. The measurements were, respectively, made at the first day before and after the weight control program. Minus sign (−) represents a decrease; plus sign (+) represents an increase.

* $P < 0.05$.

** $P < 0.01$.

weight loss, including resistin, adiponectin, ghrelin, leptin, and pro-inflammatory cytokines [19]. The participant subjects through the program only lost about 2 kg (Table 3), resulting in no significant differences in inflammatory cytokines.

It has been found that hypoadiponectinemia is a risk factor for Met Syn [8], and may be an index for subclinical atherosclerosis, such as intima media thickness and arterial compliance in obese patients [9]. In this study, we found that the adiponectin concentrations in all participant subjects demonstrated statistically ($P < 0.05$) negative correlations with BMI, body weight, lean body mass, subcutaneous fat, visceral fat, body fat, and WC (Table 2), suggesting that mild or severe obesity also may cause hypoadiponectinemia. Unfortunately, the short-term weight control program could not significantly change serum adiponectin levels in obese subjects (Table 5).

It is reported that obesity strongly associated with increased circulating TNF-α levels, while weight loss lowers systemic levels [20]. Body weight reduction in obese subjects (BMI = 39.3 ± 2.2 kg/m^2) which resulted in improved insulin sensitivity was also associated with a decrease in TNF-α mRNA expression in fat tissue [21]. TNF-α is also expressed at a higher level in the muscle tissue from insulin resistant and diabetic subjects [22]. Our results also showed that TNF-α levels in Met Syn subjects were significantly ($P < 0.05$) decreased through the weight loss program (Table 5). The results are in accordance with previous studies [20—22], suggesting that weight loss in Met Syn patients may effectively decrease systemic low-level inflammation [20]. Particularly, we first found that serum TNF-α levels in non-Met Syn subjects markedly ($P < 0.05$) increased after the 8-week weight control program (Table 5). Changes in serum TNF-α levels between Met Syn patients and non-Met Syn subjects were quite different. We hypothesized that aerobic exercise in non-Met Syn subjects may produce pro-inflammatory cytokines such as TNF-α or IL-6 at higher levels than those in non-Met Syn subjects, although the values may be considered as minor increases. However, the immunoregulatory mechanism on circulating TNF-α levels in normal or non-Met Syn subjects during weight loss through exercise remains to be further clarified. All serum

Table 4 Effects of the 8-week weight control program on body compositions in Met Syn and non-Met Syn subgroups.

Variable	Met Syn group ($n = 6$)			
	Before	After	Change	*P*
Weight (kg)	77.7 ± 16.1	75.7 ± 16.8	−1.98 ± 1.88*	0.049
BMI (kg/m^2)	29.3 ± 2.8	28.6 ± 3.3	−0.75 ± 0.69*	0.045
Fat mass (kg)	25.5 ± 4.02	24.4 ± 4.9	−1.07 ± 1.68	0.180
Body fat (%)	33.1 ± 3.1	32.5 ± 3.2	−0.62 ± 1.26	0.283
WHR	0.91 ± 0.05	0.90 ± 0.07	−0.02 ± 0.02	0.076
Subcutaneous fat (kg)	21.3 ± 3.2	20.5 ± 3.8	−0.87 ± 1.28	0.158
Visceral fat (kg)	4.15 ± 0.88	3.95 ± 1.14	−0.2 ± 0.4	0.275
Lean mass (kg)	47.8 ± 11.8	46.9 ± 11.5	−0.85 ± 0.42**	0.004
Lean/fat mass	1.87 ± 0.28	1.93 ± 0.27	+0.06 ± 0.12	0.344
Variable	Non-Met Syn group ($n = 9$)			
	Before	After	Change	*P*
Weight (kg)	79.5 ± 10.5	77.2 ± 9.8	−2.32 ± 1.58**	0.002
BMI (kg/m^2)	30.9 ± 3.9	30.0 ± 3.6	−0.87 ± 0.56**	0.002
Fat mass (kg)	27.6 ± 5.7	26.6 ± 5.5	−0.99 ± 1.47	0.078
Body fat (%)	34.7 ± 4.8	34.4 ± 4.8	−0.31 ± 1.34	0.502
WHR	0.90 ± 0.03	0.89 ± 0.03	−0.01 ± 0.02	0.274
Subcutaneous fat (kg)	23.1 ± 4.5	22.3 ± 4.4	−0.78 ± 1.07	0.059
Visceral fat (kg)	4.53 ± 1.14	4.33 ± 1.10	−0.21 ± 0.41	0.167
Lean mass (kg)	47.4 ± 7.2	46.2 ± 6.6	−1.21 ± 1.03**	0.008
Lean/fat mass	1.77 ± 0.44	1.80 ± 0.46	+0.03 ± 0.11	0.440

Values are means ± SD. Statistical significance was assayed by Wilcoxon signed-rank test. The measurements were, respectively, made at the first day before and after the weight control program. Minus sign (−) represents a decrease; plus sign (+) represents an increase.

* $P < 0.05$.

** $P < 0.01$.

cytokine except adiponectin levels are extremely low in normal but not septic situations. Therefore, adiponectin plays an anti-inflammatory role via inhibiting pro-inflammatory cytokines TNF-α and IL-6, but increasing an anti-inflammatory cytokine IL-10 productions. In our previous study, a subgroup analysis showed that obese subjects whose original BMI was <30 kg/m^2 had significantly increased serum adiponectin levels during weight loss process [23]. We assume that adiponectin, but not IL-10, plays a main role in metabolic syndrome.

There are some limitations to this study. First, numbers of participant subjects were limited. Second, the 8-week weight loss program is still a short-term for significant weight reduction. Third, many variables, such as waist, adiponectin, and HDL-cholesterol, are heavily gender-dependent. If possible, they should be analyzed gender-specific. However, this study population is too small to allow this. There is a big difference by gender in the number of patients recruited in this study. In our previous study, we discovered that the change patterns of most selected biomarkers, including adiponectin and TNF-α, in either women or men obese subjects were quite similar during the weight-loss process [23]. Therefore, we pooled all data from women and men subjects to minimize the possible deviation due to the number limit of a total of 4 obese men in this study. Fourth, the subjects might not strictly follow the dietary and exercise program. Therefore, the magnitude of weight loss through the program on serum markers has been discussed in another article [23]. Larger and longer-term studies should be conducted in the future. Nevertheless, we achieved some important results in the present study. Undoubtedly, obesity is the most important risk for the development of diabetes and predisposes individuals to hypertension and dyslipidaemia [24]. This study exhibited that obese subjects through a short-term weight control program may have a great benefit to reduce CVD via improving serum IL-6 and TNF-α levels, as well as Met Syn features. In addition, this study has shown profound significance of metabolic syndrome in Asian people. However, it should be paid careful attention to racial differences when we make an interpretation of the data provided. In general, visceral obesity and/or insulin resistance is generally upstream of metabolic syndrome. However, the impairment of insulin secretion often has

Table 5 Effects of the 8-week weight control program on Met Syn features and inflammatory mediators in Met Syn and non-Met Syn subgroups.

Variable	Met Syn group ($n=6$)			
	Before	After	Change	*P*
Waist circumference (cm)	98.7 ± 7.5	96.1 ± 8.7	−2.58 ± 1.36**	0.006
Systolic BP (mm Hg)	133 ± 14	121 ± 22	−11.5 ± 12.8	0.080
Diastolic BP (mm Hg)	82 ± 13	76.8 ± 14.4	−5.33 ± 7.09	0.125
Fasting glucose (mg/dl)	111 ± 47	103 ± 43	−8.17 ± 5.46	0.139
Cholesterol (mg/dl)	214 ± 53	197 ± 37	−17.3 ± 22.2	0.125
HDL cholesterol (mg/dl)	43.0 ± 5.9	43.3 ± 4.6	+0.33 ± 4.59	0.866
TG (mg/dl)	224 ± 87	188 ± 101	−35.5 ± 24.9*	0.018
TC/HDL	4.99 ± 1.15	4.58 ± 0.98	−0.41 ± 0.35*	0.046
Adiponectin (μg/ml)	1.32 ± 0.49	1.71 ± 0.73	+0.38 ± 0.42	0.075
TNF-α (pg/ml)	1.48 ± 0.56	0.71 ± 0.4	−0.76 ± 0.31*	0.028
IL-10[a] (pg/ml)	ND	ND	0	1.000
IL-6[b] (pg/ml)	1.73 ± 0.9	2.00 ± 0.83	+0.27 ± 1.18	0.463
Variable	Non-Met Syn group ($n=9$)			
	Before	After	Change	*P*
Waist circumference (cm)	103 ± 7.32	98.2 ± 6.75	−5.11 ± 2.64**	<0.001
Systolic BP (mm Hg)	121 ± 10	124 ± 14	+2.56 ± 13.4	0.583
Diastolic BP (mm Hg)	76.9 ± 11.5	72.6 ± 8.9	−4.34 ± 8.4	0.160
Fasting glucose (mg/dl)	87.9 ± 6.1	88 ± 6.32	+0.11 ± 5.11	0.950
Cholesterol (mg/dl)	197 ± 25	185 ± 26	−12.7 ± 7.3**	0.001
HDL cholesterol (mg/dl)	49.8 ± 11.3	46.3 ± 9.7	−3.44 ± 2.92**	0.008
TG (mg/dl)	120 ± 67	134 ± 55	+13.2 ± 28.5	0.202
TC/HDL	4.17 ± 1.13	4.20 ± 1.28	+0.03 ± 0.30	0.859
Adiponectin (μg/ml)	1.23 ± 0.56	1.15 ± 0.65	−0.07 ± 0.368	0.859
TNF-α (pg/ml)	1.13 ± 0.59	1.46 ± 0.64	+0.33 ± 0.41*	0.049
IL-10[a] (pg/ml)	1.79 ± 1.66	1.89 ± 1.21	+0.08 ± 2.79	0.276
IL-6[b] (pg/ml)	3.33 ± 1.75	3.17 ± 1.94	−0.16 ± 1.62	0.515

Values are means ± SD. Statistical significance was assayed by Wilcoxon signed-rank test. The measurements were made at the first day before and after the weight control program. Minus sign (−) represents a decrease; plus sign (+) represents an increase. ND, not detectable.

[a] The IL-10 values were obtained from 5 subjects in the non-Met Syn group.

[b] The IL-6 values were obtained from 8 subjects in the non-Met Syn group.

* $P<0.05$.

** $P<0.01$.

a strong effect on the pathogenesis of metabolic syndrome in Asian people. Because insulin secretion and resistance were not included in metabolic syndrome features [15], we did not measure serum insulin levels in the present study. Metabolic syndrome usually accompanies insulin resistance. However, metabolic syndrome subjects may have the lower insulin secretion rather than the severer insulin resistance. Insulin secretion levels in blood and insulin resistance such as homeostatic model assessment (HOMA) for insulin resistance (IR) should be determined in the future to further clarify the relationship between insulin secretion and resistance in metabolic syndrome subjects. In addition, more data from different race or regions should be accumulated to further unravel racial differences of metabolic syndrome.

Overall, the weight, BMI, and WC in subjects with android obesity were indeed improved as the weight control program finished. The improvement in net changes in TG, TC/HDL, and TNF-α in Met Syn subject was significant. This study suggest that weight loss control may be beneficial to reduce CVD via improving serum IL-6 and TNF-α levels, as well as Met Syn features. The knowledge about serum IL-6 and TNF-α levels in android obese subjects improved by short-term weight loss is still new; the results concerning Met Syn improved by weight loss are a confirmation of previous reports.

Conflicts of interest statement

The authors declare no conflict of interest.

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