

The effects of detraining on blood adipokines and antioxidant enzyme in Korean overweight children

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Abstract The present study examined the change to clarify the effects of detraining on the concentration of lipid profiles, serum adipokines and antioxidant enzyme gene expression in Korean overweight children. The subjects were normal children ($n=19$) and obese children ($n=20$) who were further subdivided into the overweight training (OT) group ($n=10$) and the overweight detraining (OD) group ($n=10$). Maximal oxygen uptake ($VO_2\max$); body composition; lipid profiles (TG, TC); adipokines (adiponectin and leptin); antioxidants (blood and gene expressions SOD and GPX) were measured before, 12 weeks, and 24 weeks after the exercise program. Body mass index (BMI) and %fat were significantly higher in the OD group only. However, waist hip ratio (WHR) and systolic blood pressure (SBP) were significantly decreased in the OT group. TG was significantly decreased in the OT group. There was a significant difference in TG level between the two groups. Besides, adiponectin was significantly increased in both the OT group and the OD group. Furthermore, leptin was significantly decreased in the OT group. There was a significant difference in leptin level

between the two groups. In training groups, the expression of SOD was significantly increased after a 12- and 24-week period ($p<0.05$). However, detraining group was significantly increased after a 12-week only ($p<0.05$). In addition, GPX was significantly increased after a 24-week only in the training group ($p<0.05$). Thus, detraining showed that negative effected on body composition and lipid profiles and maintained of uniform period on adipokines and antioxidant enzyme the protein and expression.

Keywords Overweight children · Adiponectin · Leptin · SOD · GPX

Introduction

Complications due to overweight occur in association with various factors. Particularly because various factors such as smoking, drinking and concurrent diseases may affect the treatment outcomes, it is very hard to clarify the relationship between adipokine, overweight and complications [20]. As compared with adults, children have a relatively smaller level of interference due to smoking, drinking and concurrent diseases. It is therefore easier to clarify the relationship between cytokine and overweight.

Adipose tissue has been considered a key factor that affects the insulin activity and glucose homeostasis in the whole body. This adipose tissue releases adipokine and thereby plays a positive or a negative role in association with insulin sensitivity in the skeletal muscle. Some studies have reported that the oxidative stress dysregulated the secretion of adipokine in the obesity and insulin resistance, according to which the decreased adiponectin levels are closely related to somatic markers associated with oxidative stress such as H_2O_2 [13] and 8-isoprostane- $F2\alpha$ [15].

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Regular exercise deranges not only hormone and metabolic homeostasis but also energy balance, which may affect the leptin level during stabilization. This is because the regular exercise causes an energy depletion, and it is of help for reducing the weight and body fat from a long-term perspective. Pasma et al. [31] reported that the energy expenditure due to endurance training reduces the plasma level of leptin. In children as well as adults, the concentration of serum leptin is decreased following a 12-week combined exercise [8]. Because the leptin response at stabilization following endurance training is decreased or not changed [8], however, further demonstration would be mandatory. Circulating adiponectin is inversely proportional to body mass index (BMI) and body fat. To date, however, no studies have presented consistent results associated with exercise. In healthy adults, due to the exercise, the concentration of serum adiponectin was increased [23], decreased [39] or not changed [16]. In patients with cardiovascular diseases or metabolic ones, however, it has been reported to rise due to aerobic training [17, 33].

Detraining has been defined as the partial or complete loss of training induced adaptation, in the response to an insufficient training stimulus [27]. Aerobic training in recently increased multiple measures of lower limb strength and improved aerobic fitness and body composition. These gains were largely lost in the 4 weeks detraining [22]. In addition, exercise training induced enhancement in myocardial mechanics is lost after 2 weeks of detraining in rats [4].

It is highly probable that children with obesity or overweight are vulnerable to cardiovascular diseases and diabetes mellitus after they become adults. As a matter of fact, in cases in which BMI exceeds 28 kg/m², risks of developing coronary heart disease or heart attack would be 3–4 times higher [7]. To date, however, most of the studies about risks of developing cardiovascular diseases associated with overweight have been conducted in adults. Accordingly, only a negligible extent of studies has been conducted in children. In particular, there are very insufficient studies after detraining about pediatric cases of overweight.

The relationship between adipokines and antioxidant enzyme remains to be elucidated, recent studies have demonstrated that oxidative stress in adipose tissue induces the dysregulated production of adipokines [10] and oxidative stress associated with weight reduction decreases serum adipokines levels [42]. The reason for detraining in obese children is largely unknown relative antioxidant enzymes and adipokines.

Accordingly, the objectives of the current study are to clarify the effects of detraining on the concentration of serum adipokines and antioxidant enzyme gene expression in Korean overweight children.

Methods

Subjects

In the present study, Children and Adolescent Physical Growth Standard which was proposed by The Korean Society of Pediatrics from 1998, 20 overweight children with a BMI >95% value or an obesity index (OI) >120% were assigned to the overweight group (OW) and 20 male children with normal weight were assigned to the normal group. Prior to the current study, all the participants and their caregivers submitted a written informed consent. Following a 12-week training in OW, 20 children of the obesity group were randomly assigned to the overweight training (OT) group ($n=10$) and the overweight detraining (OD) group ($n=10$). Excluding one child with normal weight who discontinued the current study, however, the normal weight group comprised 19 children. All the experimental procedures were approved by the Institutional Review Board (IRB). Physical characteristics are summarized in Table 1.

A 24-week training and a 12-week detraining program

The length of the exercise time was calculated [37] as follows; during a period ranging from weeks 1 to 3, a mean value of 3.5 kcal/kg per individual patients was established as a target heat consumption and then multiplied by their weight. The results were determined to be the total calorie consumption per exercise. Then, this was divided by energy expenditure minute (EEm) corresponding to HRR 45% of the exercise intensity. During a period ranging from weeks 4 to 24, a mean value of 4.38 kcal/kg, corresponding to the amount of exercise which was raised by 20% as compared with that during a period ranging from weeks 1 to 3. Following a 12-week training, 20 children of the obesity group were randomly assigned to the OT group ($n=10$) and the OD group ($n=10$). In the OT group, children were trained with the same amount of exercise as the period ranging from weeks 4 to 12 for another 12 weeks. In the OD group, the additional exercise was prohibited except for the basic daily lives.

Body composition and respiratory test measurements

Body composition test was performed by measuring the height, weight, % body fat and BMI with the use of an impedance analyzer (Venus 5.5, Jawon Medical, Korea). An exercise loading test was performed using a treadmill (Inter track 6025, Taeha, Korea) and a gas analyzer (Quark b₂, Cosmed, Italy) with the use of the grade of the lowest physical activity of modified Balke treadmill protocols in children. As proposed by the ACSM [1], criteria for

Table 1 Descriptive characteristics for the study participants

Items	Normal (n=19)	OW (n=20)	t Value	p Value
Age (years)	11.32±1.06	11.30±1.17	0.044	0.965
Height (cm)	146.47±7.49	149.73±6.17	-1.485	0.146
Weight (kg)	40.79±7.71	58.16±7.45*	-7.151	0.000
BMI (m ² /kg)	18.92±2.69	25.84±1.77*	-9.446	0.000
Age (years)	11.32±1.06	11.30±1.17	0.044	0.965
% body fat	11.87±5.66	25.83±2.28*	-10.001	0.000
WHR	0.81±0.04	0.93±0.04*	-9.175	0.000
SBP (mmHg)	100.95±6.03	114.93±10.30*	-5.200	0.000
DBP (mmHg)	61.11±6.10	70.68±6.78*	-4.625	0.000
Triglyceride (mg/dl)	76.44±49.37	168.42±44.89*	-6.093	0.000
Total cholesterol (mg/dl)	165.44±30.31	168.10±28.66	-0.282	0.779
Adiponectin (pg/ml)	12,063.47±3,267.74	8,215.10±1,636.11*	4.613	0.000
Leptin (pg/ml)	1,010.29±599.76	2,496.28±1,244.90*	-4.682	0.000
SOD (U/ml)	2.808±0.701	2.735±1.180	0.225	0.823
GPX (mU/ml)	35.784±10.635	50.008±4.606*	-5.207	0.000
O _{2peak} (ml kg ⁻¹ min ⁻¹)	40.301±5.78	34.38±5.09*	3.406	0.002

Means±SD; **p*<0.05, significant difference between Normal and OW

determining the maximal exercise include cases in which the intensity of exercise was increased when the oxygen intake was smaller than 2.0 ml/kg/min, those in which heart rate was not further increased, those in which the Borg rating of perceived exertion (RPE) exceeded 17, those in which the category ratio scale (CR10) exceeded 7, and those in which the respiratory exchange rate was higher than 1.15 [1].

Blood sample test

Total cholesterol (TC) and triglyceride (TG) were measured using an automated analysis method based on the enzymatic colorimetry with the use of Japan Hitachi 747.

All participants arrived at our laboratory at 08:00 AM after a 12-h overnight fast. After a 10-min rest in a comfortable chair, fasting blood was collected in a plain tube for serum separation from the median cubital vein. Each collected blood sample was centrifuged at 3,000×*g* for 10 min at 4°C and stored at -70°C until required for analysis. All serum samples were immediately frozen and submitted for competitive enzyme-linked immunosorbant assay (ELISA) measurement of adiponectin (AdipoGen, Seoul, Korea) using the standard curve method with a dilution series of a provided human adiponectin (*R*²=0.93 for standard-curve linear regression). Inter-assay coefficient of variation (CV, which was measured by a control provided by the manufacturer with each plate) was 2.8% for adiponectin; intra-assay CV (measurement of six identical serum samples on each plate) was 2.9% for adiponectin.

Leptin was measured using a Bio-plex 200 human serum adipokine (panel B) LINCOpex kit by (BioRad,

Hercules, CA, USA) with an accuracy of 93%, inter-assay variation (% CV) of <20%, and intra-assay variation (%CV) of 1.4–7.9%.

A SOD based assay kit (Cayman Chemicals, Ann Arbor, MI, USA) utilizing a tetrazolium salt was used to detect superoxide radicals generated by xanthine oxidase and hypoxanthine. The standard curve and samples were read at 450 nm. Standard measurements were performed on the same day. The intra-assay and inter-assay coefficient of variation was 3.2% and 3.7%, respectively. Plasma GPX activity was evaluated indirectly by a coupled reaction with glutathione reductase according to a previously described method with a commercially available kit (Cayman Chemicals). GPX measurements were performed on the same day, and the intra-assay and inter-assay coefficient of variation was 5.7% and 7.2%, respectively.

The isolation of PBMC (peripheral blood mononuclear cells) and RT-PCR

Blood sampling was done in the anterior brachial vein three times: prior to the training, following 12 weeks training and following 24 weeks training. The blood samples were placed in a heparin tube and then centrifuged. Following this, lymphocytes were harvested from a buffy coat. This was followed by the genetic analysis (Bionia, perkimelmb geneamp PCR system 2400). To isolate white blood cells (WBCs) and plasma components from the blood samples, a Ficoll-Paque premium was used. Thereafter, with the preservation of blood samples at a temperature of -70°C, TRIzol was used to isolate total RNA from WBCs. Following the dissolution of TRIzol 1 ml, with the addition

of 500 μ l chloroform, the reaction was performed at 4°C for 5 min. Thereafter, the centrifugation was done at 4°C and 13,000 rev/min for 20 min. Then, the supernatant was transferred to a new tube. With the addition of the same amount of isopropanol, the reaction was performed at room temperature for 10 min. After drying, RNA was dissolved in a distilled water 50 μ l containing DEPC. This was followed by the measurement of optical density using a spectrophotometer. Following this, the RT-PCR was performed. Thereafter, with the use of the following primers, polymerase chain reaction (PCR) was performed. SOD (402 bp; F primer, 5'-AGGGCATCATC AATTTTCGAG-3'; R primer, 5'-TTACAC CACAAGCCAAACGA-3'), GPx (405 bp; F primer, 5'-GCGGGCAAGGTACTACTTA-3'; R primer, 5'-CCCACCAGGAAGTTCTC AAA-3'). As the control group for the PCR, 36B4 (228 bp; F primer, 5'-CATGCTCAACATCTCCCC TTCTCC-3'; R primer, 5'-GGGAAGGTGT AATCCGTCTCCACAG-3') was used.

PCR was performed at 94°C for 1 min, at 60°C for 1 min and at 72°C for 1 min. In this condition, the PCR was performed at a total of 30 cycles. Thereafter, the PCR was further performed at 72°C for 10 min. PCR products were confirmed using a 1% agarose gel electrophoresis.

Statistical analyses

Statistical data was analyzed using SPSS/PC Windows version 14.0 statistical package, for which all measurements were expressed as mean \pm standard deviation (SD). To demonstrate the statistical significance of the difference in measurements between children with normal weight and those with overweight, an independent t-test was performed. To make a comparison of the difference depending on the time point, analysis of variance (ANOVA) based on a repeated measurement was performed. In cases in which there was a statistical significance, a contrast analysis was

Table 2 Changes in characteristics following training or detraining

Items	Group	Baseline	12 weeks	24 weeks	F value	p Value
Height (cm)	OT (n=10)	149.28 \pm 5.75	151.11 \pm 6.15 [#]	153.39 \pm 6.33 ^{†,‡}	56.357	0.000
	OD (n=10)	150.17 \pm 6.44	152.05 \pm 6.39 [#]	154.07 \pm 6.90 ^{†,‡}	262.287	0.000
	t	-0.565	-0.581	-0.398		
	p	0.574	0.564	0.692		
Weight (kg)	OT (n=10)	57.58 \pm 6.89	58.36 \pm 8.01	60.43 \pm 8.80 [†]	9.865	0.001
	OD (n=10)	58.73 \pm 7.80	59.95 \pm 8.32 [#]	65.07 \pm 8.17 ^{†,‡}	57.969	0.000
	t	-0.605	-0.754	-2.117		
	p	0.547	0.454	0.039		
%fat	OT (n=10)	25.74 \pm 1.68	25.42 \pm 2.08	25.55 \pm 2.19	0.935	0.411
	OD (n=10)	25.94 \pm 1.83	25.82 \pm 1.96	27.30 \pm 1.51 ^{†,‡}	18.054	0.000
	t	-0.434	-0.770	-3.600		
	p	0.666	0.445	0.001		
SBP (mmHg)	OT (n=10)	113.00 \pm 12.45	108.35 \pm 8.38 [#]	103.05 \pm 7.75 ^{†,‡}	7.697	0.004
	OD (n=10)	116.85 \pm 6.79	112.00 \pm 11.38	116.30 \pm 7.18 [*]	1.408	0.270
	t	-1.487	-1.415	-6.870		
	p	0.142	0.162	0.000		
DBP (mmHg)	OT (n=10)	69.35 \pm 8.44	67.00 \pm 6.02	73.45 \pm 9.57	2.997	0.075
	OD (n=10)	72.00 \pm 3.93	69.05 \pm 3.49	72.25 \pm 5.76	2.786	0.088
	t	-1.558	-1.614	0.588		
	p	0.125	0.112	0.559		
O _{2peak} (ml kg ⁻¹ min ⁻¹)	OT (n=10)	34.78 \pm 3.68	35.05 \pm 3.72	32.73 \pm 2.66	2.942	0.078
	OD (n=10)	33.98 \pm 6.08	35.90 \pm 4.19	32.06 \pm 3.18	2.207	0.139
	t	0.611	-0.829	0.885		
	p	0.544	0.410	0.380		

Means \pm SD. * p <0.05, significant difference between OT and OD

[#] p <0.05, significant difference between baseline and 12 weeks

[†] p <0.05, significant difference between baseline and 24 weeks

[‡] p <0.05, significant difference between 12 and 24 weeks

performed. Besides, to make a comparison between the OT group and the OD group, an independent *t*-test was performed. Statistical significance was set at $\alpha=0.05$.

Results

Table 1 was showed that it was descriptive characteristics of subjects for the study participants. The height and weight in Table 2 were significantly increased in both groups following a 24-week as compared with baseline and a 12-week training ($p<0.05$). BMI and %fat were significantly higher in the only OD group ($p<0.05$). There was a significant difference in BMI and %fat level between the two groups ($p<0.05$) In the OT group, however, there was no statistical significance. WHR in OT group was significantly decreased after 24-week training ($p<0.05$). SBP in OT group was significantly decreased ($p<0.05$) but OD group was not changed.

As shown in Table 3, TG was significantly decreased in the OT group ($p<0.05$). There was a significant difference in TG level between the two groups ($p<0.05$). Besides, adiponectin was significantly increased in both the OT group and the OD group ($p<0.05$). Furthermore, leptin was significantly decreased in the OT group ($p<0.05$). There was a significant difference in leptin level between the two groups ($p<0.05$). SOD blood level was significantly increased in the OT group ($p<0.05$). GPX was significantly increased in both the OT group and the OD group ($p<0.05$). In addition, there was a significant difference in SOD and GPX level between the two groups ($p<0.05$).

As shown in Fig. 1, a comparison was made in both the control group and the OW group between baseline and a 12-week training. The expression of SOD and GPX gene was significantly increased in both groups ($p<0.05$). As shown in Fig. 2, children with overweight were classified into two groups: the training group and the detraining group. Then, a comparison was made between pre, a 12-week

Table 3 Changes in blood lipid and adipokines following training or detraining

Items	Group	Pre	12 weeks	24 weeks	<i>F</i> value	<i>p</i> Value
Triglyceride (mg/dl)	OT (<i>n</i> =10)	164.79±33.03	148.50±20.72 [#]	132.83±16.04 [†]	5.368	0.015
	OD (<i>n</i> =10)	172.05±53.31	154.45±38.50	163.52±23.61 [*]	0.860	0.440
	<i>t</i>	-0.634	-0.745	-5.890		
	<i>p</i>	0.529	0.459	0.000		
TC (mg/dl)	OT (<i>n</i> =10)	167.77±31.61	163.21±43.72	157.23±32.18	0.424	0.661
	OD (<i>n</i> =10)	168.44±24.78	165.75±34.63	183.88±23.15 [*]	2.563	0.105
	<i>t</i>	-0.091	-0.250	-3.682		
	<i>p</i>	0.928	0.804	0.001		
Adiponectin (pg/ml)	OT (<i>n</i> =10)	8,188.40±2,096.22	8,673.40±1,639.80 [#]	9,240.80±1,830.00 ^{†,‡}	5.421	0.014
	OD (<i>n</i> =10)	8,241.80±930.47	8,702.10±953.70 [#]	9,231.00±1,294.00 [†]	14.554	0.000
	<i>t</i>	-0.128	-0.083	0.024		
	<i>p</i>	0.899	0.934	0.981		
Leptin (pg/ml)	OT (<i>n</i> =10)	2,652.31±1,267.42	2,459.15±1,273.02 [#]	1,573.96±532.47 ^{†,‡}	6.041	0.010
	OD (<i>n</i> =10)	2,340.24±1,178.72	2,148.07±795.31	2,150.44±1,040.66 [*]	0.561	0.580
	<i>t</i>	0.988	1.135	-2.701		
	<i>p</i>	0.327	0.261	0.009		
SOD (U/ml)	OT (<i>n</i> =10)	3.120±0.81	3.230±1.23	4.668±0.63 ^{†,‡}	7.834	0.002
	OD (<i>n</i> =10)	2.497±0.41	3.117±0.51	3.042±0.35 [*]	5.659	0.010
	<i>t</i>	-2.056	-0.255	-6.761		
	<i>p</i>	0.063	0.802	0.000		
GPX (mU/ml)	OT (<i>n</i> =10)	34.742±8.29	54.199±9.47 [#]	59.929±10.72 [†]	17.216	0.000
	OD (<i>n</i> =10)	36.827±13.01	59.956±11.05 [#]	40.27±9.31 ^{*,†,‡}	11.122	0.000
	<i>t</i>	0.405	1.187	-4.153		
	<i>p</i>	0.691	0.253	0.001		

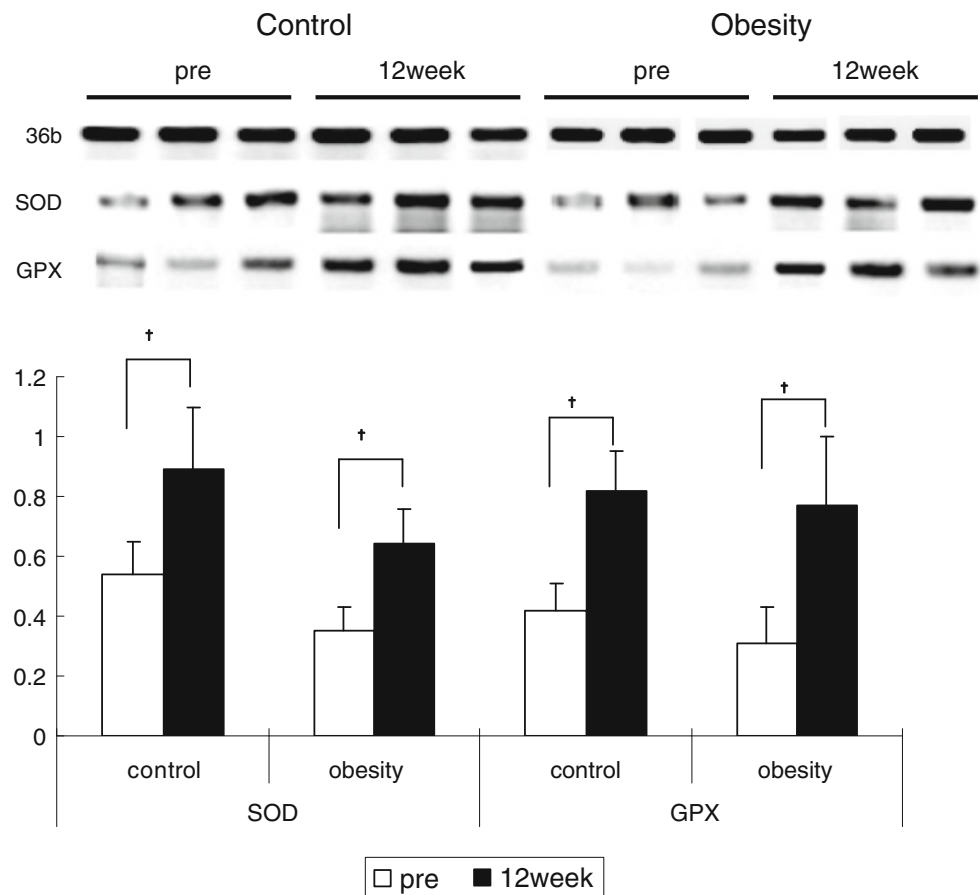
Means±SD; * $p<0.05$, significant difference between OT and OD

[#] $p<0.05$, significant difference between baseline and 12 weeks

[†] $p<0.05$, significant difference between baseline and 24 weeks

[‡] $p<0.05$, significant difference between 12 and 24 weeks

Fig. 1 Effect of training after 12 weeks in the SOD and GPX expression. Means±SD. † $p<0.05$, significant difference between pre- and 12 weeks



training and a 24-week training. In training groups, the expression of SOD was significantly increased after a 12-week and 24-week period ($p<0.05$). However, detraining group was significantly increased after a 12-week period only ($p<0.05$). In addition, GPX was significantly increased after 24 weeks only in the training group ($p<0.05$).

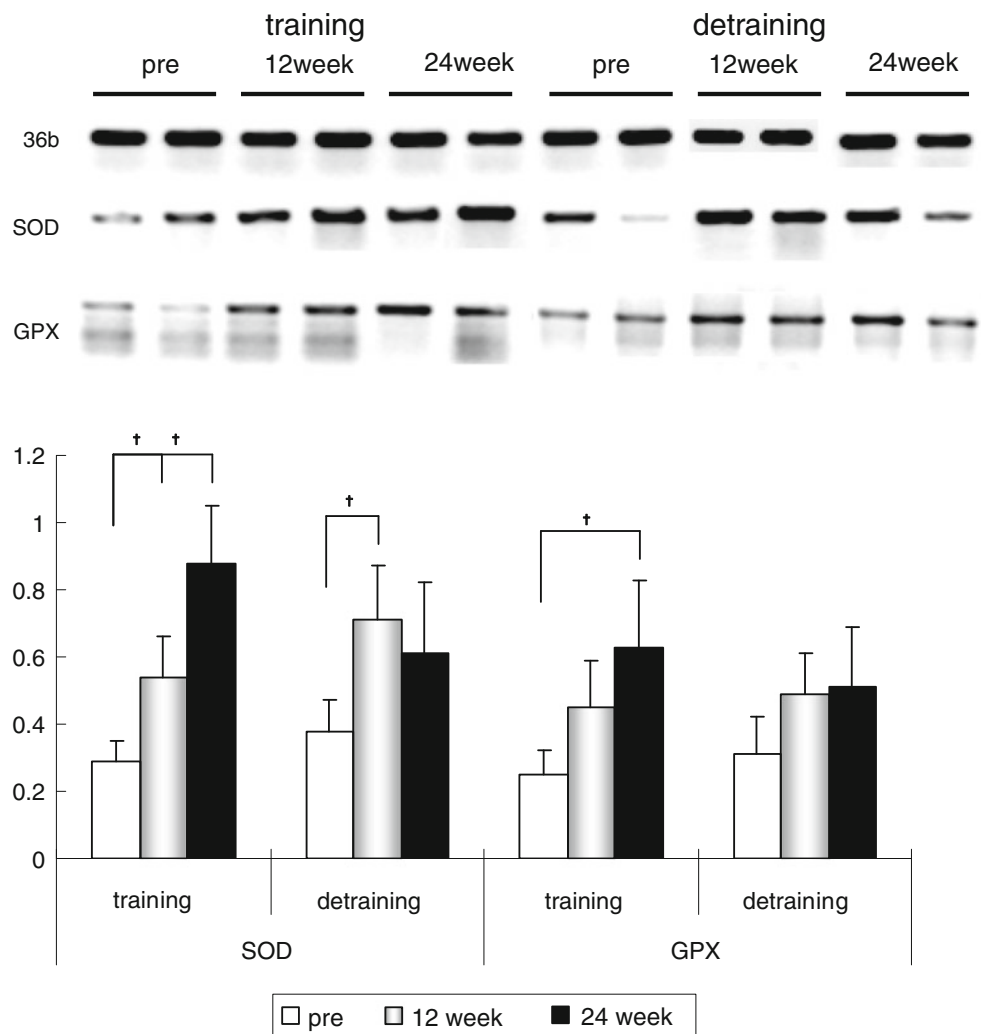
Discussion

In this study, our results showed the positive effects of 12 weeks of training on body composition, serum lipid profiles, adipokines, and antioxidant enzyme gene expressions in Korean overweight children. However, some of the results showed the adverse effects after 12 weeks of detraining in children. These results suggest that regular exercise without detraining plays a role inducing decreased oxidative stress and cardiovascular disease risk factors in overweight children.

According to a long-term study conducted by Lawlor and Leon [25], the increased BMI during childhood raised risks of developing cardiovascular diseases and stroke in adults. However, regular exercise has a positive influence in prevention and treatment of obesity such as decreasing

body weight and insulin resistance which are the causes of metabolic syndrome [3]. According to previous studies about the body composition and exercise in obese children, following a 6-week obesity management program in 16 obese children, the body weight was decreased [28]. In adolescent cases of obesity, following a 12-week gait movement, the weight and body fat ratio were significantly decreased [21]. According to Gutin and Owens [12], following a 16-week training and a 16-week detraining in obese children aged between 7 and 11 years, the body fat ratio was decreased but it was returned to the level prior to the training after the training was discontinued. These reports indicate that the detraining causes negative results that the decreased body fat ratio was increased again. According to the present study, in both the OT group and the OD group, the weight, body fat and BMI were increased during a 24-week period. But the incremental rate seen during a period ranging from weeks 12 to 24 was shown to be 3.5%, 3.3% and 0.5% in the OT group and 7.1%, 10.6% and 4.2% in the OD group in the corresponding order. These results suggest that the incremental rate of body weight and body fat was found to be relatively higher in the OD group, which was partly in agreement the reports made by Gutin and Owens [12].

Fig. 2 Effect of training and detraining in the SOD and GPX expression. Means±SD. †*p*<0.05, significant difference between baseline and 12 weeks. ‡*p*<0.05, significant difference between baseline and 24 weeks



Adiponectin is an anti-inflammatory marker and it is determined to affect the anti-atherogenic due to oxidative stress [24, 40, 41]. Moreover, a lower level of adiponectin has been reported to have a higher level of oxidative stress [14]. By contrast, a higher level of adiponectin has been reported to reduce the oxidative stress [26, 34]. According to the present study, both the OT group and the OD group featured an increased level of adiponectin and the expression of SOD and GPX was also increased. These results were in agreement with previous reports. Besides, our results also showed that the concentration of serum adiponectin was increased despite the increased body weight. But this was not in agreement with previous reports made by Kelly et al. [19]. Furthermore, following the detraining, the concentration of serum adiponectin might be maintained during a certain length of time.

Leptin, a well-known adipokine produced mainly by white adipose tissue, correlates with fat mass and plays a pivotal role in regulating energy homeostasis and metabolism [9, 38]. In addition leptin is mediator of the

inflammatory response that is linked to oxidative stress. Exercise and training is one of negative energy balance factors as well as oxidative stressor. Also, leptin has been demonstrated to be a cause of platelet aggregation and thrombosis. As a pro-inflammatory marker which is contradictory to adiponectin, that is contribute to the early stages of atherosclerosis by promoting endothelial dysfunction [29, 30]. Plasma leptin levels and the free leptin index were increased in both obesity and Type 2 diabetes mellitus [33]. In addition, a few studies reported both higher leptin and lower soluble leptin receptor concentrations in obese kids compared with lean children [5, 32]; however, these studies did not include participants with T2DM. Also, leptin levels in the study of Codoñer-Franch et al. [6] were increased in obese children compared with non-obese children. In the present study, the concentration of serum leptin showed no changes until 12 weeks following exercise. In cases in which the exercise was performed continuously for 24 weeks, the concentration of serum leptin was significantly decreased. In addition, there was a

significant difference following a 24-week period between the two groups. These results indicate that a continuous, regular exercise could improve the concentration of serum leptin.

As the primary mechanisms of antioxidant enzymes, by which oxygen radicals are neutralized, SOD removes the peroxide. As the secondary mechanism, GPX neutralizes H_2O_2 [2]. In the present study, the degree of the protein and expression of GPX was lower prior to training in the overweight group as compared with the normal group. In the overweight group, however, it was increased following a 12-week as compared with prior to training. Following a comparison between the OT group and the OD group, the protein and expression of GPX was increased as time elapsed. On the other hand, the protein and expression of SOD showed no significant changes between baseline and a 12-week training in both groups (the overweight group and the normal group). Besides, following a comparison between the OT group and the OD group, there was no significant difference depending on the period. But both groups had an increasing tendency. Garcia-Lopez et al. [11] reported that a 21-week aerobic exercise affected the activity of anti-oxidative enzymes and it also increased the expression of anti-oxidant enzyme gene. In addition, Thompson et al. [35] also reported that the level of mRNA encoding antioxidant enzyme following exercise. The expression of GPX mRNA was shown to be consistent with the results of the present study. In the present study, however, the protein and expression of SOD was not increased following a 12-week training in the overweight group. The protein and expression of anti-oxidation enzyme is subject to various stress-related factors. In association with this, Wilson and Johnson [36] maintained that the transcription of mRNA was the most common regulator for the expression of antioxidant enzymes. It has also been reported that there are specific proteins to DNA sequences encoding regulatory materials known as AREs (antioxidant response elements) in the prokaryotes. Based on the reports that these prokaryotes regulate the oxidation–reduction transcription factors which are involved in the regulation of the expression of antioxidant enzymes [43]. Based on these reports, because the transcription factors were variably used according to whether the related genes are GPX or SOD, the level of expression would vary. In other words, the expression of GPX is up-regulated by oxidation–reduction sensibility protein (oxy R) to which AREs bound. Because the expression of SOD is regulated by other types of proteins (sox R), however, this might have produced the different results depending on the types of antioxidant enzymes [18]. We found only one study in which strength or endurance training increases mRNA levels of antioxidant enzymes and the protein content of CuZnSOD in untrained men [8]. There was changed significant difference of the

protein level of antioxidant enzymes between the training group and detraining groups because of increased gene expression of antioxidant in the training group. Based on these findings, it can therefore be inferred that it is not abruptly decreased but increased during a certain length of time. It is important to emphasize, thus, regular exercise-induced in the children was protected from oxidative stress factors.

Conclusion

This study showed that regular aerobic exercise in obese children improved body composition, adipokines, lipid profiles, and antioxidant enzyme protein and expression. However, detraining showed that negative effected on body composition and lipid profiles and maintained of uniform period on adipokines and antioxidant enzyme protein and expression. In conclusion, these data suggested that regulatory exercise training over 12 weeks enhanced body composition, lipid profiles, adipokines and antioxidant enzyme in obese children. Thus, constant exercise suggested that improved lipid metabolism and protected oxidative stress, and cardiovascular disease in obese children.

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Conflict of interest The authors declare that they have no conflict of interest. This study was not sponsored by any external organization.

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