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Original Article

The combined effects of physical exercise training and detraining on adiponectin in overweight and obese children

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ABSTRACT

Background: The purpose of this study is to assess the combined exercise programs (12 weeks' physical exercise training, resistance and aerobic) and 6 weeks' detraining on the correlation of metabolic syndrome (MS) markers and plasma adiponectin level in two groups.
Methods: Participants were divided into two groups [physical exercise training group (EG, $n = 8$) and control group (CG, $n = 7$)]. The EG performed a 12-week training program (aerobic and resistance training twice/wk, more than 40 min/d). After 12 weeks' exercise training and 6 weeks' detraining, we also evaluated MS markers and plasma adiponectin at three time periods (baseline, EBP; 12 weeks' exercise program, 12 EP; 12 weeks' and 6 weeks' detraining, 12 + 6 EDP) in overweight and obese children.
Results: Compared with the CG, After the 12 weeks' exercise treatment, weight, body mass index (BMI), waist girth, percent body fat, lean body mass (LBM), percentage lean body, systolic blood pressure, and insulin and homeostatic model assessment (HOMA) indices were lowered in the EG, and plasma adiponectin levels were not altered in the EG. After 6 weeks' detraining, insulin, insulin resistance, and plasma adiponectin levels were significantly increased in the EG. In the adiponectin level, there were positive correlations with LBM and percent lean body and negative correlations with percent body fat, insulin, and insulin resistance after 12 weeks' physical exercise intervention and 6 weeks' detraining.
Conclusion: These findings suggest that combined physical training is a useful tool in the management of MS markers in the training periods. Moreover, there was an additive effect even after the 6-weeks detraining period.

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1. Introduction

Metabolic syndrome (MS) is a combined medical disorder that affects the risk of developing type 2 diabetes mellitus (T2DM) and cardiovascular diseases. It has been revealed that the estimated prevalence is up to 25% of the US population.[1]

It is well known that MS is the clustering of dyslipidemia, impaired insulin sensitivity, hypertension, obesity, T2DM, and cardiovascular disease.[2,3]

It is well reported that regular physical exercise can enhance MS markers and decrease cardiovascular diseases and premature mortality.[4] Also, various kinds of physical activity programs based either on aerobic exercise, resistance training, or a combined type of physical activity may promote insulin sensitivity and weaken or suppress MS in children.

Several sets of criteria are in use for MS diagnosis: atherogenic dyslipidemia, hypertension, elevated blood glucose, obesity, insulin sensitivity, and T2DM.[5] Adiponectin level is also an important factor in estimating MS.

Adiponectin is known as an endogeneous insulin sensitizer, which plays an important role as a modulator of peroxisome proliferator activated receptor gamma action. It has a reverse correlation with obesity and serum levels.[6]

It has been found that plasma adiponectin plays a key role in regulating obesity, carbohydrate metabolism, and insulin resistance,[7,8] and it has been revealed that it decreases in obese individuals and T2DM patients.[9–11]

Plasma adiponectin also increases insulin sensitivity through enhancing fatty acid oxidation in skeletal muscle, liver, and blood vessels.[12,13] It has also been reported that exercise training improves insulin sensitivity activating 5′ adenosine monophosphate-activated protein kinase (AMPK) in skeletal muscle.[14] Most of the previous researches have revealed the negative relationship between insulin level and plasma adiponectin level.[11,15] Several papers revealed that the adiponectin level increased meaningfully with the enhancement of insulin resistance.[15–21] However, some research papers report that there is no connection between insulin sensitivity and adiponectin level. Other studies reported no change in plasma adiponectin level or even decreased rather than increased levels after exercise intervention, despite the enhanced level of insulin.[18,19,22–24]

Therefore, we can conclude that regular exercise training enhances MS with insulin resistance. Generally, there are two possible mechanisms: the first is adiponectin dependent, and the second is adiponectin independent. Physical exercise increases insulin sensitivity through AMP kinase pathway activation. It has recently been revealed that adiponectin also improves muscular insulin sensitivity through the same process. However, it is generally understood that several weeks of exercise training increases adiponectin levels with insulin sensitivity.

Further research is needed on the correlation between plasma exercise training and adiponectin level. However, little research has been carried out on the mechanisms and correlation of exercise and adiponectin responses. Therefore, in our study, we analyzed the relationships between plasma adiponectin level and insulin sensitivity following 12 weeks' exercise training and 6 weeks' detraining.

2. Methods

2.1. *Participants*

Fifteen young overweight [known body mass index (BMI) percentile 85–94] and obese children (known BMI percentile > 95) participated in the present study.[25] The criteria for participating in the current research included no experience of cardiovascular disease,[1,2] no use of medical substances,[3] no experiences of regular exercise training, and no smoking.

The 15 participants were unintentionally divided into two groups [exercise training group (EG, $n = 8$) and control group (CG, $n = 7$)] and three periods (baseline, EBP; 12 weeks' exercise program, 12 EP; 12 weeks' and 6 weeks' detraining, 12 + 6 EDP).

Informed consent was taken from the participants and their parents after they were given a detailed explanation about the study's purpose and methods. This experiment was approved by the Dong-Eui University and Pusan National University Ethics Committee.

2.2. *Exercise protocol*

The exercise program was chosen from a randomized and controlled clinical trial designed to study the combined effects of physical exercise programs. The combined exercise training program was composed of walking exercises and band exercises, which were supervised by many exercise specialists.

Walking exercise was practiced twice a week at 55–64% heart rate max (HRmax) and 65–75% HRmax, based on the Karvonen formula, for Weeks 1–6 and Weeks 7–12, respectively, as an aerobic exercise (30 min for 1–6 wk and 35 min for 7–12 wk).

Rubber band exercises were practiced twice a week as resistance training (50 min/d). Rubber band exercises were composed of nine exercises: squat, sit-up, seated row, knee extension, knee curl, seated leg press, overhead press, elbow curl, and bench press.

Each movement was performed at 70% of the maximal single repetition. Resistance exercise was increased slowly when the individuals could perform 20 repetitions without recess.

The participants increased the thickness of the rubber to increase the exercise intensity. This was accomplished by utilizing the different thicknesses of the band products.

All physiological variables were assessed at three periods (EBP, 12 EP, and 12 + 6 EDP).

2.3. *Baseline, 12 weeks' exercise training, and 6 weeks' detraining measurements*

Body weight and height were gauged using a digital scale while wearing pants but no shoes. Waist girth was also gauged using a tapeline between the iliac crest at the bottom of the rip.

Blood pressure was measured using an automatic device (DINMAP; Critikon, Inc., Lockbourne, OH, USA) and measured twice on arms after a 10-minute rest on the bench with an appropriate-sized cuff, and the mean value was used in the result.

Body composition was determined using dual-energy X-ray absorptiometry (DEXA, Lunar Prodigy; GE Medical Systems, Waukesha, WI, USA), a sensitive test for quantifying