Ilam University of Medical Sciences

Journal of Basic Research in Medical Sciences

Online ISSN: 2383-0972
Print ISSN: 2383-0506

Homepage: https://jbrms.medilam.ac.ir

The Effect of Eight Weeks Functional Exercise Training and Low Carbohydrate Diet on The Levels of Adiponectin, CRP and Lipid Profile in Overweight Women

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Article Info

Article type:
Research article

Article History:
Received: Jul. 14, 2023
Revised: Aug. 10, 2023
Accepted: Sep. 09, 2023
Published Online: Jan. 14, 2024

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A B S T R A C T

Introduction: Exercise training and a low-carbohydrate diet have shown favorable effects on inflammatory cytokines. This study examines the combined impact of an eight-week functional exercise training program and a low-carbohydrate (LC) diet on adiponectin, CRP, and lipid profiles in overweight women.

Material & Methods: Forty-eight overweight women, aged 25-35, were randomly selected for the intervention and allocated into control (n=12), LC diet (n=12), training (n=12), and training+ LC diet (n=12) groups. Functional training, conducted three times weekly for eight weeks at 50-80 percent of maximum heart rate, was paired with an LC diet restricting carbohydrate intake to 50g/d. Blood samples collected before and 48 hours after the final session were analyzed for adiponectin and CRP levels using the ELISA method. Data were analyzed using SPSS software with analysis of covariance and Bonferroni post hoc tests.

Results: Adiponectin levels did not significantly change following the eight-week interventions (p=0.135). However, CRP levels significantly decreased in the LC diet group compared to the control group (p=0.003). Cholesterol, LDL, and triglyceride levels significantly decreased in all experimental groups compared to the control group (p<0.05), while HDL levels significantly increased in the LC diet (p=0.009) and training+ LC diet (p=0.001) groups compared to the control group.

Conclusion: The study underscores the positive impact of LC diet and functional training, either alone or in combination, on improving lipid profiles. However, there seems to be no synergistic effect of LC diet with functional training on changing CRP and adiponectin levels.

Keywords: Exercise Therapy, Adiponectin, C Reactive Protein, Overweight

➢ **How to cite this paper**
Shapouri M, Gholami M, Ghazalian F. The Effect of Eight Weeks Functional Exercise Training and Low Carbohydrate Diet on The Levels of Adiponectin, CRP and Lipid Profile in Overweight Women. J Bas Res Med Sci. 2024; 11(1):9-21.

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Introduction

Low-grade inflammation is a well-recognized characteristic of the obese state, with adipose tissue releasing various inflammatory mediators into circulation (1). This chronic inflammation is a key player in the pathogenesis of several chronic diseases associated with obesity, such as metabolic syndrome, cardiovascular disease, non-alcoholic fatty liver disease (NAFLD), diabetes, hypertension, and certain cancers (2). During adipose tissue expansion and obesity, there is an alteration in the secretion profile of adipokines. This involves the upregulation of inflammatory adipokines, including interleukin 6 (IL-6), monocyte chemoattractant protein-1 (MCP-1), tumor necrosis factor-alpha (TNF-α), alongside a decrease in the levels of anti-inflammatory adipokines such as adiponectin and IL-10 (3). Human adiponectin, a 30-kDa protein abundantly expressed in adipose tissue (4), exerts local actions on various body tissues, leading to the suppression of hepatic gluconeogenesis and improved insulin sensitivity, thus enhancing whole-body energy homeostasis (5). Adiponectin is recognized as a crucial anti-inflammatory adipokine, and a negative correlation between proinflammatory factors and adiponectin has been established (6). Nevertheless, plasma adiponectin levels decrease in obese individuals, showing a negative correlation with plasma insulin and a positive correlation with triglycerides, highlighting its importance in the link between insulin resistance and obesity (7). Due to its protective effects, some researchers propose adiponectin as a potential therapeutic target for obesity, diabetes, and endothelial dysfunction (8).

In contrast to adiponectin, C-reactive protein (CRP) serves as a significant inflammatory marker, with elevated levels observed in overweight and obese individuals. Higher body mass index (BMI) is associated with increased CRP concentrations (9). CRP appears to play a crucial role in various pathological conditions, being significantly associated with elevated insulin levels and insulin resistance. Consequently, CRP is considered a major risk factor for insulin resistance and diabetes (10). Despite the pathological effects of inflammation, exercise training has been shown to attenuate chronic inflammation (11). The positive effects of exercise training on cardiometabolic diseases, such as diabetes and cardiovascular diseases, are partly attributed to its anti-inflammatory effects (12). Additionally, low-carbohydrate (LC) diets have proven to be effective interventions for treating obesity and type 2 diabetes (13). However, conflicting findings regarding the effects of LC diets on the levels of inflammatory mediators have been reported, with some studies showing no changes (14) and others indicating a decrease (15) in inflammation following LC diets. Furthermore, comparing the effects of LC diets with or without exercise training has demonstrated a greater reduction in percent body fat, triglycerides, glucose, and inflammation, as well as a more significant increase in cardiorespiratory fitness in the LC diet combined with exercise training compared to exercise training alone (16). This emphasizes the synergistic effect of combining an LC diet with exercise training. Nevertheless,

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limited information exists regarding the combined effects of LC diets and exercise training, especially on inflammatory and anti-inflammatory mediators. Consequently, our study aims to investigate the changes in CRP and adiponectin levels following an eight-week LC diet and functional training intervention in overweight women.

Materials and methods

Study Subjects

In this semi-experimental research, the Iranian (Tehran) overweight women age ranging 25-35 years old recruited, and 48 women randomly selected among eligible women for conducting present interventions.

Study Design

Following the selection of study participants, they were randomly allocated into four equal groups, each consisting of 12 subjects: control (n=12), LC diet (n=12), training (n=12), and training+LC diet (n=12) groups. All subjects voluntarily participated in this study, and various intervention stages were conducted in accordance with the principles of the Declaration of Helsinki. Participants had a sedentary lifestyle in the preceding years, and all were overweight (BMI: 25-29.9 kg/m²). They were in good health and had no cardiometabolic disorders (including type 2 diabetes, hypertension), with no physical or medical limitations preventing the completion of exercise sessions. Overweight women provided informed consent by signing the consent form and willingly accepted all research conditions, including potential benefits and side effects. Subsequently, baseline testing (including blood sampling, height, and weight measurements) was performed, and after one week, the eight-week interventions (control, training, training+LC diet, LC diet) commenced as agreed upon by the participants.

Functional Training Program

The exercise training conducted in the present study utilized functional circuit training (FCT), which emphasized functional balance and lower-body strength-based exercises. FCT sessions occurred three times a week over a 12-week period. Prior to each training session, participants engaged in a 10-minute warm-up by walking at their usual pace, and the training session concluded with a 5-minute cool-down involving stretching exercises. Throughout the FCT program, participants in the control and LC diet groups maintained their daily routine lifestyle without incorporating regular physical activity. The details of the FCT training program are provided in a previous research publication (17).

Low Carbohydrate Diet

Participants in the low-carbohydrate (LC) diet and training+LC diet groups received three pre-prepared meals and two interim mid-meal snacks daily throughout the eight-week intervention. Neither diet group was provided with a specific calorie or energy goal. The pre-prepared meals for the LC diet group did not exceed a total of 50 g of carbohydrates (CHO) per day. Daily food intake, including all foods and quantities, was meticulously recorded for both groups, and adherence to the diet was documented daily using a 24-hour food log. The specific LC diet employed

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in this study had previously been utilized by Perissiou and colleagues (16).

Biochemical Measurement

Blood sampling was conducted both before and after the completion of the eight-week experiment. A total of 7 ml of blood was drawn from each participant by a laboratory specialist. The collected blood samples were then transferred into a Falcon tube and centrifuged at 3000 rpm for 10 minutes. Subsequently, the serum samples were carefully separated and stored in the freezer for subsequent biochemical measurements.

The levels of components in the lipid profile were determined using a special kit from Pars Azmoon (Iran). Additionally, fasting glucose and C-reactive protein (CRP) levels were measured using Pars Azmoon kits. The serum levels of adiponectin were quantified using an enzyme-linked immunosorbent assay (ELISA) kit from Cusabio (China), with a sensitivity of 1.102 ng/mL and catalog number CSB-E07270h.

Body composition analysis was carried out using the BOCA-X1 analyzer. This comprehensive approach to blood sampling and biochemical analysis, along with body composition assessments, ensured a thorough evaluation of the participants' physiological responses over the course of the eight-week experiment.

Statistical Analyses

The study data were analyzed using SPSS software version 24. Prior to analysis, the normality of data distribution was confirmed through the Shapiro-Wilk test. As the data exhibited a normal distribution, parametric tests were deemed appropriate for the analysis. Between-group differences among the control, LC diet, training, and training+LC diet groups were determined using Analysis of Covariance (ANCOVA) and Bonferroni post-hoc tests. Additionally, intragroup changes, reflecting the difference between baseline and final analysis, were assessed using paired t-tests. A significance level of $p<0.05$ was considered for all statistical analyses, ensuring a robust evaluation of the study's findings.

Results

All 48 participants in the present research successfully completed the prescribed interventions, and no subjects were excluded from the study. All participants were included in the statistical analysis. The pre-test and post-test measurements of participants' body weight, BMI, body fat percentage, cholesterol, triglycerides, LDL, HDL, and fasting blood glucose in the different groups are reported as mean±standard deviation in Table 1. This comprehensive inclusion of data allows for a thorough examination of the effects of the interventions on various physiological parameters across the study groups.

Table 1. The levels of study variables (Mean ± SD).

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Variables	Stage	Control	Training	LC diet	Training+LC Diet	Inter-Group P Value
Glucose (mg/dl)	pre test	87.5±6.54	89.1±10.32	90.9±5.74	91.8±5.87	**0.976**
	post test	88.3±6.27	88.4±4.87	89.6±5.14	89.6±5.34	
Paired t test		0.999	0.709	0.480	0.011	-
Cholesterol (mg/dl)	pre test	198.4±9.65	201.1±9.36	188.1±14.48	192.5±9.67	**<0.001**
	post test	200.1±8.03	178.6±9.45	178.3±12.25	174.5±10.04	
Paired t test		0.316	<0.001	<0.001	<0.001	-
Triglyceride (mg/dl)	pre test	176.6±20.13	179.4±20.61	180.3±10.93	174.9±9.80	**<0.001**
	post test	178.1±16.52	171.4±16.84	171.6±7.78	166.6±8.87	
Paired t test		0.555	0.004	<0.001	<0.001	-
LDL (mg/dl)	pre test	118.2±10.61	121.2±7.61	118.7±7.17	118.9±5.40	**<0.001**
	post test	118.1±9.95	115.1±6.99	112.1±6.50	109.1±3.53	
Paired t test		0.960	<0.001	<0.001	<0.001	-
HDL (mg/dl)	pre test	54.1±8.39	56.7±7.59	57.2±6.66	58.8±2.12	**<0.001**
	post test	54.6±6.81	59.7±5.89	61.5±5.55	63.7±3.10	
Paired t test		0.673	0.078	0.001	<0.001	
Percent Body Fat (%)	pre test	31.7±3.04	33.9±3.69	34.6±4.12	33.5±3.54	**<0.001**
	post test	32.3±3.26	30.4±2.87	29.1±3.25	28.2±2.42	
Paired t test		0.47	<0.001	<0.001	<0.001	
BMI ($kg.m^2$)	pre test	28.9±1.04	29.4±0.69	28.9±1.02	28.7±1.10	**<0.001**
	post test	29.1±1.06	28.6±0.62	27.9±0.96	27.4±0.98	
Paired t test		0.24	0.009	<0.001	<0.001	

The levels of cholesterol, triglyceride, LDL, HDL, percent body fat, and BMI exhibited a significant between-group difference as determined by the ANCOVA test ($p<0.001$). However, there was no significant difference between research groups for glucose levels ($p=0.976$). Bonferroni post-hoc test findings indicated a significant decrease in cholesterol levels in the LC diet, training, and training+LC diet groups compared to the control group ($p<0.001$). Additionally, a significant decrease in cholesterol levels was observed in the

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training group compared to the LC diet group (p=0.036). Furthermore, triglyceride and LDL levels significantly decreased in the LC diet, training, and training+LC diet groups compared to the control group (p<0.001). Notably, there was a significant increase in HDL levels in the LC diet (p=0.009) and training+LC diet (p<0.001) groups compared to the control group. Conversely, a significant decrease in percent body fat and BMI was observed in the LC diet, training, and training+LC diet groups compared to the control group (p<0.001) (Table 1).

The ANCOVA test results revealed that the change in adiponectin levels after the eight-week intervention was not significant between the different groups (p=0.135). Additionally, paired t-tests demonstrated no significant changes in adiponectin levels after the eight-week intervention in the control (p=0.266), LC diet (p=0.358), training (p=1.000), and training+LC diet (p=0.067) groups. The serum levels of adiponectin in the various groups before and after eight weeks are depicted in Figure 1.

Analysis of CRP findings suggested a statistically significant difference between the various groups (p=0.004). The Bonferroni post-hoc test indicated that CRP levels significantly decreased only in the LC diet group compared to the control group (p=0.003). Intragroup analysis revealed a significant decrease in CRP levels only in the LC diet group (p=0.002). However, there was no significant change in CRP levels in the control (p=0.339), training (p=0.551), and training+LC diet groups (p=0.256) (Figure 2).

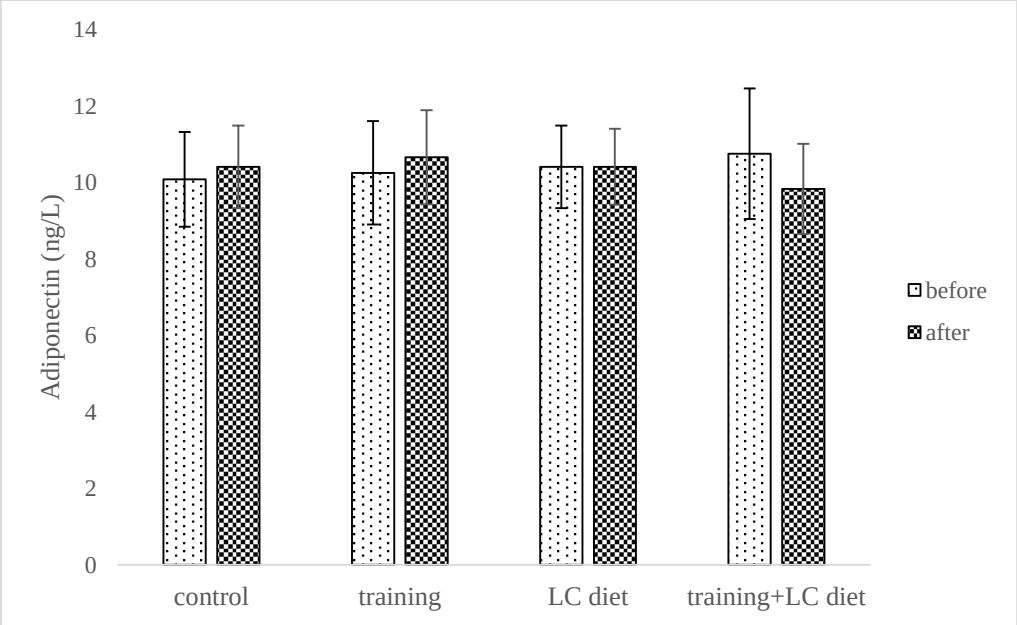


Figure 1. Changes in Serum Adiponectin Levels Before and After the Eight-Week Intervention in Various Groups (Mean±SD).

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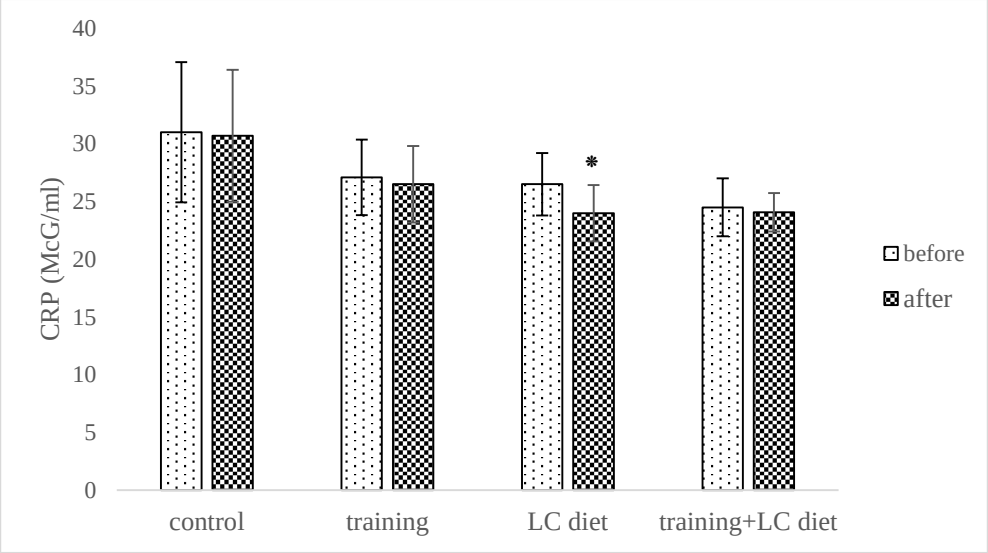


Figure 2. Changes in Serum CRP Levels Before and After the Eight-Week Intervention in Different Groups (Mean±SD). * Indicates a significant decrease compared to the control group.

Discussion

The present study aimed to investigate the effect of eight weeks of functional training and an LC diet on the levels of CRP, adiponectin, and lipid profile in overweight women. The main finding was that functional training alone and with an LC diet had no effect on CRP and adiponectin levels, but the eight-week LC diet led to a significant decrease in CRP levels compared to the control group. Adiponectin, an important adipokine, plays a crucial role in the regulation of glucose and lipid metabolism and insulin sensitivity, with these positive effects attributed to its anti-inflammatory, anti-fibrotic, and antioxidant properties (18). Various molecular mechanisms, such as the upregulation of adenosine monophosphate-activated protein kinase (AMPK) in the liver and skeletal muscles, seem to be involved in the positive effects of adiponectin (19).

Due to the benefits of adiponectin, it is considered a therapeutic target for obesity and its related disorders (20). Exercise training and nutritional diets are effective interventions in increasing adiponectin levels; however, the present findings showed that functional training alone and in combination with an LC diet did not have a significant effect on adiponectin levels. Exercise training has different effects on the levels of adiponectin, with an increase (21), decrease (22), and no change (23) reported after exercise training. These contradictory findings may be caused by various factors such as the type, intensity, and duration of exerted training programs, as well as the physical characteristics, age, and gender of participants. Ando et al. (2009) confirmed our findings and suggested that 12 weeks of combined training (endurance-resistance) in healthy subjects had no effect on adiponectin levels. They concluded that combined training without reducing body weight cannot have a significant effect on adiponectin levels, or its effect is very limited (24).

In another study, researchers observed the positive effects of aerobic training,

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specifically improvement in lipid profile, independent of changes in adiponectin levels (25). Despite adiponectin's recognized role in enhancing insulin sensitivity, some studies suggest that exercise-induced reductions in insulin resistance may occur independently of changes in adiponectin levels. For instance, Ahmadizad et al. (2007) reported a significant decrease in insulin resistance after 12 weeks of aerobic and resistance training in overweight men, but the change in adiponectin levels was not significant. The researchers concluded that the lack of change in adiponectin levels was likely due to the low intensity of the training program (26).

In line with these findings, Fatouros et al. (2005) demonstrated that the increase in adiponectin levels in overweight and obese elderly populations is dependent on the intensity of resistance training. A six-month low-intensity resistance training program (45-50% 1RM) had no effect on adiponectin levels, while moderate (60-65% 1RM) and high-intensity (85-85% 1RM) resistance training was associated with a significant increase in adiponectin levels. Notably, the high-intensity group exhibited a further increase in adiponectin levels. The researchers attributed the upregulation of adiponectin to the downregulation of inflammatory mediators (IL-6 and TNF-α) levels (27), although, unfortunately, these circulating levels were not measured in our study.

Another finding of the present study was that CRP levels in the functional training, LC diet, and functional training+LC diet groups decreased by 2.21%, 9.43%, and 8.55%, respectively. However, the reduction in CRP was statistically significant only in the LC diet group. It is well-documented that CRP, IL-6, and other inflammatory markers increase in obese populations compared to those with normal weight. Conversely, the reduction of visceral adipose tissue has been associated with a decrease in inflammatory marker levels (28). Libardi et al. (2012) supported the present findings and suggested that 16 weeks of aerobic, resistance, and combined training do not affect pro-inflammatory cytokines such as CRP, IL-6, and TNF-α, although improvement in lipid profile was observed in the different exercise training groups (29).

In contrast to the present findings, it has been reported that eight weeks of aerobic training leads to a significant downregulation of CRP, concluding that the reduction in CRP can occur independently of changes (decrease) in body weight (30). The researchers attributed this reduction to the possible mechanisms of reducing adipose tissue, given the role of adipose tissue-secreted cytokines (IL-6 and TNF-α) in CRP secretion. Increasing antioxidant capacity and improving endothelial function are also suggested mechanisms for reducing CRP levels with exercise training (30, 31). In the present study, a decrease in CRP levels with functional training was observed, though not statistically significant. Considering that the anti-inflammatory effects of exercise training depend on the intensity and duration of exercise sessions (32), insufficient intensity and duration in our training program could be a possible reason for the non-significant changes in CRP levels. However, the exact mechanism needs to be determined in future studies.

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The anti-inflammatory effects of exercise training are exerted through various mechanisms. Some studies attribute these effects to the reduction of visceral fat mass, leading to a decrease in adipokine secretion from adipose tissue and the induction of an anti-inflammatory environment (33). Additionally, the release of heat shock proteins, changes in the phenotype of immune cells, and the reduction of tissue hypoxia are other crucial mechanisms contributing to the anti-inflammatory effects of exercise training (34).

Another finding of present study was that eight weeks LC diet alone and combined with functional training did not have a significant effect on adiponectin levels. There is insufficient evidence for positive effects of LC diet on the levels of adiponectin and leptin, and researchers suggested that LC diet does not have a significant effect on adiponectin levels (35). Some researchers indicated that despite LC diet role in upregulation of adiponectin, the observed changes wasn't significant statistically (36), and some researchers showed that six weeks LC, low-fat or hypocaloric diets in overweight subjects don't affect adiponectin levels despite weight loss (37). On the other hand, the present findings showed that LC diet cause to a significant decrease in CRP levels, but LC diet with functional training wasn't associated with significant change in the CRP level. Luscombe et al (2005) confirmed our findings and reported that 12 weeks restricted carbohydrate diet led to a significant (24%) reduction in CRP levels in overweight and obese individuals, and researchers considered weight loss as effective mechanisms for CRP reduction (38). In another study, Sharman et al (2004) confirmed present findings and showed that LC diet in overweight men can exert anti-inflammatory effects and significantly reduce the levels of inflammatory cytokines such as CRP, TNF-α and IL-6, They attributed the anti-inflammatory properties of LC diet to the reduction of adipose tissue as a main source for inflammatory cytokine, as well as decrease in fasting and post-meal triacylglycerol levels, which these changes in turn is associated with reducing the formation of other lipoproteins such as LDL and VLDL (39). The anti-inflammatory effects of ketogenic diet or caloric restriction can be related to the action of β-hydroxybutyrate (βHB). It has been proven that βHB inhibits histone deacetylase enzymes, free fatty acid receptors and NLRP3 inflammasome, which results in inhibiting inflammatory pathways, oxidative stress and preventing the progression of chronic diseases (40). Cipryan et al (2021) indicated that 12 weeks (three to five sessions per week) high intensity interval training (HIIT) combined with LC diet result in significant increase in adiponectin, significant decrease in leptin levels and increase in the adiponectin/leptin ratio. Moreover, they suggested that LC diet with HIIT has a further and synergistic effects on inflammatory markers, and considered LC diet as an effective intervention to prevent chronic diseases (41). The short duration of intervention (eight weeks versus 12 weeks), the different type of exerted exercise training program, and the low volume of training program (low training sessions per week) in the present study compared to the above-mentioned study can be possible reasons for observed

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contradiction findings regarding changes in the adiponectin level. According to the findings of present study, functional training alone and together with LC diet does not have a significant effect on CRP and adiponectin levels. Nevertheless, functional training, LC diet and their combination led to a significant improvement in lipid profile. It seems that functional training and LC diet alone could not provide the enough stimulation for significant changes in the levels of inflammatory (CRP) and anti-inflammatory (adiponectin) adipokines in overweight women, which may be related to insufficient duration or intensity of functional training.

Conclusion

The present findings highlight the positive effects of an LC diet, with or without functional training, in improving lipid profiles. According to the results, it can be inferred that the beneficial outcomes of the interventions applied (LC diet, functional training, or both), including improvements in lipid profiles, body weight loss, and reduced body fat percentage, may occur independently of changes in adiponectin and CRP levels. The exact mechanisms underlying these effects should be investigated in future studies.

Acknowledgements

This article is based on an M.Sc. exercise physiology thesis, and the researchers extend their gratitude to all study participants.

Financial support

No financial support was received for this research.

Conflict of interest

The authors declare no conflict of interest.

Authors' contributions

M SH and M GH designed, executed, and monitored the research protocol and interventions. All authors participated in data collection. F GH conducted the statistical analysis. Additionally, the draft and final manuscript were edited and approved by all authors.

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