Research Article

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The effects of various strength training intensities on blood cardiovascular risk markers in healthy men

Sağlıklı erkeklerde farklı kuvvet egzersizi yoğunluklarının kan kardiyovasküler risk belirteçleri üzerindeki etkileri

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Abstract

Objectives: Regular physical exercise, especially aerobic exercise, is known to have a protective effect on cardiovascular health. The aim of this research is to look at the impact of two separate resistance training programs on blood biomarkers that are associated with the early detection of cardiac risk.

Methods: Forty-five male participants (mean 41 years) were randomly divided into three groups: The low-intensity resistance exercise group (LIEG), the moderate-intensity resistance exercise group (MIEG), and the control group (CG). The programs were implemented three times a week and in two sets. MIEG consisted of 8–10 repeats at 70–80%

density of one repetition maximum load (1RM), while LIEG consisted of 15–17 repeats at 50–60% density of 1RM. CG did not participate in any exercise program. Two-factor mixed-design ANOVA assessed the data.

Results: Before, fourth week, and after the exercise program in repeated measurements, there was a significant decrease in body mass (–1.7%), body mass index (–1.7%), apelin (–44%), and pentraxin 3 (–39%) levels in MIEG ($p < 0.05$). Additionally, our study noted a decrease in pentraxin 3 (–25%, $p < 0.05$) and interleukin 6 (–21%) levels, while there was an increase in creatine kinase (18%), and lactate dehydrogenase (7.4%) levels in LIEG. Strength levels improved significantly in exercise groups.

Conclusions: Eight weeks of moderate-resistance training can potentially reduce the cardiovascular risk in healthy men.

Keywords: apelin; cardiovascular risk; pentraxin 3; regular exercise; strength.

Öz

Amaç: Düzenli fiziksel egzersizin, özellikle aerobik egzersizin, kardiyovasküler sağlık üzerinde koruyucu bir etkisi olduğu bilinmektedir. Bu çalışmada amaç, iki farklı yoğunluktaki direnç egzersiz programının kardiyak riskin erken teşhisinde yer alan kan belirteçleri üzerindeki etkilerini araştırmaktır.

Gereç ve Yöntem: Kırk beş erkek katılımcı (ort. 41 yaş) rastgele üç gruba ayrıldı: Düşük yoğunluklu direnç egzersiz grubu (LIEG), orta yoğunluklu direnç egzersiz grubu (MIEG)

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ve kontrol grubu (CG). Programlar haftada üç kez ve iki set olarak uygulandı. MIEG bir tekrarlı maksimum yükün (1RM) %70–80 yoğunluğunda 8–10 tekrar ile uygulanırken, LIEG 1RM'nin %50–60 yoğunluğunda 15–17 tekrardan oluştu. CG herhangi bir egzersiz programına katılmadı. Veriler, iki faktörlü karma tasarım ANOVA ile değerlendirildi.

Bulgular: Egzersiz programının öncesi, 4. haftası ve sonrasındaki tekrarlı ölçümlerde; MIEG'de vücut kütle indeksi (-1.7%), vücut kütle indeksi (-1.7%), apelin (-44%) ve pentraksin 3 (-39%) düzeylerinde anlamlı azalma vardı ($p < 0.05$). Ayrıca, çalışmamızda LIEG'de pentraksin 3 (-25% , $p < 0.05$) ve interlökin 6 (-21%) düzeylerinde azalma ile kreatin kinaz (18%) ve laktat dehidrojenaz (7.4%) seviyelerinde artış gözlemlendi. Egzersiz gruplarında kuvvet seviyeleri önemli ölçüde arttı.

Sonuç: Sekiz haftalık orta yoğunluklu direnç antrenmanı sağlıklı erkeklerde kardiyovasküler riski potansiyel olarak azaltabilir.

Anahtar Kelimeler: apelin; düzenli egzersiz; kardiyovasküler risk; kuvvet; pentraksin 3.

Introduction

A sedentary lifestyle is one of the main risk factors that cause cardiovascular diseases. Inactivity also results in a 1% decrease in muscle mass every year in people over the age of 40, leading to a reduction in muscle strength and endurance [1]. Resistance exercise positively affects muscle mass and strength, muscular endurance, flexibility, and balance and helps prevent symptoms of many chronic diseases like cardiovascular diseases, type 2 diabetes, and hypertension [2, 3]. Due to these features, resistance exercises are popular among athletes and regular individuals who participate in physical activities for health protection.

Resistance exercise adaptations, both acute and chronic, are dependent on the combination of critical components [4, 5]. Low intensity-high frequency resistance training, for example, focuses on muscle endurance. On the other hand, the main aim of moderate intensity-volume resistance training ($\sim 70\text{--}80\%$ of one-repetition maximum load [1RM]) is to cause hypertrophy, which increases the cross-sectional muscle area. However, long-term adaptations of both training programs result similar to aerobic exercises through increased capillary intensity and oxygen utilization capacity [6].

Resistance training can cause localized muscle tissue injury. Two of the best indirect markers of muscle damage are serum creatine kinase (CK) and lactate dehydrogenase (LDH). Several studies found a moderately positive association between a resistance training session's volume load and serum CK and LDH levels [7].

The various advantages of exercise for cardiometabolic health and weight management are partly regulated by the formation of myokines in contracting muscles, which are released into the bloodstream. Myokines exert endocrine, autocrine, or paracrine effects between the muscle and other organs, including the brain, adipose tissue, bone, liver, gut, pancreas, vascular bed, skin, and muscles. Myokines affect cognition, lipid, and glucose metabolism, white fat browning, bone formation, endothelial cell activity, hypertrophy, skin structure, and tumor development, among other biological functions [8, 9]. Resistance training's anti-inflammatory effects are known to be determined by changes in body composition, muscle mass increase, and fat mass loss. According to the physiological mechanisms that justify the beneficial effects of increased muscle mass on inflammation, resistance training improves energy consumption and insulin sensitivity. Higher muscle mass can create anti-inflammatory myokine [10].

One of the myokines, apelin, is upregulated by insulin, effective in exercise and glucose homeostasis [8, 11]. Apelin preserves its efficacy in the cardiovascular system by lowering blood pressure and providing vasodilation through endothelium-dependent nitric oxide [12]. Apelin levels increased through aerobic training in type 2 diabetes and obese patients [13–15]. However, a one-season follow-up of professional soccer players did not improve circulating apelin levels, and these differences concluded that they were not linked to performance in trained soccer players [8]. The studies' contrasting findings tend to be affected by improvements in insulin sensitivity, lipid and glycemic profiles achieved by exercise programs. The effects of strength exercise on healthier individuals, on the other hand, remain unknown.

Pentraxin 3 (PTX3) belongs to the multimeric protein family, which includes short and long pentraxins. All these proteins play a critical role in innate immunity and are often referred to as acute phase immune proteins. However, their effects on modulation of the cardiovascular system affect various events such as inflammation and angiogenesis [16]. PTX3 levels are low in the blood serum and tissues of healthy individuals. However, PTX3 expression, on the other hand, quickly increases in response to inflammatory stimuli in a variety of diseases, including infectious, autoimmune, and degenerative disorders [11, 17]. Despite the fact that there are only a few findings on the interaction between PTX3 and exercise, current data shows that there is no difference in PTX3 levels after a 30-min submaximal aerobic exercise [18], although plasma PTX3 levels improve relative to the control group [14]. Similarly, PTX3 levels increased in untrained individuals with acute moderate and intense aerobic exercise [19].

Interleukin 6 (IL6) is a proinflammatory cytokine with several functions that mediate immune and inflammatory

responses. Firstly, IL6 is the most important stimulus of acute-phase response reaction to inflammation or tissue injury [17]. Therefore, while IL6 levels in healthy individuals are very low in the homeostatic state, its synthesis increases during inflammation. It is reported that after cycling for 120 min in 55–60% VO_2peak , the exercise-induced IL6 response was higher in the obese population compared to the normal weight population [11]. In contrast, no reduction in IL6 was observed in overweight or non-obese individuals following a 10-week resistance or aerobic exercise regimen [17].

Since cross-sectional trials showed a substantial negative association between aerobic fitness level and chronic systemic inflammation [2, 20], it is recommended that aerobic-based dynamic exercises or linear strength training be used as a therapeutic tool [17]. The recent research on resistance training programs, however, offers conflicting results on the apelin, PTX3, and IL6 markers used for the early detection of cardiac risk. As a result, the aim of this study is to measure how eight weeks of resistance trainings at various intensities affects serum apelin, IL6, PTX3, CK, and LDH levels in healthy men.

Materials and methods

Participants

Participants on daily medication, who regularly consume alcohol and tobacco, have any chronic condition, or muscle and joint injuries were excluded from the study based on medical background inquiries. Forty-five sedentary male participants aged 35–45 were randomly assigned to one of the three groups: moderate-intensity exercise group (MIEG, $n=15$), low-intensity exercise group (LIEG, $n=15$), and control group (CG, $n=15$). The participants filled the nutrition-related part of the Healthy Lifestyle Behaviors Scale questionnaire [21]. The questionnaire showed no difference between the participants' daily diets; thus, they were warned not to change their eating habits during the study period.

Participants were informed about the study procedures and associated risks. All participants provided written informed consent. The Local Scientific Research Ethics Board accepted the experimental procedure (approval no: 20478486-261).

Experimental design

A repeated measures study design was used for this prospective laboratory experiment. Exercise groups were administered seven movements $\times$ two sets of bouts targeting large muscle groups, three times per week, for eight weeks, following the American College of Sports Medicine (ACSM) [22] guidelines for health and fitness. In determining the training loads, 1RM loads were estimated using the 10-repetition maximal method (10 RM) [23]. 70% of training loads for MIEG and 50% training loads for LIEG were calculated for the muscle

groups initially. Loads determined individually for each participant for different exercises. Loads were recorded on the training cards and used as a guide in subsequent training programs. In the fourth week of the exercise program, the exercise groups' 1RM loads were recalculated, and new loads of 80 and 60% were determined for MIEG and LIEG, respectively. At the end of the eighth week, all tests conducted prior to the training period were administered to all groups again. Each workout session lasted about 60 min, including a 15-min warm-up period and a 10-min cool down time. Detailed description of the resistance exercise program has been reported in Table 1.

Participants were instructed not to engage in any physical activity during the study. They did not consume alcohol or caffeine for at least 24 h before the tests, which were scheduled at least three hours after a meal.

Height and body mass were measured using standard methods while wearing minimal clothing (Seca 767, Hamburg, Germany).

Maximal strength test

After a complete 48-h rest period, strength tests were performed. While performing the analysis, participants were given 5–10 min of dynamic warm-up and 7–8 repetitions with a low resistance load to related muscle groups. The exercises consisted of seven different movements, including chest press, seated row, shoulder press, knee flexion, knee extension, biceps curl, triceps extension, abdominal crunch, back hyperextension. The rest period between sets was 1.5–2 min for LIEG; 3 min for MIEG.

Blood analysis

Following a 10- to 12-h overnight fast, venous blood samples (9 mL) were collected from an antecubital vein in the sitting position after a 20-min rest between 8:00 and 9:00 am. Serum was separated by centrifugation, and samples were stored at -80°C until a batch assessment assay was conducted in all samples (within two months).

Serum apelin, PTX3, and IL6 levels were analyzed by Enzyme-Linked Immunosorbent Assay (ELISA) method by commercial reagents Sigma-Aldrich, Saint Louis, Mo, USA. R&D Systems Bio-Techne, Minneapolis, USA and Diaclone Sas, Besancon, Cedex, France, respectively. The inter and intra-assay coefficient of variation (CV) for apelin at level 5.84 pg/mL were <15 and $<10\%$, PTX3 at level 0.025 ng/mL were 6.2 and 3.8%, for IL6 at level 2 pg/mL were 7.7 and 3.6%, respectively. Catalog numbers for Apelin, IL-6 and PTX3, respectively; RAB0018-1KT, 950.030.096, DPTX30.QC151.

CK and LDH assays were performed by enzymatic spectrophotometric methods with Advia 1800 autoanalyzer (Siemens Medical Solutions Diagnostics Limited, NY, USA). The lab experts performed daily internal quality checks and monthly external quality tests on a routine basis.

Statistical analysis

Statistical analyses were conducted on the SPSS (version 23.0, SPSS Inc, Chicago, IL, USA) statistical package program, for normality Shapiro-Wilk testing was used. The 3×3 (Group $\times$ Time) two-factor mixed-design analysis of variance (ANOVA) was used to evaluate possible interaction effects. A repeated-measures ANOVA with post-hoc Bonferroni test was used to assess differences within the groups.

Table 1: Eight-week resistance exercise program.

Exercises	MIEG			LIEG		
	Repeat	Set	Intensity	Repeat	Set	Intensity
Chest press	10	3	%70–80	10	3	%50–60
Seated row	10	3	%70–80	10	3	%50–60
Shoulder press	10	3	%70–80	10	3	%50–60
Knee flexion	10	3	%70–80	10	3	%50–60
Knee extension	10	3	%70–80	10	3	%50–60
Biceps curl	10	3	%70–80	10	3	%50–60
Triceps extension	10	3	%70–80	10	3	%50–60
Abdominal crunch	10	3	%70–80	10	3	%50–60
Hyperextension	10	3	%70–80	10	3	%50–60

One-way ANOVA with posthoc Tukey test was used to assess differences between groups. The Pearson correlation analysis test was used to examine the association between BMI and metabolic parameters in each group. Descriptive statistics were reported as the mean $\pm$ SD. The statistical significance level was set to $\alpha < 0.05$.

Results

No significant Group $\times$ Time interaction effect was found for mass [F(3.41, 72.1) = 2.591, $p=0.052$, $\eta_p^2 = 0.110$], IL6 [F(3.45, 72.4) = 0.885, $p=0.465$, $\eta_p^2 = 0.040$], CK [F(4, 84) = 0.213, $p=0.931$, $\eta_p^2 = 0.010$], LDH [F(3.51, 73.8) = 0.748, $p=0.546$, $\eta_p^2 = 0.034$] levels, indicating that changes in these variables over the course of the study showed similar patterns in the exercise and control groups. However, it was shown significant differences regarding the time for BMI [F(3.42, 71.8) = 3.002, $p=0.030$, $\eta_p^2 = 0.125$], apelin [F(3.41, 71.5) = 44.564, $p=0.000$, $\eta_p^2 = 0.680$] and PTX3 [F(3.29, 69.1) = 3.961, $p=0.009$, $\eta_p^2 = 0.159$] levels.

A significant improvement was observed in all measured strength exercises: Chest press [F(2.45, 51.5) = 92.337, $p=0.000$, $\eta_p^2 = 0.815$], seated row [F(2.94, 61.8) = 46.295, $p=0.000$, $\eta_p^2 = 0.688$], shoulder press [F(2.70, 56.6) = 24.778, $p=0.000$, $\eta_p^2 = 0.541$], knee flexion [F(2.33, 48.9) = 62.526, $p=0.000$, $\eta_p^2 = 0.749$], knee extension [F(2.74, 57.4) = 30.457, $p=0.000$, $\eta_p^2 = 0.592$], biceps press [F(2.97, 62.4) = 14.476, $p=0.000$, $\eta_p^2 = 0.408$], triceps extension [F(2.38, 50.1) = 49.618, $p=0.000$, $\eta_p^2 = 0.703$], crunch [F(3, 54.5) = 119.271, $p=0.000$, $\eta_p^2 = 0.850$], and hyperextension [F(2.35, 49.4) = 32.671, $p=0.000$, $\eta_p^2 = 0.609$].

Table 2 shows the total values and percentage deviations from the physical and biochemical analyses of the

groups before (first measurement), during (second measurement, fourth week), and after (third measurement) resistance training with various intensities. As a result, at the conclusion of the 8-week training program, there was a substantial reduction in body mass in MIEG ($p=0.026$) and BMI ($p=0.039$). The level of apelin in MIEG decreased significantly in the third measurement relative to the first and second measurements ($p < 0.001$). In both MIEG and LIEG, the PTX3 level decreased dramatically in the final measurement relative to the first and second measurements ($p < 0.05$). When the collected results are compared according to percentage differences between groups; after the training period, substantial increases in body mass, BMI, and apelin level were found in MIEG compared to CG, and in PTX3 level in MIEG and LIEG compared to CG.

When the training period is assessed in terms of strength performance, there is a substantial increase in the second and last measurements relative to the first measurement in MIEG and LIEG in all muscle groups measured. When the obtained findings are compared between the groups; significant improvement was found in exercise groups compared to CG, with the greatest improvement in MIEG (Table 3).

For each group, there is no significant relationship between BMI and metabolic parameters.

Discussion

This main finding of this research is the significant improvement in body mass, BMI, apelin and PTX3 levels after 8-weeks of moderate-intensity resistance training, as well as enhanced PTX3 levels after low-intensity resistance training. Although there were differences in MIEG levels, there was no statistically significant change in IL6, an inflammatory marker, or CK and LDH levels, muscle

Table 2: Change of physical and cardiovascular risk markers after eight wk of resistance training with different intensities.

Parameters	Groups	wk 0	wk 4	Δ 0–4 wk	wk 9	Δ 0–9 wk	p (RM)	Post hoc
Body mass, kg	MIEG	86.2 ± 9.72	85.6 ± 9.20	−0.61	84.8 ± 9.71	−1.67	0.007*	1>3 (p=0.026)
	LIEG	86.3 ± 7.48	86.3 ± 7.70	0.01	85.8 ± 7.26	−0.51	0.198	
	CG	85.1 ± 8.64	85.0 ± 8.30	0.0	85.1 ± 8.78	0.08	0.948	
	p (BG)			0.297		0.019 ^f		
	Post hoc					1<3 (p=0.017)		
BMI, kg/m ²	MIEG	27.8 ± 2.97	27.6 ± 2.82	−0.63	27.3 ± 3.08	−1.69	0.013*	1>3 (p=0.039)
	LIEG	27.7 ± 2.50	27.7 ± 2.63	−0.18	27.5 ± 2.38	−0.68	0.105	
	CG	25.7 ± 2.06	25.7 ± 2.09	−0.07	25.8 ± 2.02	0.31	0.567	
	p (BG)			0.417		0.009 ^f		
	Post hoc					1<3 (p=0.007)		
Apelin, pg/mL	MIEG	1,704 ± 202	1,653 ± 231	−2.98	936 ± 17.2	−44.0	<0.001*	1>3 (p<0.001), 2>3 (p<0.001)
	LIEG	1,104 ± 371	993 ± 220	−5.43	982 ± 225	−6.88	0.179	
	CG	1,613 ± 76.6	1,649 ± 166	2.19	1,647 ± 89.8	2.14	0.425	
	p (BG)			0.245		<0.001 ^f		
	Post hoc					1>3 (p<0.01)		
PTX3, ng/mL	MIEG	2.89 ± 1.42	2.73 ± 1.32	0.24	1.55 ± 1.19	−39.3	<0.001*	1>3 (p=0.001), 2>3 (p=0.003)
	LIEG	2.10 ± 1.09	2.08 ± 1.0	5.11	1.50 ± 0.94	−24.6	0.004*	
	CG	2.01 ± 1.32	2.35 ± 1.29	35.5	2.71 ± 1.88	74.2	0.487	
	p (BG)			0.163		<0.001 ^f		
	Post hoc					1<3 (p=0.003), 2<3 (p=0.011)		
IL6, pg/mL	MIEG	2.66 ± 1.56	2.50 ± 1.22	10.1	1.90 ± 0.76	−20.9	0.222	
	LIEG	2.74 ± 0.65	2.86 ± 0.89	13.0	2.39 ± 0.69	−7.76	0.165	
	CG	1.92 ± 0.75	2.07 ± 0.73	12.4	1.99 ± 0.96	16.7	0.685	
	p (BG)			0.988		0.137		
	Post hoc							
CK, U/L	MIEG	202 ± 97.1	222 ± 150.9	38.6	205 ± 80.1	17.9	0.856	
	LIEG	230 ± 188	236 ± 188	4.67	253 ± 197	22.9	0.447	
	CG	163 ± 95.9	174 ± 90.0	10.9	171 ± 84.5	10.9	0.619	
	p (BG)			0.517		0.796		
	Post hoc							
LDH, U/L	MIEG	194 ± 43.0	211 ± 31.5	10.9	202 ± 43.8	7.41	0.465	
	LIEG	216 ± 50.1	224 ± 45.1	4.77	208 ± 58.5	−2.48	0.368	
	CG	178 ± 42.1	191 ± 40.1	12.9	196 ± 44.1	15.7	0.174	
	p (BG)			0.602		0.303		
	Post hoc							

BMI, Body mass index; PTX3, pentraxin 3; IL6, interleukin 6; CK, creatine kinase; LDH, lactate dehydrogenase; RM, repeated measures ANOVA, *p<0.05, wk 0 (1), wk 4 (2), wk 9 (3); BG, between groups (One-way ANOVA), ^fp<0.05, Week (wk).

damage markers, in the exercise groups. Following the exercise programs, the strength test parameters improved dramatically.

Obesity causes low levels of inflammation, which leads to an excess of adipokine secretion, which increases the inflammation and reduces insulin sensitivity. This mechanism supports the studies on obese individuals by clearly demonstrating the therapeutic impact of exercising [24]. However, it is unclear how different intensity resistance exercises affect apelin, PTX3, and IL6 levels, which are considered as cardiovascular risk markers in healthy individuals.

Since there is a lot of scientific evidence on the health benefits of resistance exercises, ACSM recommends that they should be integrated into fitness programs for people of all ages [23, 25, 26]. Strength development is a process that requires the synchronized functioning of both neural and muscular mechanisms. The strength training time for this study was set at 8 weeks because significant changes in maximal dynamic strength are known to be attained with 6–21 weeks of resistance exercise, as well as neuronal adaptations, which are considered to be responsible for the initial increase of strength, arise in the first 6–8 weeks [27].

Table 3: Chance of strength output after eight wk of resistance training with different intensities.

Parameters	Groups	wk 0	wk 4	Δ 0–4 wk	wk 9	Δ 0–9 wk	p (RM)	Post hoc
Chest press, kg	MIEG	39.0 ± 5.07	49.0 ± 5.07	26.1	59.7 ± 3.52	55.1	0.000*	1<2, 1<3, 2<3 (p<0.001)
	LIEG	41.0 ± 4.31	44.7 ± 5.81	8.72	50.7 ± 6.23	23.5	0.000*	1<2, 1<3, 2<3 (p<0.001)
	CG	45.0 ± 7.79	45.0 ± 7.79	0.0	45.7 ± 8.63	1.29	0.381	
	p (BG)			<0.001 ^f				<0.001 ^f
	Post hoc			1>2, 1>3, 2>3 (p<0.001)				1>2, 1>3, 2>3 (p<0.001)
Seated row, kg	MIEG	43.3 ± 7.48	51.3 ± 6.40	19.5	60.0 ± 4.42	41.6	0.000*	1<2, 1<3, 2<3 (p<0.001)
	LIEG	35.0 ± 4.63	39.3 ± 5.63	12.7	46.0 ± 6.04	32.3	0.000*	1<2, 1<3, 2<3 (p<0.001)
	CG	38.7 ± 6.67	38.7 ± 6.67	0.0	38.7 ± 6.67	0.57	1.000	
	p (BG)			<0.001 ^f		<0.001 ^f		
	Post hoc			1>3, 2>3 (p<0.001)				1>3, 2>3 (p<0.001)
Shoulder press, kg	MIEG	19.0 ± 5.41	23.0 ± 6.21	22.7	30.0 ± 5.67	63.3	0.000*	1<2, 1<3, 2<3 (p<0.001)
	LIEG	18.3 ± 3.62	20.7 ± 4.95	13.6	26.3 ± 5.82	46.2	0.000*	1<2, 1<3, 2<3 (p<0.001)
	CG	18.3 ± 4.08	17.7 ± 3.72	-2.11	16.7 ± 4.50	-7.67	0.180	
	p (BG)			0.009 ^f		<0.001 ^f		
	Post hoc			1>3 (p=0.007)				1>3, 2>3 (p<0.001)
Knee flexion, kg	MIEG	40.0 ± 3.78	47.7 ± 3.72	19.6	55.0 ± 4.63	38.4	0.000*	1<2, 1<3, 2<3 (p<0.001)
	LIEG	38.7 ± 5.50	38.7 ± 5.50	0.0	43.7 ± 5.50	13.2	0.000*	1<3, 2<3 (p<0.001)
	CG	39.7 ± 6.40	39.7 ± 6.40	0.0	38.3 ± 7.24	-3.33	0.272	
	p (BG)			<0.001 ^f		<0.001 ^f		
	Post hoc			1>2, 1>3 (p<0.001)				1>2, 1>3, 2>3 (p<0.001)
Knee extension, kg	MIEG	40.3 ± 5.16	46.7 ± 4.88	16.2	53.7 ± 4.81	34.1	0.000*	1<2, 1<3, 2<3 (p<0.001)
	LIEG	38.3 ± 5.56	40.3 ± 4.81	5.82	45.3 ± 4.81	19.1	0.000*	1<2 (p=0.026) 1<3, 2<3 (p<0.001)
	CG	37.0 ± 3.68	37.0 ± 3.68	0.0	38.0 ± 5.92	2.71	0.516	
	p (BG)			<0.001 ^f		<0.001 ^f		
	Post hoc			1>2, 1>3 (p<0.001), 2>3 (p=0.030)				1>2 (p=0.030), 1>3, 2>3 (p ≤ 0.001)
Biceps press, kg	MIEG	11.3 ± 3.64	14.7 ± 3.52	35.6	19.7 ± 3.52	84.4	0.000*	1<2, 1<3, 2<3 (p ≤ 0.001)
	LIEG	11.2 ± 4.32	11.8 ± 4.17	7.78	16.3 ± 4.81	51.1	0.000*	1<3, 2<3 (p<0.001)
	CG	15.0 ± 4.53	15.0 ± 4.53	0.0	16.7 ± 4.40	15.0	0.136	
	p (BG)			<0.001 ^f		<0.001 ^f		
	Post hoc			1>2 (p=0.002), 1>3 (p=0.002)				1>2 (p=0.027), 1>3 (p<0.000), 2>3 (p=0.015)
Triceps extension, kg	MIEG	19.0 ± 3.87	25.3 ± 2.97	36.2	31.7 ± 3.62	72.4	0.000*	1<2, 1<3, 2<3 (p<0.001)
	LIEG	13.0 ± 3.68	14.0 ± 4.31	7.78	19.0 ± 4.31	48.9	0.000*	1<3, 2<3 (p<0.001)
	CG	14.3 ± 3.72	14.3 ± 3.72	0.0	15.3 ± 4.42	6.67	0.082	
	p (BG)			<0.001 ^f		<0.001 ^f		
	Post hoc			1>2, 1>3, 2>3 (p<0.001)				1>3, 2>3 (p<0.001)
Crunch, kg	MIEG	23.0 ± 4.42	29.0 ± 5.07	44.9	39.0 ± 5.07	96.4	0.000*	1<2, 1<3, 2<3 (p<0.001)
	LIEG	17.7 ± 6.78	19.3 ± 6.23	13.3	24.7 ± 6.67	48.6	0.000*	1<3, 2<3 (p<0.001)
	CG	17.7 ± 6.51	17.7 ± 6.61	0.0	18.0 ± 6.49	3.33	0.730	
	p (BG)			<0.001 ^f		<0.001 ^f		
	Post hoc			1>2, 1>3 (p<0.001)				1>2, 1>3, 2>3 (p<0.001)
Hyperextension, kg	MIEG	20.7 ± 4.58	28.0 ± 4.55	38.3	35.3 ± 5.81	76.7	0.000*	1<2, 1<3, 2<3 (p<0.001)
	LIEG	21.0 ± 6.04	22.0 ± 5.61	6.67	25.7 ± 6.78	26.7	0.005*	1<3 (p=0.023), 2<3 (p=0.009)
	CG	21.7 ± 6.72	21.7 ± 6.72	0.0	21.3 ± 6.67	0.33	0.875	
	p (BG)			<0.001 ^f		<0.001 ^f		
	Post hoc			1>2, 1>3 (p<0.001)				1>2, 1>3 (p<0.001)

RM, repeated measures ANOVA, Week (wk) *p<0.05, wk 0 (1), wk 4 (2), wk 9 (3); BG, between groups (One-way ANOVA), ^fp<0.05.

Resistance training improves body composition by increasing fat oxidation, energy consumption, and lipolysis in the abdominal fat tissue. They also increase oxygen utilization capacity and provide negative energy balance by rising resting metabolic rate and lean mass. Improved

oxygen utilization capacity raises the amount of oxygen consumed by skeletal muscles, which increases fat oxidation and reduces the size of fat cells, and their content [28]. Correspondingly, it is reported that regular resistance training of varying intensity ranges adopted by healthy and

obese individuals or coronary heart disease patients of various ages resulted in a significant decrease in body mass and BMI and a significant increase in strength level, which is consistent with our research findings [29–32].

According to the existing data, the acute response to resistance training results in increased myokine secretion, while long-term adaptations result in lower basal levels of certain inflammatory cytokines [25]. In our research, we founded that four weeks of resistance exercise in MIEG was insufficient to improve apelin levels, while eight weeks of resistance exercise resulted in a substantial decrease in apelin levels. It has been shown in two studies investigating the relationship between apelin and exercise in the literature that an 8-week aerobic exercise regimen conducted on a daily basis (3 days/week) lowered plasma apelin [33] and insulin levels. Also, BMI and body fat rate decreased proportionally with apelin [34]. However, body mass, BMI, and waist to hip ratio were significantly reduced after circuit resistance training intervention in obese young men, but apelin plasma concentration did not improve significantly [24]. Another research investigated how 8 weeks of aerobic and resistance exercise affected apelin-12 and apelin-36 levels in middle-aged obese women. The apelin-12 levels changed significantly in both the aerobic and resistance exercise groups. They found it to be beneficial in treating obesity and lowering blood apelin-12 concentrations, which are linked to metabolic syndrome markers [35]. Although there is broad variation in the quantification of myokines caused by exercise among published papers (which may be due to timing of sample selection, pre-analytic sample processing, analytical technique, and estimation, and other factors [36, 37] such as form, volume, or strength of exercise), it can be assumed that apelin levels are positively associated with BMI. Furthermore, when the study findings are analyzed in-depth, it can be stated that an increase in apelin is found in cases of decreased insulin resistance and improved lipid and glycemic profile after the exercise program. The finding of insulin resistance attenuation and LDL reduction as independent predictors of exercise-induced alterations in apelin levels also supports these ideas.

Studies examining the relationship between PTX3, which is a new indicator of inflammation, and regular exercise programs are scarce in the literature. Existing findings demonstrated that trained endurance athletes had higher plasma PTX3 levels than the control group [38], and an aerobic exercise program increased PTX3 levels in postmenopausal women [39]. Fukuda et al. [40] reported that 3–6 months of cardiac rehabilitation using aerobic exercise in patients with cardiovascular disease decreased plasma PTX3 level. Multivariate regression models in a

cohort sample of healthy people over the age of 65 indicated that the correlation between higher levels of physical activity and lower levels of inflammation markers might be mediated by body mass index and glucose [30]. The effect of combined exercise training on pentraxins and pro-inflammatory cytokines was investigated in patients with multiple sclerosis. The level of PTX3 was stated to have improved as a part of a 12-week program that included resistance, endurance, balance, and stretching exercises [41]. Our results revealed that the 4-week resistance training program in the exercise groups was ineffective in terms of changing body mass and PTX3 level. On the other hand, by the end of eight weeks, it had greatly reduced all parameters.

IL6 is a pleiotropic cytokine that can act as a pro- or an anti-inflammatory messenger depending on the situation it is produced. Adequate loads of exercise provoke the acute release of cytokines, especially IL6 [25]. In a major cross-sectional sample, participants in moderate-intensity exercises or aerobic + resistance exercises are found to associate with a higher degree of inflammation than participants in high-intensity or just aerobic exercises, provided the same average MET-hour physical activity [42]. In accordance with our results, 6-weeks of intense resistance training was found to be adequate to reduce inflammatory markers [43]. The general idea is that recurrent exercise in IL6 has inflammatory-reducing effects reflected in low basal inflammatory levels [25]. However, two separate 6-week resistance training programs (45–55% vs. 80–90% 1RM) [44], a 10-week resistance or aerobic exercise regimen [20], and 12 weeks of resistance training with linear periodization [45] were found not to yield improvements in IL6. In young men, IL6 increased three hours after exercise and remained unchanged after 12 weeks of resistance training. As a result, it has been stated that intense exercise temporarily raises IL6 level and maintained this effect until the end of the exercise period [46]. In our research, we discovered a 20% decrease in IL6. While this decrease is not statistically significant, it supports previous studies that found a positive impact on IL6.

Our research showed that 8 weeks of resistance training had little effect on CK and LDH levels. We believe that the adaptation provided by regular repetitions with low exercise loads has an effect on the results. Adaptations behind the repeated exercise effect are considered to include a shift towards greater recruitment of slow-twitch motor units and the generation of new sarcomeres in series. This shift reduces the extent of microtrauma and downregulation of inflammation, which would limit the degree of post-exercise cell damage in the following days after the exercise [7].

One potential limitation of this study is that the results cannot be extended to other age ranges since the research was only performed on 35–45-year-old males. Furthermore, since participants were not checked for cardiovascular health during this study, investigating the impact of exercise on cardiovascular health warrants additional research.

Eight weeks of regular low and moderate-intensity resistance training resulted in significant strength development. The MIEG group experienced a greater increase in strength. Moderate-intensity resistance training had positive effects on body mass, BMI, apelin, and PTX3 levels, whereas low-intensity resistance training enhanced PTX3 levels. According to our findings, cardiac risk can be reduced in healthy men with moderate-intensity resistance exercises. Further studies are required to elucidate the beneficial effects of resistance training on cardiovascular risk markers in other populations (e.g., children, women, and the elderly).

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