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ORIGINAL ARTICLE

Comparing the effects of resistance exercise type on serum levels of oxidative stress and muscle damage markers in resistance-trained women

Sara Motameni[1] · Hossein TaheriChadorneshin[1] · Ali Golestani[1]

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Abstract

Purpose The aim of the current study is to compare the effects of hypertrophy-, strength-, and power-type resistance exercise training types on hydrogen peroxide (H_2O_2), malondialdehyde (MDA), total lactate dehydrogenase (LDH), and total creatine kinase (CK) in resistance-trained women.

Methods After determining one-repetition maximum (1-RM), ten resistance-trained women (age 26.30 ± 4.95 years; body mass index 22.07 ± 2.02 kg/m^2; body fat 24.64 ± 4.98%) conducted hypertrophy-type (70% of 1-RM), strength-type (90% of 1-RM), and power-type (45% of 1-RM) resistance exercise for three consecutive weeks. The movements included lever leg extension, reverse-grip lat pull-down, horizontal leg press, standing biceps cable curl, lying leg curl, machine bench press, standing cable triceps extension, and seated calf raises. Fasting blood samples were obtained immediately before and immediately after each trial. Statistical analyses were performed using the *t* test, Wilcoxon, and analysis of covariance. The significance level was set at $P < 0.05$ level.

Results The results indicated that one bout of hypertrophy-, strength-, and power-type resistance exercises had no significant effects on H_2O_2, MDA, and total LDH levels. However, serum total CK level significantly increased after all the three types of resistance exercise. Power resistance exercise resulted in a higher total CK level than hypertrophy and strength types.

Conclusion Although the three types of hypertrophy, strength, and power exercise cause muscle damage, they do not exacerbate oxidative stress in resistance-trained women.

Keywords Resistance exercise · Hydrogen peroxide · Malondialdehyde · Lactate dehydrogenase · Creatine kinase

Introduction

A free radical (FR) is a chemical species that contains one or more unpaired electrons and reacts with other molecules to achieve stability. It is now acknowledged that low-to-moderate levels of FR have multiple regulatory roles in the cell, including the modulation of cell signaling pathways and the regulation of gene expression, while high levels of FR can damage cellular structures [1]. Moreover, it is well established that skeletal muscles are a significant source of oxidant production during exercise and that they contribute to exercise-induced oxidative stress [2]. Oxidative stress refers to an imbalance between the production of reactive oxygen species and a biological system's ability to readily detoxify them or to repair the resulting damage [3]. Superoxide, as a primary FR, is produced through an incomplete reduction of oxygen in the electron transport system and can be easily converted to hydrogen peroxide (H_2O_2) spontaneously or by action of the superoxide dismutase (SOD) [1, 2]. H_2O_2 as a stable oxidant is considered a relatively weak cytotoxic and oxidizing agent [1]. Because of long half-life, H_2O_2 diffuses within cells and across cell membranes [1]. H_2O_2 is unable to oxidize DNA or lipids directly [1]. However, H_2O_2 can readily generate hydroxyl radicals through the Fenton reaction, which in turn results in a threefold increase in malondialdehyde (MDA) as the most mutagenic product of lipid peroxidation [4, 5]. MDA is the primary indicator of aldehyde that results from polyunsaturated fatty acids peroxidation and is frequently used as a biomarker of oxidative stress in response to exercise and clinically serious metabolic

✉ Hossein TaheriChadorneshin
h.taheri@ub.ac.ir

[1] Department of Sport Sciences, University of Bojnord, Bojnord 9453155111, Islamic Republic of Iran

impairments [5]. It has been concluded that an increase in FRs results in MDA overproduction [5]. Cell membrane damage and muscle damage induced by FR attacks [6, 7] increase the instability of cell membrane, which in turn increases creatine kinase (CK) and lactate dehydrogenase (LDH) releases into the bloodstream [8]. In this regard, it has been revealed that subjects with a greater increase in markers of muscle damage (i.e., CK and LDH) experience a more considerable increase in serum concentration after eccentric exercise [4]. Moreover, it has been reported that post-race increase in plasma MDA levels significantly correlates with increased plasma CK and LDH levels [5].

With regard to oxidative stress markers, it has been demonstrated that five sets of 15 eccentric maximal voluntary contractions on an isokinetic dynamometer result in more MDA levels in untrained than resistance-trained individuals [9]. In addition, one bout of Olympic weightlifting at an intensity corresponding with 85–90% one-repetition maximum (1RM) has been reported to increase serum MDA levels significantly and to remain elevated 48 h after the morning training session in elite weightlifters [10]. Moreover, plasma MDA levels have reportedly increased after three sets of upper- and lower-body resistance exercise at low intensity (25–30% 1RM) in untrained male students [11]. In contrast, both chronic hypertrophy- and strength-intensity whole-body resistance decrease MDA concentration in previously untrained men [12]. In the context of muscle damage markers, no significant change has been observed in serum CK levels up to 48-h post-exercise after one acute bout of intense back squat exercise (10 sets of 10 repetitions at 70% of 1RM [13]. In contrast, it has been revealed that a single bout of 5 resistance exercises (3 sets of 10 maximum repetitions) leads to significant increases in serum CK and LDH up to 48-h after exercise without isometric strength decrease in sedentary men [14]. Upper-body resistance exercise (3 sets at 8-repetition maximum) has also been associated with an increase in muscle damage biomarkers, CK, and LDH after training in both trained and untrained men. Unlike CK, the activity of LDH reduces during 1 h of training [15]. Both high-intensity (8 sets of 3 repetitions at 90% 1RM) and high-volume (8 sets of 10 repetitions at 70% 1RM) resistance exercises significantly elevate markers of muscle damage (CK and LDH) 30-min and 24-h post-exercise in resistance-trained men [16].

Resistance training is a well-established mode of exercise conditioning for many different populations wishing to increase physical fitness. Before entering the competition phase, athletes perform three types of resistance exercises to increase muscle hypertrophy, strength, and power. These characteristics are achieved through changes in resistance exercise variables such as intensity, number of repetitions, total work, and rest intervals. It has been shown that different types of resistance-training exercises induce different responses from muscles and the neurological system [17, 18]. However, to the best of our knowledge, the effects of different types of resistance exercise on markers of oxidative stress and muscle damage have not yet been well examined. Conducting such protocols will increase the knowledge and insights of athletes about three types of resistance exercises and will help them to make possible modifications in their resistance exercise programs and take appropriate nutritional approaches to reduce oxidative stress and muscle damage. Hence, the aim of the present study was to compare the effects of hypertrophy-, strength-, and power-type resistance exercises on markers of oxidative stress (H_2O_2, MDA) and muscle damage (CK, LDH) in resistance-trained women.

Materials and methods

Participants

The research protocol was approved by the Institutional Review Board (University of Bojnord, Iran) prior to subject enrollment. Ten healthy young resistance-trained women (26.30 ± 4.95 years old) volunteered to participate in the current quasi-experimental study. Subjects were informed via advertisements placed in gyms as well as information disseminated via Telegram groups. Participants were selected according to the following criteria: [1] living in Mashhad (Northeast of Iran), [2] being 20 years old or older, [3] performing regular and recreational resistance exercises for 3 days per week in the past one year, and [4] having regular menstrual periods. Exclusion criteria were cardiovascular diseases, especially blood pressure; use of any medication, hormones, or nutritional supplements; smoking; alcohol consumption; and pregnancy. The following ethical considerations were considered in the present study: [1] All participants signed written informed consent forms after they had been informed of all risks, discomforts, and benefits involved; [2] participants agreed to participate in the study on a voluntary nature; [3] subjects were allowed to leave the protocol without penalty at any time; and, finally, [4] the study was performed in accordance with the 1975 Declaration of Helsinki and its 1996 revision. Before the beginning of the exercise protocol, the anthropometric profiles of the subjects were measured by a body composition analyzer (X-Contact 356 model, Jawon medical Co., Ltd. South Korea) in light indoor clothing. The participants' body composition profiles are presented in Table 1. The schematic timeline for the experimental protocol is displayed in Fig. 1.

Determination of 1-RM

1-RM loads were established for all agonist and antagonist muscle groups in two separate sessions one week

Table 1 Participants' body composition profiles

Body composition	Mean ± SD
Weight (kg)	59.51 ± 7.78
Height (cm)	164 ± 7.22
LBM (kg)	44.73 ± 5.60
SLM (kg)	41.31 ± 5.21
TBW (kg)	32.19 ± 4.02
MBF (kg)	14.78 ± 3.97
BMI (kg/m2)	22.07 ± 2.02
BFP (%)	24.64 ± 4.98
SMM (kg)	26.92 ± 7.04
WHR	0.75 ± 0.05
AC (cm)	73.26 ± 4.67
ECW/TBW	0.38 ± 0.005

AC abdominal circumference, *BFP* body fat percentage, *BMI* body mass index, *ECW/TBW* extracellular water to total body water, *LBM* lean body mass, *MBF* mass of body fat, *SMM* skeletal muscle mass, *SLM* soft lean mass, *TBW* total body water, *WHR* waist-to-hip ratio

before the experimental intervention initiated. Strength tests were always preceded by 5-min warm-up on a cycle ergometer. Then, the participants lifted light weights to warm up upper- and lower-body muscle groups. Weights were selected according to the participant so that the participants could lift the weights at least once and up to 10 times. Finally, subjects' 1RM strength was determined by the Brzycki equation as follows: 1RM = Weight/[1.027 − (0.027 × Number of repetitions)] [12]. The results of 1-RM are presented in Table 2.

Table 2 Participant's 1-RM data

Resistance exercise	Mean ± SD
Lever leg extension (kg)	57.84 ± 12.92
Reverse-grip lat pull-down (kg)	39.23 ± 6.61
Horizontal leg press (kg)	105.09 ± 19.55
Standing biceps cable curl (kg)	27.26 ± 5.82
Lying leg curl (kg)	33.91 ± 10.12
Machines bench press (kg)	29.92 ± 7.18
Standing cable triceps extension (kg)	31.92 ± 6.86
Seated calf raise (kg)	17.12 ± 4.83

Resistance exercise protocol

Resistance exercises were conducted in three sessions one week apart under staff supervision [19, 20]. The resistance exercise protocol involved upper- and lower-body exercises, which were performed at 8 stations (lever leg extension, reverse-grip lat pull-down, horizontal leg press, standing biceps cable curl, lying leg curl, machine bench press, standing cable triceps extension, and seated calf raise). These exercises require all the muscle groups and major joints involved in strength training interventions [21]. Participants performed hypertrophy-type resistance exercise for 3 sets of 10–12 repetitions at an intensity corresponding to 70% of 1-RM with a 90-s rest period between sets, whereas the strength-type resistance exercise was performed for 3 sets of 3–5 repetitions at an intensity corresponding to 90% of 1-RM with a 120-s rest period between sets [12]. In addition, power-type resistance exercise was performed for 3 sets of 8–10 repetitions at an intensity corresponding to 45%

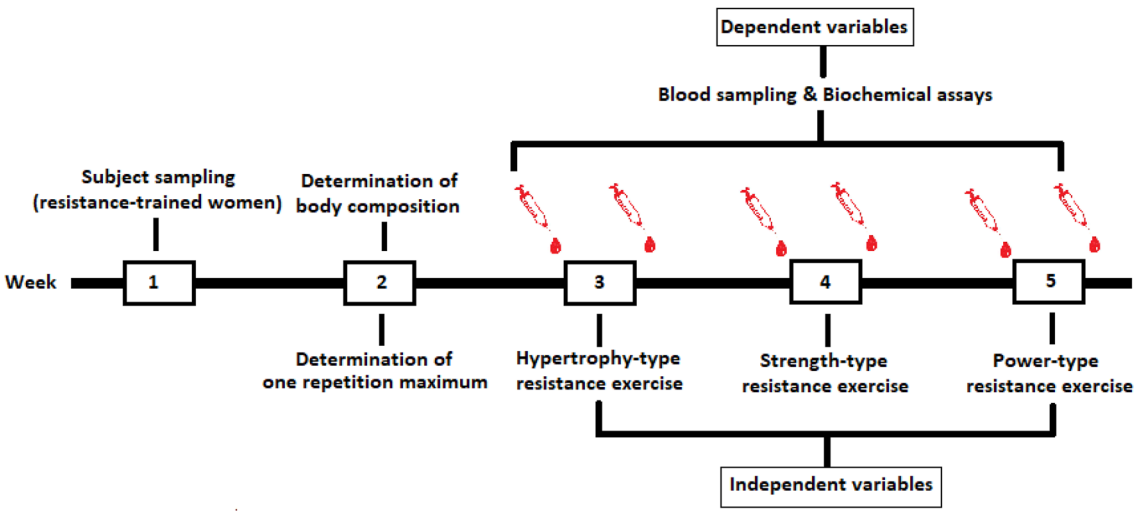

Fig. 1 Protocol timeline

of 1-RM with a 180-s rest period between sets [22]. The velocity of movement during hypertrophy- and strength-type resistance exercises was moderate; however, power-type resistance exercise was performed as explosive [12]. Each session started with a 10-min warm-up and ended by 5-min cold-down. Participants were prohibited from any exercise for 48 h after each protocol. In addition, they were contacted regularly during the protocol to ensure that they were not engaged in any resistance exercise [22].

Biochemical assays

Seven milliliters of fasting blood samples was obtained from the participants' antecubital vein immediately before and immediately after each resistance exercise. Samples were centrifuged (Universal Model, Behdad Corporation, Iran) with 1500 rpm for 10 min at 4 °C [23]. The serum was stored at − 80 °C until it was used. We employed the commercially 96-well colorimetric assay kits to measure the serum H2O2 (#ZB-HPO-96A, ZellBio GmbH, German) and serum MDA (#ZB-MDA-96A, ZellBio GmbH, German). The sensitivities of the kits were 5 and 0.1 µM for H_2O_2 and MDA, respectively. In addition, both serum total CK and total LDH assays were carried out by the photometric assay method using a commercial kit (Pars Azmoon Co., Karaj, Iran) with a 5 U/L assay sensitivity. All analyses were performed in accordance with the manufacturers' recommendations and measured by BioTek microplate reader (Epoch 2 model, USA).

Diet considerations

A week before the protocol initiated, a 24-h diet recall interview was conducted for three consecutive days to analyze the participants' diets. The results demonstrated that the calorie intake from carbohydrate, protein, and fat was 58%, 15%, and 27%, respectively. In addition, the subjects had no experience of weight changes more than ± 1 kg in the past 6 months. In addition, subjects were asked to have carbohydrate loading for three days prior to each resistance exercise trial. The subjects were asked to refrain from consuming drinks containing alcohol, caffeine, or any other nutritional supplementation or pharmacological interventions during the resistance exercise protocol.

Statistical analysis

All statistical analyses were performed using the 16.0 version of SPSS (statistical package for social sciences, SPSS Inc). The normality of data distribution was assessed by the Shapiro–Wilk test. Wilcoxon and paired *t* test were used to examine the intra-group differences for nonparametric and parametric data, respectively. In addition, analysis of covariance and Bonferroni post hoc tests were used to examine the inter-group differences. Data are expressed as mean ± standard error. Finally, *P* values smaller than 0.05 were considered statistically significant.

Results

With regard to stress oxidative markers, intra-group comparisons showed that hypertrophy- (99.41 ± 5.48 and 81.71 ± 8.87 µM for pre- and post-test, respectively) ($P = 0.377$), strength- (83.08 ± 7.18 and 87.77 ± 9.01 µM for pre- and post-test, respectively) ($P = 0.753$), and power-type resistance exercises (67.52 ± 8.33 and 67.52 ± 6.85 µM for pre- and post-test, respectively) ($P = 0.093$) made no significant changes in serum H_2O_2 levels (Fig. 2a). In addition, intra-group comparisons showed that hypertrophy- (35.48 ± 1.70 and 31.12 ± 1.34 µM for pre- and post-test, respectively) ($P = 0.063$), strength- (35.97 ± 2.35 and 33.71 ± 0.59 µM for pre- and post-test, respectively) ($P = 0.449$), and power-type resistance exercises (33.39 ± 2.06 and 33.17 ± 2.19 µM for pre- and post-test, respectively) ($P = 0.680$) made no significant changes in serum MDA levels (Fig. 2b). Furthermore, no significant difference was observed between serum H_2O_2 ($P = 0.796$)

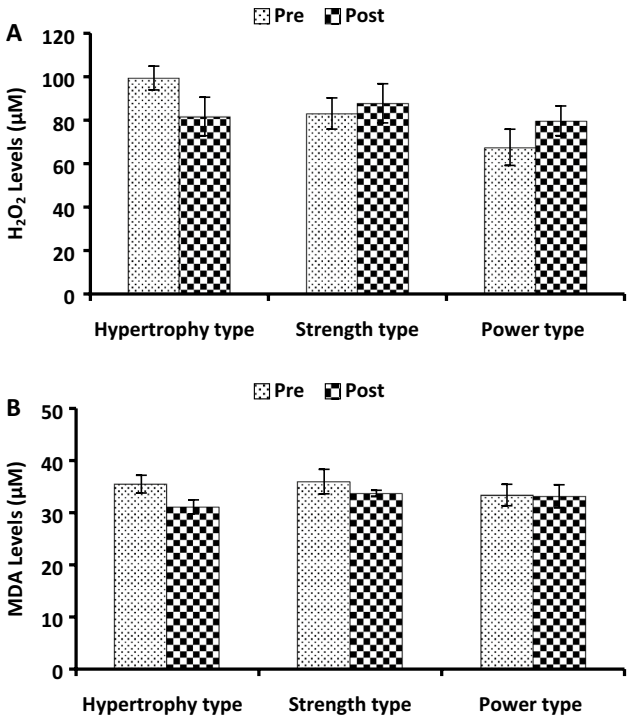

Fig. 2 Serum H_2O_2 (**a**) and MDA (**b**) concentrations for hypertrophy-, strength-, and power-type resistance exercises. H_2O_2 hydrogen peroxide; *MDA* malondialdehyde. Data are presented as mean ± standard error

and MDA ($P=0.562$) responses to hypertrophy-, strength-, and power-type resistance exercises (Fig. 2a, b).

In the context of muscle damage markers, intra-group comparisons revealed that hypertrophy- (34.73 ± 5.45 and 42.30 ± 5.61 U/L for pre- and post-test, respectively) ($P=0.002$), strength- (33.01 ± 5.01 and 38.65 ± 5.71 U/L for pre- and post-test, respectively) ($P=0.020$), and power-type (27.20 ± 4.02 and 46.95 ± 3.75 U/L for pre- and post-test, respectively) ($P=0.001$) resistance exercises induced a significant increase in serum total CK. Besides, power-type resistance exercise resulted in a remarkable increase in serum total CK compared to the hypertrophy- ($P=0.001$) and strength-type ($P=0.001$) resistance exercises (Fig. 3a). In contrast, intra-group comparisons showed that hypertrophy- (146.94 ± 11.15 and 162.43 ± 20.48 U/L for pre- and post-test, respectively) ($P=0.838$), strength- (131.98 ± 10.47 and 182.20 ± 34.87 U/L for pre ad post, respectively) ($P=0.065$), and power-type resistance exercises (125.86 ± 9.63 and 149.61 ± 17.98 U/L for pre- and post-test, respectively) ($P=0.207$) had no significant effect on serum total LDH levels. In addition, no significant difference was found between the serum total LDH ($P=0.614$) response to resistance exercise trials (Fig. 3b).

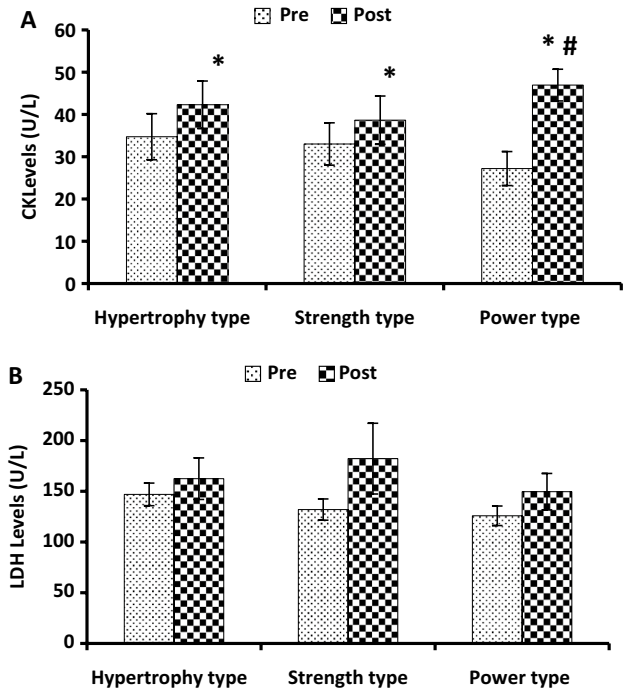

Fig. 3 Serum CK (**a**) and LDH (**b**) concentrations for hypertrophy-, strength-, and power-type resistance exercises. *CK* creatine kinase; *LDH* lactate dehydrogenase. The asterisk (*) indicates a significant difference from baseline values. The hash sign (#) indicates a significant difference from hypertrophy- and strength-type resistance exercises at the pos-test. Data are presented as mean ± standard error

Discussion

Regular exercise training has been demonstrated to have several health benefits, including lowered risks of all-cause mortality, cardiovascular disease, cancer, and diabetes. Paradoxically, it is also revealed that contracting skeletal muscles generate free radicals and that intensive exercise can cause oxidative damage to cellular compartments. However, our findings showed that hypertrophy-, strength-, and power-type resistance exercises did not produce significant changes in oxidative stress markers. Although none of the resistance exercises had a dramatic effect on LDH levels, all the three types of resistance exercises increased serum CK levels, and this increase was higher after the power-type compared to strength- and hypertrophy-type resistance exercises.

As a significant source of reactive oxygen species, H_2O_2 is a weak oxidant with a relatively long half-life; this long half-life permits diffusion within cells and across cell membranes [1]. Results of the present study demonstrated that serum H_2O_2 levels did not change significantly after the three types of resistance exercise in resistance-trained women. Acute aerobic running on the treadmill has been shown to increase mitochondrial H_2O_2 production in gastrocnemius and quadriceps femoris [24], which does not correspond with our findings. When an individual performs aerobic exercises, electron leakage at specific redox centers during mitochondrial electron transfer chain reactions is considered responsible for generating H_2O_2 [24]. Part of the differences in the results may be due to differences in exercise type and the measurement site of H_2O_2. From the production site to its placement in the serum, H_2O_2 is repeatedly exposed and converted to water by the catalase (CAT) enzyme [1]. In this regard, evidence indicates that serum CAT enzyme activity significantly increases immediately after multi-joint or single-joint resistance exercise [25]. Therefore, it appears that the reason for the non-significant change in serum H_2O_2 levels is the increased serum CAT level after resistance exercise.

Cells continuously produce FRs as part of metabolic processes. When FRs are produced, they attack polyunsaturated fatty acids in cell membranes and lead to a chain of chemical reactions called lipid peroxidation [5]. Aldehydes, especially MDA, have been frequently used as an indicator of lipid peroxidation in response to exercise [5]. MDA has been demonstrated as a primary lipid peroxidation product and can reflect the degree of cellular injury. In the current study, serum MDA levels did not significantly change immediately after resistance exercises, which suggest the non-occurrence of lipid peroxidation. Our finding is consistent with previous reports that recorded no significant change in serum MDA after one acute bout

of intense back squat exercise (10 sets of 10 repetitions at 70% of 1RM) [13] and moderate-intensity whole-body circuit resistance exercise (3 sets of 10 repetitions at 10 RM) [26] in resistance-trained males. Similarly, Park and Kwak demonstrated that a graded exercise test on a treadmill did not affect plasma MDA levels in aerobically and anaerobically trained athletes [27].

In contrast, our results are inconsistent with studies that reveal an increased level of serum MDA after submaximal circuit resistance exercise [28] and after an acute bout of upper- and lower-body resistance exercises at low intensity (3 sets of 20–30 repetitions at 25–30% of 1RM) [11] in non-resistance-trained subjects. Moreover, high-intensity circuit resistance exercise leads reportedly to a more considerable increase in serum MDA levels in comparison with the low-intensity circuit resistance exercise in sedentary males [23]. Moreover, one bout of intensive circuit whole-body resistance exercise has been shown to increase serum MDA levels in sedentary women [29]. Therefore, it appears that the differences in the subjects' physical fitness status may have contributed to the discrepancy in the results. In this context, one study has reported a non-significant difference between aerobically trained athletes, anaerobically trained athletes, and untrained individuals in terms of resting plasma MDA levels. However, the study has highlighted a significant increase in plasma MDA levels after a graded exercise test in untrained individuals, but not in aerobically and anaerobically trained athletes [27]. It is thought that the non-substantial change in MDA levels of trained subjects is associated with higher levels of antioxidant status induced by exercise training, thus preventing an increase in H_2O_2 and MDA production in response to acute resistance exercise. In this regard, it has been shown that mRNA levels of CAT, glutathione peroxidase, and both mitochondrial and cytosolic SOD increase in peripheral blood mononuclear cells after long-term strength training in previously untrained men [30].

CK and LDH are fragments of the myosin heavy chain and are related to muscle damage. These molecules are cytoplasmatic and do not have the capacity to cross the sarcoplasmic membrane barrier. For this reason, increased serum levels of these molecules are used as an indicator of damage to muscle membrane and other tissue structures [31]. In this respect, our findings revealed that hypertrophy-, strength-, and power-type resistance exercises induced a significant increase in serum CK and a non-significant increase in serum LDH levels in resistance-trained women. Besides, the power-type resistance exercise resulted in a remarkable increase in serum CK compared to the other two types. In reality, the CK activity observed in this study showed that muscle tissue damage occurred well after all resistance exercise protocols. Consistent with the findings of the present study, it is shown that both bi-set and multiple sets of resistance exercise (70–80% of 1RM) result in increased serum CK level, while no significant change has been observed in LDH levels in recreationally trained men [31]. In addition, a remarkable increase in CK and LDH levels has been noted in response to high-intensity resistance exercise in trained and untrained men [15] and sedentary women [29]. Moreover, it has been revealed that both low-intensity (3 sets of 20–30 repetitions at 20–35% of 1RM) and high-intensity (3 sets of 2–8 repetitions at 80–95% 1RM) resistance programs result in an equal change in CK plasma levels in sedentary males [23]. It seems that the observed higher level of CK following power-type resistance exercise than strength- and hypertrophy-type resistance exercises in the current study is a function of the intensity of the resistance program.

Intriguingly, it has been reported that both high-intensity and high-volume resistance exercises enhance serum levels of CK and LDH 30 min and 24 h after exercise in participants with 6.3 ± 3.4 years of resistance training experience [16]. Gonzalez et al. have reported that an acute bout of lower-body resistance exercise protocol induces an increase in plasma LDH in subjects with 6.7 ± 4.6 years of resistance training experience [32]. Hence, the experience of resistance exercise training may not prevent muscle damage and CK and LDH release into the bloodstream. Alongside this, Pareja-Blanco and colleagues reported such an increase in CK levels in trained men with 2–4 years of exercise experience [6].

Although it has been suggested that high-volume resistance exercise causes more considerable muscle damage than high-intensity resistance [6, 16], the results of our study showed no significant difference concerning serum levels of CK and LDH between hypertrophy- and strength-type resistance exercises conducted, respectively, at high volume and high intensity. Interestingly, the power-type resistance exercise that was performed for 8–10 repetitions resulted in higher levels of CK than hypertrophy- and strength-type resistance exercises. It has been reported that fast-velocity lengthening contractions result in more CK levels than slow-velocity lengthening contractions in active [33] and sedentary men [34]. Therefore, it appears that the higher CK level in response to power-type resistance exercise than the other two types is due to the explosive speed.

Finally, micronutrients play a substantial role in regulating enzymes that moderate oxidative damage and muscle damage. Non-assessment of micronutrients can be one of the limitations of the present study, which is suggested to be assessed in future studies.

Overall, the results of the current study showed that none of the resistance exercise types exacerbate oxidative stress in resistance-trained women. Conversely, in relation to muscle damage, it was shown that hypertrophy-, strength-, and power-type resistance exercises increase the

CK level as a primary muscle damage marker. In particular, the power-type resistance exercise, which was performed at an explosive speed, resulted in a more significant increase in muscle damage. Therefore, women who undergo resistance exercises can alleviate the occurrence of muscle damage induced by resistance exercise by minimizing the speed of contraction.

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Author contributions All authors conceived the study, its design, and coordination. SM, HT, and AG were involved in the data collection, data analysis, and drafting of the manuscript. Finally, all authors read and approved the final version of the manuscript and agreed with the order of the presentation of the authors.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interests.

Ethics approval All procedures performed in current study involving human participants were in accordance with ethicalstandards of the institutional research committee and with the 1975 Helsinki Declaration and its later amendments or comparable ethical standards. The study was approved by ethical committee of Bojnord Univertsity (No. 3925).

Informed consent In addition, all participants signed written informed consent form that was approved by ethical committee.

References

1. Powers SK, Jackson MJ (2008) Exercise-induced oxidative stress: cellular mechanisms and impact on muscle force production. Physiol Rev 88(4):1243–1276
2. Jackson MJ, Vasilaki A, McArdle A (2016) Cellular mechanisms underlying oxidative stress in human exercise. Free Radic Biol Med 98:13–17
3. Andersson KE (2018) Oxidative stress and its possible relation to lower urinary tract functional pathology. BJU Int 121(4):527–533
4. Maughan RJ, Donnelly AE, Gleeson M, Whiting PH, Walker KA, Clough PJ (1989) Delayed-onset muscle damage and lipid peroxidation in man after a downhill run. Muscle Nerve 12(4):332–336
5. Urso ML, Clarkson PM (2003) Oxidative stress, exercise, and antioxidant supplementation. Toxicology 189(1–2):41–54
6. Pareja-Blanco F, Rodríguez-Rosell D, Sánchez-Medina L, Ribas-Serna J, López-López C, Mora-Custodio R, Yáñez-García JM, González-Badillo JJ (2017) Acute and delayed response to resistance exercise leading or not leading to muscle failure. Clin Physiol Funct Imaging 37(6):630–639
7. Gadruni K, Mahmmadpour H, Gadruni M (2015) Effect of elastic-band exercise on muscle damage and inflammatory responses in Taekwondo athletes. Rev Bras Med Esporte 21(4):297–301
8. Koch AJ, Pereira R, Machado M (2014) The creatine kinase response to resistance exercise. J Musculoskelet Neuronal Interact 14(1):68–77
9. Spanidis Y, Stagos D, Papanikolaou C, Karatza K, Theodosi A, Veskoukis AS, Deli CK, Poulios A, Koulocheri SD, Jamurtas AZ, Haroutounian SA (2018) Resistance-trained individuals are less susceptible to oxidative damage after eccentric exercise. Oxid Med Cell Longev. https://doi.org/10.1155/2018/6857190
10. Ammar A, Chtourou H, Hammouda O, Trabelsi K, Chiboub J, Turki M, AbdelKarim O, El Abed K, Ben Ali M, Hoekelmann A, Souissi N (2011) Acute and delayed responses of C-reactive protein, malondialdehyde and antioxidant markers after resistance training session in elite weightlifters: effect of time of day. Chronobiol Int 32(9):1211–1222
11. Sepehri MH, Masoud Nikbakht M, Habibi A, Moradi M (2014) Effects of a single low-intensity resistance exercise session on lipid peroxidation of untrained male students. Am J Sports Sci 2:87–91
12. Çakir-Atabek H, Demir S, PinarbaSili RD, Gündüz N (2010) Effects of different resistance training intensity on indices of oxidative stress. J Strength Cond Res 24(9):2491–2497
13. Levers K, Dalton R, Galvan E, Goodenough C, O'Connor A, Simbo S, Barringer N, Mertens-Talcott SU, Rasmussen C, Greenwood M, Riechman S (2015) Effects of powdered Montmorency tart cherry supplementation on an acute bout of intense lower body strength exercise in resistance trained males. J Int Soc Sports Nutr 12(1):41
14. Barquilha G, Silvestre JC, Motoyama YL, Azevedo PH (2018) Single resistance training session leads to muscle damage without isometric strength decrease. J Hum Sport Exerc 13(2):267–275
15. Ashtary-Larky D, Lamuchi-Deli N, Milajerdi A, Salehi MB, Alipour M, Kooti W, Ashtary-Larky P, Alamiri F, Sheikhi A, Afrisham R (2017) Inflammatory and biochemical biomarkers in response to high intensity resistance training in trained and untrained men. Asian J Sports Med 8(2):e13739
16. Bartolomei S, Sadres E, Church DD, Arroyo E, Gordon JA III, Varanoske AN, Wang R, Beyer KS, Oliveira LP, Stout JR, Hoffman JR (2017) Comparison of the recovery response from high-intensity and high-volume resistance exercise in trained men. Eur J Appl Physiol 117(7):1287–1298
17. Kraemer WJ, Ratamess NA (2004) Fundamentals of resistance training: progression and exercise prescription. Med Sci Sports Exerc 36(4):674–688
18. Bird SP, Tarpenning KM, Marino FE (2005) Designing resistance training programmers to enhance muscular fitness. Sports Med 35(10):841–851
19. Ahmadi MA, Zar A, Krustrup P, Ahmadi F (2018) Testosterone and cortisol response to acute intermittent and continuous aerobic exercise in sedentary men. Sport Sci Health 14(1):53–60
20. Machado M, Koch AJ, Willardson JM, Pereira LS, Cardoso MI, Motta MK, Pereira R, Monteiro AN (2011) Effect of varying rest intervals between sets of assistance exercises on creatine kinase and lactate dehydrogenase responses. J Strength Cond Res 25(5):1339–1345
21. Jones TW, Howatson G, Russell M, French DN (2016) Performance and endocrine responses to differing ratios of concurrent strength and endurance training. J Strength Cond Res 30(3):693–702
22. Tsoukos A, Veligekas P, Brown LE, Terzis G, Bogdanis GC (2018) Delayed effects of a low-volume, power-type resistance exercise session on explosive performance. J Strength Cond Res 32(3):643–650
23. Güzel NA, Hazar S, Erbas D (2007) Effects of different resistance exercise protocols on nitric oxide, lipid peroxidation and creatine kinase activity in sedentary males. J Sports Sci Med 6(4):417–422

24. Wang P, Li CG, Qi Z, Cui D, Ding S (2015) Acute exercise induced mitochondrial H_2O_2 production in mouse skeletal muscle: association with p66Shc and FOXO3a signaling and antioxidant enzymes. Oxid Med Cell Longev
25. Zembron-Lacny A, Ostapiuk J, Slowinska-Lisowska M, Witkowski K, Szyszka K (2008) Pro-antioxidant ratio in healthy men exposed to muscle-damaging resistance exercise. J Physiol Biochem 64(1):27–35
26. Dixon CB, Robertson RJ, Goss FL, Timmer JM (2006) The effect of acute resistance exercise on serum malondialdehyde in resistance-trained and untrained collegiate men. J Strength Cond Res 20(3):693
27. Park SY, Kwak YS (2016) Impact of aerobic and anaerobic exercise training on oxidative stress and antioxidant defense in athletes. J Exerc Rehabil 12(2):113
28. Ramel A, Wagner KH, Elmadfa I (2004) Plasma antioxidants and lipid oxidation after submaximal resistance exercise in men. Eur J Nutr 43(1):2–6
29. Hosseinzadeh M, TaheriChadorneshin H, Ajam-Zibad M, Abtahi-Eivary SH (2017) Pre-supplementation of *Crocus sativus* Linn (saffron) attenuates inflammatory and lipid peroxidation markers induced by intensive exercise in sedentary women. J Appl Pharm Sci 7(05):147–151
30. García-López D, Häkkinen K, Cuevas MJ, Lima E, Kauhanen A, Mattila M, Sillanpää E, Ahtiainen JP, Karavirta L, Almar M, González-Gallego J (2007) Effects of strength and endurance training on antioxidant enzyme gene expression and activity in middle-aged men. Scand J Med Sci Sports 17(5):595–604
31. Callegari GA, Novaes JS, Neto GR, Dias I, Garrido ND, Dani C (2017) Creatine kinase and lactate dehydrogenase responses after different resistance and aerobic exercise protocols. J Hum Kinet 58(1):65–72
32. Gonzalez AM, Hoffman JR, Townsend JR, Jajtner AR, Boone CH, Beyer KS, Baker KM, Wells AJ, Mangine GT, Robinson EH IV, Church DD (2015) Intramuscular anabolic signaling and endocrine response following high volume and high intensity resistance exercise protocols in trained men. Physiol Rep 3(7):e12466
33. Chapman DW, Newton MJ, McGuigan MR, Nosaka K (2011) Effect of slow-velocity lengthening contractions on muscle damage induced by fast-velocity lengthening contractions. J Strength Cond Res 25(1):211–219
34. Chapman DW, Newton M, Mcguigan M, Nosaka K (2008) Effect of lengthening contraction velocity on muscle damage of the elbow flexors. Med Sci Sports Exerc 40(5):926–933