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ORIGINAL RESEARCH

The Effect of Progressive Resistance Training on Oxidative Stress and Antioxidant Enzyme Activity in Erythrocytes in Untrained Men

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This study was undertaken to investigate the effects of progressive resistance-training (PRT) on plasma oxidative stress and antioxidant enzyme activity in erythrocytes. Twenty male volunteers were randomly assigned to 2 groups: PRT and control. Blood samples were collected before and after 8 wk of PRT and analyzed for enzymatic activities of superoxide dismutase (SOD) and glutathione peroxidase (GPx) in erythrocytes, plasma total antioxidant capacity (TAC), and malondialdehyde concentration (MDA, an index of lipid per oxidation in plasma). Resistance training commenced with 8 exercises on nonconsecutive days for 8 wk at 50% of estimated 1-repetition maximum (E1RM) and reached 80% E1RM by Week 8. The results showed that PRT significantly increased erythrocyte SOD activity (1,323 ± 212.52 vs. 1,449.9 ± 173.8 U/g Hb, $p = .014$). Plasma concentration of MDA also decreased (5.39 ± 1.7 vs. 3.67.4 ± 0.7 nmol/ml, $p = .030$), although TAC (1.42 ± 0.21 vs. 1.61 ± 0.19 mmol/L, $p = .1530$) and GPx (39.87 ± 11.5 vs. 48.18 ± 14.48 U/g Hb, $p = .883$) activity did not undergo any considerable changes. Based on these data, the authors conclude that an 8-wk program of PRT strengthens the defensive system of erythrocytes against free-radical damage and therefore can be applied as a useful approach to alleviate oxidative stress.

Keywords: superoxide dismutase, glutathione peroxidase, lipid peroxidation, weight training

An increase in macromolecule oxidation has been demonstrated after both aerobic and anaerobic exercise of sufficient intensity (Bloomer et al., 2006). The generation of reactive oxygen and nitrogen species (RONS) such as singlet oxygen, superoxide radical, hydroxyl radical, and peroxynitrite occurs as a consequence of normal cellular metabolism and is increased under conditions of physical stress such as prolonged exercise (Sen, Packer, & Hanninen, 1994). In anaerobic exercise, however, other pathways of RONS generation exist (Bloomer & Goldfarb, 2004) including ischemia reperfusion, xanthine and nicotinamide adenine dinucleotide phosphate oxidase production, prostanoid metabolism, phagocytic respiratory-burst activity, disruption of iron-containing proteins, and altered calcium homeostasis (Bloomer et al., 2006). The production of RONS via these pathways may result partly from eccentric muscle action, which causes muscle microtrauma and subsequent inflammation (McHugh, Connolly, Eston, & Glein, 1999).

There is some evidence that resistance exercise can also induce oxidative stress and generation of free radicals akin to those observed during prolonged aerobic and anaerobic exercise (Goldfarb et al., 2008; Hudson et al., 2008; Ramel, Wagner, & Elmadfa, 2004). For example, erythrocytes are subject to mechanical and oxidative stress during strenuous contractile work (Moore, Gioioso, Sills, & Mendelson, 1999); it has been shown that reactive oxygen species are released into the circulation by immune, endothelial, and muscle cells (Radak, Chung, & Goto, 2008; Valko et al., 2007) and impair normal erythrocyte function through lipid-membrane peroxidation.

There are various defense mechanisms, endogenous and exogenous (dietary; Clarkson & Thompson, 2000), the former of which are up-regulated after exercise (Berzosa et al., 2011; Gomez-Cabrera, Domenech, & Viña, 2008). However, antioxidant- and lipid-peroxidation-related adaptations after resistance training have been poorly investigated, and studies on the effect of resistance exercise on oxidative stress report contradictory findings (Hoffman et al., 2007; McBride, Kraemer, Triplett-McBride, & Sebastianelli, 1998; Ramel et al., 2004). Furthermore, the effect of resistance training on oxidative stress in erythrocytes and antioxidant capacity has not yielded consistent results (Liu et al., 2005; Parise, Brose, & Tarnopolsky, 2005; Vincent, Bourguignon, & Vincent, 2006). This could be due to either differences in prescribed training intensity (Liu et al., 2005; Parise, Phillips, Kaczor, & Tarnopolsky, 2005) or lack of a control group (Cakir-Atabek, Demir, Pinarbasili, & Gunduz, 2010). Proper examination of this issue would require a

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progressively well-controlled training protocol to ensure ability to compare with a control condition.

Therefore, the current study was carried out with the aim of investigating the effect of progressive resistance training on erythrocytes' antioxidant enzyme activity and oxidative stress in untrained men. The information gathered from this study will help determine the suitability of prescribing resistance training where a specific aim is to improve oxidative defense.

Methodology

Subjects

Twenty untrained men with no experience of resistance training or regular physical activity volunteered to participate in the study and were assigned to one of two homogeneous groups: Progressive resistance training (PRT, $n = 10$) and control ($n = 10$).

The subjects followed a generally sedentary lifestyle and had not practiced formal physical exercise for more than 1 hr/week during the preceding 3 months . Subjects recorded all their physical activities in 15-min intervals in activity diaries over a 4-day period before initiation of the study (Ainsworth et al., 2000). In addition, the groups were matched according to age, body mass, height, percentage body fat, body-mass index, and estimated 1-repetition-maximum (E1RM) values (Table 1). They were informed of the purposes and methods of the study before they provided written consent. None of the participants were taking any form of medication, nor did they have alcohol, smoking, or vitamin supplement habits. The subjects were warned against taking nonsteroidal anti-inflammatory drugs including aspirin and naproxen during the research project. The study design and experimental procedures were approved by the Regional Research Ethics Committee of Islamic Azad University, Central Tehran Branch.

Physiological Measurements

Subjects' height and weight (Seca, Mod 220, Germany) were measured, and their percentage body fat was estimated through measuring skinfold thickness (Lafayette, Mod 01127, USA). Skinfold thickness was measured at three sites—abdomen, suprailiac, and triceps—and percentage body fat was estimated using the equation of Jackson and Pollock (1985).

PRT Protocol

PRT subjects underwent a whole day of weight training and safety precautions after the collection of pretraining blood samples.

Session 1 included a general warm-up consisting of 3–5 min of low-intensity running, 5–10 min of stretching exercises, and four warm-up sets leading up to the final set to determine predicted maximum strength (see Table 2). This procedure was repeated for both upper body (UB) and lower body (LB) exercises. For both UB and LB exercises, the initial loads used during the warm-up sets were based on percentages of the participants' self-reported estimate of their 1-RM. The warm-up sets leading to the final maximum-repetition attempt had a dual purpose of warming up the participant for the attempt and familiarizing him with the metronome-established cadence for the repetitions.

After the fifth set, additional sets were added if the load for the fifth allowed for more than three repetitions. The pattern of added sets was continued until a 3-RM was achieved. Thus a ≤3-RM load (85% self-reported estimate of 1-RM) for the final set was used as the upper bound (Arnold, Mayhew, LeSuer, & McCormick, 1995).

Only repetitions performed to the cadence of metronome were counted during the last set. The participant was warned once to keep up with the cadence during the maximum-strength-testing set. If the participant could not keep up with the metronome cadence, the set was ended and the repetition that the participant was able to complete on cadence was counted as the final repetition. The completed repetitions during the last set were used to predict the 1-RM for UB and LB exercises using the equation from Brzycki (1993) both in the PRT and the control group.

Table 1 Physical Characteristics of Subjects at the Start of the Study, *M* ± *SD*

	Resistance-training group	Control group	*t*	*p*
Age (years)	21.2 ± 2.1	23.3 ± 2.5	–1.45	.157
Body mass (kg)	72.61 ± 3.86	71.37 ± 3.27	0.660	.517
Height (cm)	173 ± 3.67	176 ± 6.68	–1.25	.225
Body fat (%)	19.51 ± 6.12	18.54 ± 5.52	0.805	.431
Body-mass index (kg/m^2)	24.28 ± 2.17	23.09 ± 1.45	1.47	.158
E1RM chest press (kg)	33.5 ± 6.2	31.47 ± 7.2	0.943	.358

Note. E1RM = estimated 1-repetition maximum.

Table 2 Sets and Repetitions for Session 1, Familiarization and Maximum-Strength Protocol

Set	Number of repetitions
1: 50% of self-reported maximum	10
2: 50–60% of self-reported maximum	8–10
3: 70% of self-reported maximum	8
4: 80% of self-reported maximum	3
5: ≥85% of self-reported maximum	Maximum repetition but fewer than 3

All these procedures were repeated after the end of the fourth and eighth weeks. The accuracy of Brzycki's (1993) prediction equations for estimating 1-RM performance has been demonstrated by Lesuer, McCormick, Mayhew, Wasserstein, and Arnold (1997).

Resistance training was performed with progressive load three times per week on nonconsecutive days for 8 weeks, with circuit training. The movements involved were UB and LB exercises: chest press, lat pull-down, leg extension and flexion, biceps and triceps curl, squat, and sit-ups for abdominal muscles (with 1- to 2- and 3- to 5-min intervals between sets and cycles, respectively). The training started with 50% of E1RM at Week 1 with 8–12 repetitions, three sets in each exercise. The load for each exercise increased by approximately 5% each week such that the intensity of training reached 80% of E1RM by the end of the eighth week. For the fourth and fifth weeks, the intensity of training was identical. A 10-min light warm-up and cooldown using aerobic exercise was included at the beginning and after the end.

Subjects' E1RM was measured again at the end of the fourth week, and the prescribed loads were adjusted accordingly.

Blood Collection and Erythrocyte Isolation

After a 10-hr fast, between 8 and 10 a.m. and before the determination of E1RM, a 10-ml blood sample was obtained from an antecubital vein. The same procedure took place 72 hr after the completion of the training program (at the end of the eighth session). Immediately after blood collection, 150 µl of ethylenediamine tetra acetic acid (Merck, Germany) was added to the blood samples, and the mixture was centrifuged at 2,500–2,700 rpm for 7 min. Then, plasma and the surface layer were separated from erythrocytes. Plasma samples were used to measure total antioxidant capacity and MDA. In addition, superoxide dismutase activity (SOD) and glutathione peroxidase (GPx) were assayed in erythrocytes. For this purpose, erythrocytes were washed three times with 0.9% NaCl solution (normal saline) at 4 °C. Distilled water was added to the erythrocytes to obtain ~50% dilution. For complete erythrocyte lysis, samples were stored at room temperature for 5–10 min. The mixture was then centrifuged for 10 min at 4 °C. Then, the clear supernatant solution was divided into equal volumes of 0.5 ml and stored in Eppendorf tubes at –70 °C for analysis of SOD and GPx.

Biochemical Analyses

Erythrocyte SOD was measured using a commercially available kit (Ransod, Cat. No. SD 125, Randox, UK). In this method, xanthine and xanthine oxidase are employed to generate superoxide radicals that react with 2-(4-iodophenyl)-3-(4-nitrophenol)-5-phenyltetrazolium chloride to form a red formazan dye whose absorbance is measured at 505 nm (Mc Cord & Fridovich, 1969). The activity of erythrocyte GPx was measured using a commercially available kit (Ransel, Cat. No. RD 505, Randox UK) that employs the method of Paglia and Valentine (1967). SOD and GPx activities were expressed relative to the hemoglobin concentration. Similarly, a commercially available kit (Randox, Cat No, NX 2332, UK) was used to measure antioxidant capacity in the plasma (Miller, Rice-Evans, Davies, Gopinathan, & Miller, 1993). Plasma MDA was measured based on the method of Buege and Aust (1978).

Nutrition

All subjects completed a detailed daily food diary in which they recorded all food and drink consumed for the 3 consecutive days before beginning the training protocol. At the end of the training period, subjects again recorded food intake for 3 consecutive days (before blood sampling).

Subjects were also provided with necessary information concerning recording the amounts of food consumed and the correct method of recording. Diet was analyzed using Food Processor software (Esha Research, Salem, OR, USA) with regard to antioxidant and macronutrient content.

Statistical Analyses

All data are presented as $M \pm SD$. First, the Kolmogorov-Smirnov test was used to determine the normality of data distribution. Dependent variables were compared using two-way repeated-measures (pre- and posttraining) ANOVA to investigate the influence of time and the

training intervention (PRT vs. control). A $p < .05$ level of significance was used. A significant interaction between group and time indicates a significant effect of training on the dependent variable of interest. All statistical analyses were carried out using SPSS version 16.0.

Results

Before training, the daily energy intake of the PRT group consisted of 51% ± 3%, 31% ± 2%, and 18% ± 3%, respectively, for contributions from carbohydrate, fat, and protein, proportions not significantly different from those of the control group (48% ± 3%, 33% ± 2%, 19% ± 3%). Although individual and total macronutrient intake increased during training, there remained no significant difference between groups. Nutrient analysis of the dietary records of the PRT and control groups before and after the training period using independent *t* test are presented in Table 3.

There was no significant difference between the PRT and control groups regarding percentage body fat. There was a significant interaction between group and time ($p = .014$) for SOD activity but no main effect for time, showing that SOD activity increased as a function of the PRT program and not simply time between tests (Figure 1). Similarly, MDA was significantly decreased with PRT ($p = .030$), but there was also a tendency for a main (time only) effect ($p = .058$; Figure 1). Neither total antioxidant capacity nor GPx changed during the course of the study (Figure 1).

With PRT, estimated muscle strength increased by 31.2% ($p = .001$) and by 43.4% ($p = .001$) in chest press and squat, respectively. No significant change was found in the control group. Percentage body fat was also not

Table 3 Nutrient Analysis of the Dietary Records of the Resistance and Control Groups Before and After the Training Period, *M* ± *SD*

	Resistance-training group	Control group	*t*	*p*
Protein (g/day) before training	88.60 ± 20.56	101.80 ± 15.27	–1.620	.181
after training	114.0 ± 12.2	108.0 ± 20.2	0.780	.621
Carbohydrates (g/day) before training	259.7 ± 45.0	287.0 ± 83.8	0.919	.370
after training	280.1 ± 35.3	271.3 ± 41.1	0.711	.614
Fat (g/day) before training	70.10 ± 13.31	83.10 ± 21.20	1.640	.118
after training	87.21 ± 12.40	79.80 ± 7.70	1.130	.272
Vitamin C (mg/day) before training	61.20 ± 21.03	53.00 ± 19.04	0.914	.373
after training	70.12 ± 12.80	58.30 ± 14.20	2.027	.058
α-tocopherol (mg/day) before training	5.30 ± 2.11	4.40 ± 2.06	0.946	.348
after training	6.05 ± 2.02	4.80 ± 1.90	1.670	.167
Vitamin A (µg/day) before training	663.50 ± 191.18	522.20 ± 174.35	1.720	.101
after training	698.70 ± 121.50	542.98 ± 45.90	1.021	.126
β-carotene (µg/day) before training	522.9 ± 1,306.0	1,050.0 ± 655.9	1.010	.328
after training	571.3 ± 987.2	879.6 ± 457.3	1.210	.066
Manganese (mg/day) before training	2.5 ± 6.3	2.4 ± 7.7	0.446	.661
after training	2.3 ± 2.7	2.6 ± 4.4	0.514	.458
Iron (mg/day) before training	15.00 ± 2.30	16.90 ± 4.22	1.240	.230
after training	16.90 ± 1.52	15.60 ± 0.80	1.350	.120
Copper (mg/day) before training	1.42 ± 0.68	1.65 ± 0.66	0.742	.468
after training	1.55 ± 0.21	1.32 ± 0.11	0.821	.123
Zinc (mg/day) before training	29.60 ± 15.63	27.80 ± 10.76	0.300	.768
after training	33.60 ± 10.10	22.30 ± 9.70	1.910	.071
Selenium (mg/day) before training	69.70 ± 18.23	81.70 ± 28.01	1.130	.271
after training	78.90 ± 16.03	82.30 ± 15.30	0.222	.827

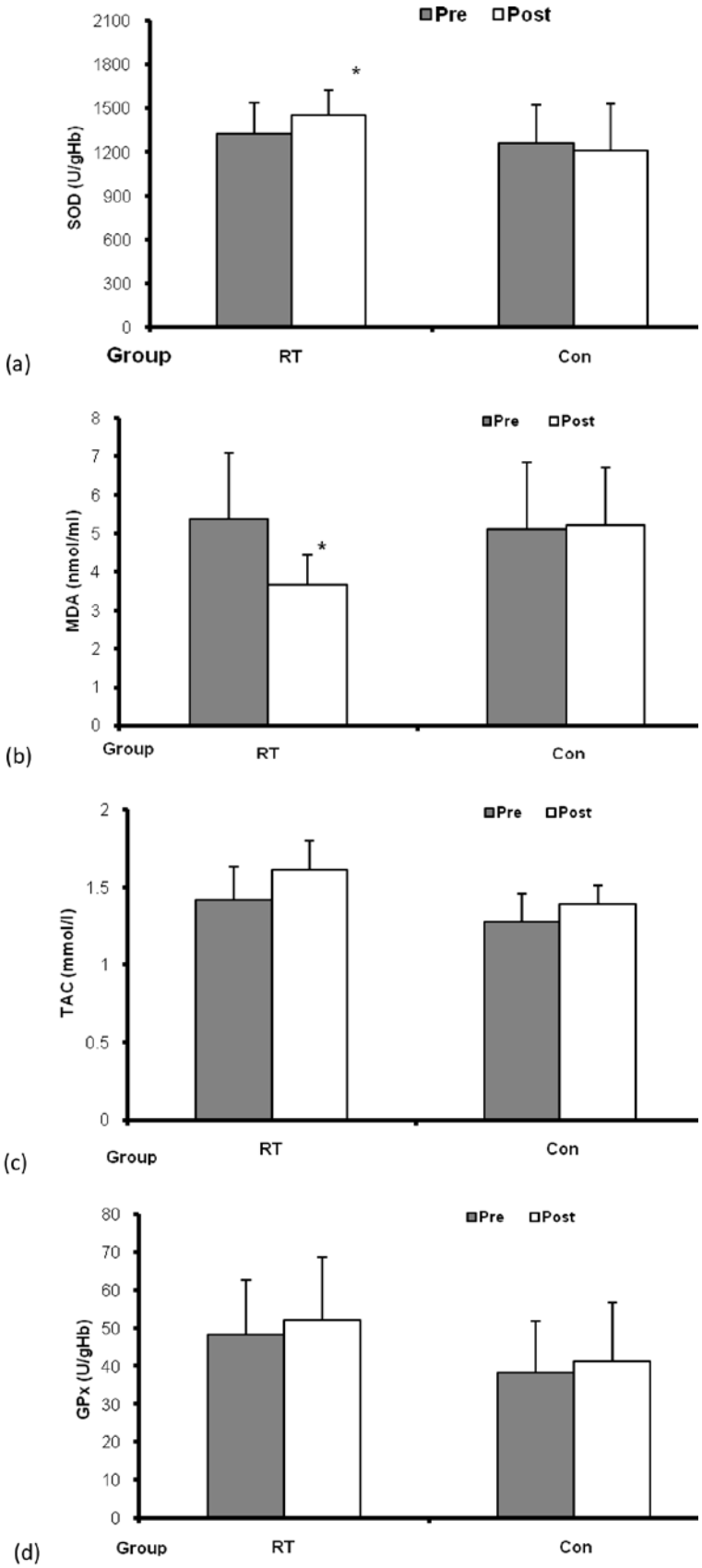

Figure 1 — Pre- and posttraining values of (a) superoxide dismutase, (b) malondialdehyde, (c) plasma total antioxidant capacity, and (d) glutathione peroxidase between the two groups over 8 weeks of resistance training, *M* and *SD*. RT = resistance training; Con = control. *Significant difference from pretraining.

changed with PRT, and no significant (4.2%) increase in body mass was seen.

Discussion

This study was designed to investigate whether resistance training would produce an increase in antioxidant capacity and a decrease in markers of lipid peroxidation of the blood. Subjects were exposed to PRT for 8 weeks. These data provide evidence that PRT for 8 weeks attenuates oxidative stress in young adult men while simultaneously enhancing antioxidant defense at rest. The results also clearly show that 8 weeks of PRT led to a significant increase in estimated maximal strength in chest press and squat, which was indicative of the functional efficacy of the training. However, resistance training with the used protocol was not able to produce a significant change in body composition.

Comparison of pretest values showed that the activities of SOD and GPx, plasma antioxidant capacity, and MDA were not different between the PRT and control groups initially, which implies homogeneity of groups before the intervention. However, after 8 weeks of PRT there were demonstrable changes in SOD and MDA that were absent in the control group. This conclusively shows that PRT produces an increase in antioxidant defense in the circulation and, subsequently, an adaptive reduction in oxidative-stress levels.

It has been proposed that despite the lower absolute energy demands of resistance training than with endurance training, there is still generation of free radicals. The possible pathways for the production of these reactive species include the xanthine oxidase pathway, respiratory burst of neutrophils, catecholamine autooxidation, local muscle ischemia, and conversion of the weak superoxide anions to strong hydroxyl radical through lactic acid (Inal, Akyuz, Turgut, & Getsfrid, 2001; Ji, Stratman, & Lardy, 1988; Smith & Miles, 2000). However, it seems unlikely that all these mechanisms are involved in generating oxidative stress. For example, activation of neutrophils usually takes place after muscle damage elicited by oxidative stress, mechanical stress, and/or muscle disruption (Meydani & Evans, 1993). Unfortunately, we are unable to ascertain the exact mechanism behind our observations, so any attempt to provide a mechanistic explanation would be purely speculative.

Previously published studies on the effect of resistance training on SOD and GPx activity have reported contrasting findings (Liu et al., 2005; Parise, Brose, & Tarnopolsky, 2005; Parise, Phillips, et al., 2005; Vincent, Vincent, Braith, Lennon, & Lowenthal, 2002). The current study did not detect a significant difference in GPx with training. In contrast, Liu et al. reported a decrease in GPx activity and a rise in oxidative stress by 1 week of acute resistance training.

This dissonance may be explained by the difference in intervention duration and related ability to adapt, which may affect oxidative stress (Hudson et al., 2008).

Catalase and GPx have a role similar to that of H_2O_2 because hydrogen peroxide transmutates to water, but they are different in H_2O_2 concentration; glutathione peroxidase needs high H_2O_2 concentration and catalase needs low H_2O_2 concentration (Antunes, Han, & Cadenas, 2002). Probably during the resistance-training sessions in the current study H_2O_2 production was not high enough to stimulate the glutathione peroxidase enzyme adaptation to erythrocyte, and it seems that in this exercise program most neutralization is done by catalase enzyme. In other words, this even is due to lack of adaptive effect of the mentioned enzyme and less need of the GPx path in neutralizing free radicals that were made during resistance training in this study. Conversely, it has been reported that mitochondrial GPx is more adaptive to training in skeletal muscles than cytosol GPx (like red cells; Ji et al., 1988).

Although erythrocyte GPx activity did not significantly increase, we do not know what changes might have occurred in other tissues. A GPx isoenzyme in other tissues may have influenced lipid peroxidation, even markers in the plasma.

In the current study, erythrocyte SOD showed a small (~10%) but significant increase with PRT. As the main defense against superoxide radical and oxidation stress, a rise in SOD causes superoxide radical to form H_2O_2. It is not clear whether there has been a rise in protein content, although an increase in the activities of the aforementioned enzymes aimed at neutralizing superoxide radical is indicative of posttranslational adaptation (Oh-ishi et al., 1997). In this regard, Parise, Phillips, et al. (2005) reported that 12 weeks of resistance training could upregulate antioxidant enzymes in muscles of older men and has an effect on muscle-fiber antioxidant capacity in such a way that a significant increase in Cu,Zn SOD and catalase enzyme activity was also observed. However, the same training barely had any effect on Mn SOD activity (Parise, Phillips, et al., 2005). The same researchers, on the other hand, also reported that for older men Cu,Mn SOD concentration level did not increase with a 14-week resistance-training program (Parise, Brose, & Tarnopolsky, 2005). The differing results could be a function of subjects' age difference and thus responses to resistance training. Antioxidant enzyme activity in our study was based on red blood cells, so comparison is difficult (Bloomer, Fry, Falvo, & Moore, 2007).

Although significant, the increase in red blood cell SOD was small, and it seems unlikely that the observed decrease in plasma MDA levels could be attributed to this. Indeed, others (de Gonzalo-Calvo et al., 2013) have shown similar differences between the trained and untrained state in erythrocyte SOD activity, but with increased markers of lipid peroxidation.

In addition, the current study's results showed that in the PRT group plasma antioxidant capacity increased insignificantly 14%. Plasma antioxidant-capacity evaluation is one of the most common methods to evaluate the humoral antioxidant state in body fluids (Repetto et al., 1996). In this method, the sample capacity is evaluated

by neutralizing and trapping free radicals (Lissi, Salim-Hanna, Pascual, & Del Castillo, 1995).

In the current study, plasma antioxidant capacity was evaluated by Miller et al.'s (1993) method. In this method, all the materials in plasma that have antioxidant property are examined, so the humoral antioxidants such as ascorbic acid, GSH, uric acid, and bilirubin are examined completely. The problem with this method is that plasma antioxidant capacity depends on changes in plasma uric acid (Kusano & Ferrari, 2008).

It is not clear that how much of the elevation in plasma antioxidant capacity is related to uric acid elevation and how much is related to elevation of other antioxidants. Many researchers have investigated plasma antioxidants individually. For example, Cakir-Atabek et al. (2010) used glutathione concentration, Liu et al. (2005) used vitamin E, and Ramel et al. (2004) used tocopherol, beta-carotene, lycopene, ascorbic acid alpha-tocopherol, and gamma-tocopherol.

It is impossible to compare the research results because of measuring different variables and different methods of measuring plasma humoral antioxidants.

The results of the current study showed that PRT is associated with decreases in plasma MDA concentrations by approximately 12%. Cakir-Atabek et al. (2010) similarly report that 6 weeks of resistance training (three sets with 12 repetitions, 70% intensity of high repetitions, and 90-s rest and three sets with six repetitions, 85% 1RM, and 180-s rest) is associated with reduced plasma MDA. On the other hand, Liu et al. (2005) report that 1 week of intense resistance training increases oxidative stress and cell damage in female weight lifters in line with a reduction in plasma antioxidant capacity on the TBARs level and in MDA^{+} 4-hydroxy 2-(E)-nonenal. Clearly, timing of blood sampling is important in relation to the last training session; sampling too close to the final training session may reflect the acute effects of exercise on oxidative damage rather than the adaptive (training) effects. The difference in the relationship between erythrocyte antioxidant activity and markers of damage between the current and other recent research (de Gonzalo-Calvo et al., 2013) may reflect the long time (72 hr) between the last training session and the blood sampling.

Our study recruited subjects of an age range of 18–26 years. Some reports suggest a more significant increase in antioxidant capacity and, consequently, a more dramatic decrease in oxidative stress in young men than in older subjects (Dixon et al., 2006). The applicability of the current data therefore is limited to a younger cohort, and this makes comparison more difficult. Nevertheless, we can confirm that improved antioxidant capacity is a benefit to partaking in a program of resistance training that of the usual functional, anthropometrical, and metabolic benefits reported. These occur in a relatively short period of time (8 weeks), even before changes in body composition are detectable.

Finally, we have studied only the activities of antioxidant enzymes in erythrocytes and MDA in plasma. A better understanding of the effect of resistance training (in particular) on antioxidant defense would be gained by measuring the activities in skeletal-muscle tissue since this is the principle site of energetic flux. Furthermore, to better understand the relation between erythrocyte antioxidant enzymes and oxidative stress, measuring MDA in the red cells rather than the plasma would be preferential.

Conclusion

Eight weeks of PRT can improve antioxidant defense of erythrocytes and reduce lipid peroxidation in these cells. This suggests that resistance training, alongside nutritional interventions, is effective in supporting the antioxidant defensive system and thereby reducing oxidative damage. Additional research appears to be warranted to further examine the effect of gender, age, and training status to examine how a longer period of training can affect such changes. In addition, those doing further research may consider using more biomarkers of oxidative stress and more antioxidant enzymes.

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