Available online at www.scholarsresearchlibrary.com

Scholars Research Library

Scholars Research Library

Annals of Biological Research, 2012, 3 (12):5490-5493
(http://scholarsresearchlibrary.com/archive.html)

Annals of Biological Research
Scholars Research Library
ISSN 0976-1233
CODEN (USA): ABRNBW

Oxidative DNA damage responses to an acute session of hypertrophy- and strength-intensity resistance exercise

[1]Ali Khorjahani, [2] Rahman Rahimi, [3]Vahid Moghadam

[1]Department of Physical Education and Sport Science, Takestan Branch, Islamic Azad University, Takestan, Iran
[2]Department of Physical Education and Sport Science, University of Kurdistan, Sanandaj, Iran
Department of Physical Education and Sport Science, Qazvin Islamic Azad University, Qazvin, Iran

---

ABSTRACT

The purpose of the present study was to compare oxidative modification of DNA in the 24 hours following hypertrophy- and strength-intensity resistance exercise (RE) protocols. A week after one repetition maximum (1RM) test, 10 resistance-trained men performed strength-intensity RE (4 sets of 5 exercises to failure at 90% of the 1RM, with 3 min rest) and hypertrophy-intensity RE (4 sets of 5 exercises to failure at 75% of the 1RM, with 90 second rest) in a randomized and crossover design. Urine samples were collected before (Pre), after (Post), 3h after (3h Post) and 24 h after RE (24h Post) for analyzing oxidative DNA damage as measured by urinary 8-hydroxy-2-deoxyguanosine (8-OHdG). The findings indicated that urinary 8-OHdG significantly increased at 3h Post and 24 h Post in both hypertrophy (75% of the 1RM)- and strength (90% of the 1RM)-intensity resistance exercise protocols ($p<0.05$). However, there were not significant differences in oxidative DNA damage between to RE protocols ($p>0.05$). In conclusion, a single session of hypertrophy- and strength-intensity resistance exercise lead to increase oxidative DNA damage and that these effects seems to be independent of the RE intensity.

Key Words: 8-hydroxy-2-deoxyguanosine, resistance exercise intensity, oxidative stress

---

INTRODUCTION

An increase in free radicals and reactive oxygen species (ROS) generation has been demonstrated following both aerobic and anaerobic exercise [1-3]. It has been shown that excess ROS generation may lead to oxidative damage to DNA, lipids and proteins [1,3-5]. Oxidative stress-induced DNA damage may play a significant role in processes that cause to many chronic diseases including cancer, cardiovascular disease and aging [6]. The DNA adduct, 8-hydroxy-2-deoxyguanosine (8-OHdG) is a reliable marker of oxidative DNA damage that can be measured in body fluids such as plasma and urine [7].

Previous studies have been shown that a single bout of RE can increase ROS generation, and leading to oxidative stress [4,8-10]. However, there are paucity data that examined 8-OHdG responses, a biomarker of oxidative DNA damage, after a single session of RE [11], and the findings of these studies demonstrated inconsistent results, including increased oxidative DNA damage after eccentric exercise compared to resting levels [11,12] and reported no significant changes [12]. Recently, Rahimi [4] demonstrated a significant increase in urinary 8-OHdG

concentration 24 h after a RE protocol (7 sets of 4 exercises using 60–90 1 repetition maximum) in the flat pyramid loading pattern. However, there aren't any data regarding the intensity of RE on oxidative DNA damage in athletes. For these reasons, the purpose of this study was to measure the oxidative DNA damage response to moderate and high intensity RE in young athletes.

MATERIALS AND METHODS

Subjects. Ten resistance trained men (age: 21.00±2.30 year; weight: 74.71±8.73 kg; height: 175.14±5.95 cm) who had at least 1 year experience in whole-body resistance training volunteered to participate in this study [13]. The experimental procedure was explained in details to all subjects and they completed a written informed consent approved by the local institutional ethics committee. Subjects were on their ordinary diet, and did not consume anabolic steroids or any other anabolic agents known to increase performance. No subjects were smoked or used antioxidant supplementation.

Experimental Design. One week before the experimental resistance exercise workouts, subjects came to the Exercise Physiology Lab for measuring height, weight and one repetition maximum (1RM) for the bench press, leg press, seated bar shoulder press, arm curls and lat pull down exercises (Table 1) [4,14]. Prior to 1RM testing, all subjects performed warm-up, which consisted of 3 min running, 5-10 repetitions at 50% of perceived maximum strength and stretching period. The warm-up procedure was held constant throughout all the testing sessions. Each subject performed 2 RE protocols of different intensities in crossover design. At least 72 hours of recovery time were allowed between each training session. Subjects were instructed not to train or be involved in strenuous activity for 48 hours before or after the experimental RE trial. All the subjects completed two RE protocols that consisted of 4 sets of the bench press (BP), leg press (LP), seated bar over-head press (OP), arm curls (AC) and lat pull down (LPD) exercises at 90% of 1RM with 3 min rest between sets (strength-intensity RE) and at 75% of 1RM with 90s rest between sets (hypertrophy-intensity RE), and each set was performed to exhaustion [13]. To ensure that all subjects were moving at approximately the same velocity for each repetition, each set was timed using a handheld stopwatch. The spotter called out a cadence for the eccentric and concentric phases of each repetition. The repetition velocity consisted of a 3-second eccentric phase followed by a 1-second concentric phase.

Table 1. 1RM value of the subjects

Exercise	1RM (M±SD)
Bench press (BP)	99.03±24.75
Lat pull down (LPD)	81.94±10.70
Seated bar over-head press (OP)	66.93±18.12
Arm curls (AC)	53.32±11.89
Leg press (LP)	203.24±45.98

Urine Collection and Biochemical Analyses. Urine samples were collected before the RE trail (Pre), immediately post (Post), 3 h post (3h Post) and 24 h post-exercise (24h Post) for analysis of urinary 8-OHdG excretion level. Urine samples were stored at -20º C until analyzed. Urinary 8-OHdG levels were analyzed using enzyme immunoassay (EIA) according to the procedures recommended by the manufacturer (Cayman Chemical, Catalog No. 589320, USA). The assay was carried out in duplicate utilizing the manufacturer's instructions.

Statistical Analyses:

Data are expressed as Mean ± SD. Statistical evaluation was performed with SPSS (SPSS, Chicago, IL) for windows. The data obtained for urinary 8-OHdG levels were analyzed using a 2 condition × 4 times repeated measures analysis of variance (ANOVA). Multiple comparisons with confidence interval adjustment by the *Bonferroni* method were used as post hoc when repeated measures ANOVA yielded significant results. The significance level was set at $p < 0.05$.

RESULTS

Urinary 8-OHdG concentrations before and following (Post, 3h Post and 24h Post) RE protocols with 90% of 1RM (strength-intensity RE) and 75% of 1RM (hypertrophy-intensity RE) are presented in Figure 1. Urinary 8-OHdG

concentrations significantly increased at 3h Post and 24 h Post RE with 75% of 1RM and 90% of 1RM ($p<0.05$, respectively). However, there were no significant differences in urinary 8-OHdG concentrations between two RE protocols ($p>0.05$).

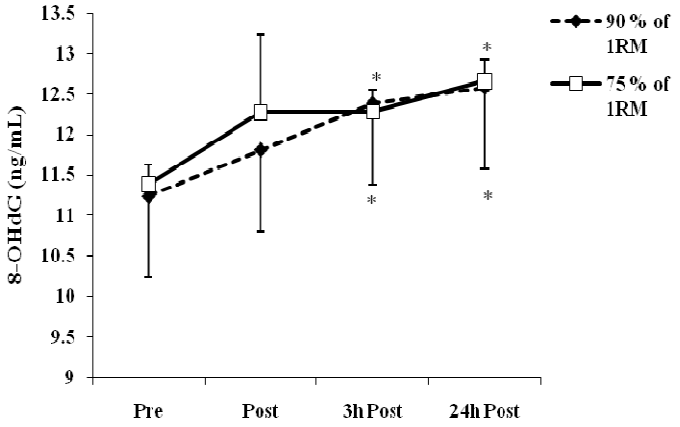

Figure 1. Urinary 8-OHdG concentration at pre-, post-, 3h post- and 24h post- resistance exercise with 90% (strength-intensity RE) and 75% of 1RM (hypertrophy-intensity RE) load.
** Significant difference with Pre at $p<0.05$.*

DISCUSSION

Acute RE may induce oxidative stress via enhanced formation of reactive oxygen and nitrogen species (RONS) [10,15]. The possible mechanisms responsible for RE-induced RONS formation includes xanthine-xanthine oxidase pathway, respiratory burst of neutrophils, catecholamine autooxidation, local muscle ischemia-hypoxia, and conversion of the weak superoxide to the strong hydroxyl radical by lactic acid, and alteration of calcium homeostasis [16]. It is clear that RE has the potential to result in increased free radical production, which may result in damage to DNA [4], protein [5,8] and lipids [17,18].

The present study compared oxidative DNA damage during 24-hour period following different RE intensity in young athletes. To our knowledge, this is the first study to compare the acute effects of RE performed at intensities corresponding to hypertrophy- and strength-intensity protocol, both performed to failure on biomarker of oxidative DNA damage (8-OHdG) in athletes. The results of this study showed that the urinary 8-OHdG concentration significantly increased 3-hour and 24-hour after both hypertrophy- and strength-intensity protocols. Previous investigations that examined the effects of RE on oxidative DNA damage reported inconsistent results [4,11,12]. It has been shown that, urinary 8-OHdG concentration significantly increased from pre-exercise level at 24 h post RE in athletes, performed at an intensity corresponding to 60–90 1RM in the flat pyramid loading pattern [4].

Furthermore, our results demonstrated a significant increase in 8-OHdG concentration that occurs independently of exercise intensity (75% vs. 90% of 1RM) after a single session of RE performed to failure. While no study has investigated RE intensity on 8-OHdG response, however, corroborating to our findings, three studies have reported an increase in biomarkers of oxidative stress (MDA concentration) after both low- (5 set × 15 repetition of squat at 60% of 1RM with 3 min rest) and high-intensity (5 set × 4 repetition of squat at 90% 1RM with 3 min rest) RE as well as after moderate- (75% of 1RM with 90s rest in back-squat exercise) and high-intensity RE (90% of 1RM with 5 min rest in back-squat exercise) in trained men [17,18]. However, it has been demonstrated that lipid peroxidation as measured by urinary 8-iso $PGF_{2\alpha}$ concentrations were significantly higher at Post and 3h Post RE in 75% of 1RM compared to RE with 90% of 1RM [13].

In conclusion, evidence from the present investigation suggests that hypertrophy- and strength-intensity RE protocols increased oxidative DNA damage as measured by urinary 8-OHdG level, and this increment occurred independently of RE intensity. It should be noticed that, in the present study, we have analyzed acute effects of

hypertrophy- and strength-intensity RE protocols. Further studies are necessary to evaluate chronic effects of such RE protocols on oxidative DNA damage. Since oxidative modifications of DNA can lead to mutations [6] and RE protocols in the current forms are associated with increment in oxidative DNA damage, so it would be useful that athletes consider the use of antioxidant supplementation in an attempt to attenuate any potential increase in oxidative stress.

Acknowledgements
The authors are grateful to the subjects who participated in this study. The authors also would like to acknowledge Mr Mehdi Malaki Masoleh at the Fadai Pathobiology and Endocrinology Laboratory for technical assistance with ELISA procedures. This research was supported by a grant from the Department of Physical Education, Takestan Branch, Islamic Azad University, to Mr Ali Khorjahani and Dr. Rahman Rahimi.

REFERENCES

[1] Bloomer RJ, Goldfarb AH. *Can J Appl Physiol* **2004**, 29: 245-63.
[2] Deaton CM. and Marlin DJ. *Clinical Techniques in Equine Practice*, **2003**, 2(3):278-291.
[3] Fisher-Wellman, K. and Bloomer, RJ. *Dynamic Medicine*, **2009**, 8:1.
[4] Rahimi R. *Journal of Strength and Conditioning Research*, **2011**, 25(12):3448-55.
[5] Sharafi H, and Rahimi R. *World Applied Sciences Journal*, **2011**,14(1): 121-126.
[6] Wu LL, Chiou CC, Chang PY, and Wu JT. *Clinical Chemistry Acta*, **2004**, 339, 1-9.
[7] De Zwart LL, Meerman JHN, Commandeur JNM, & Vermeulen NPE. *Free Radical Biological Medicine*, **1999**, 26, 202-226.
[8] Bloomer, RJ, Fry AC, Falyo MJ. and Moore CA. *Journal of Science and Medicine in Sport*, **2007**, 10: 411-417.
[9] McBride JM, Kraemer WJ, Triplett McBride T, Sebastianelli W. *Med Sci Sports Exerc* **1998**, 30:67-72.
[10] Uchiyama S, Tsukamoto H, Yoshimura S. and Tamaki T. *Pflugers Arch European Journal of Physiology*, **2006**, 452: 109-116.
[11] Radak Z, Pucsok J, Meeseki S, Csont T, Ferdinandy P. *Free Radic Biol Med* **1999**, 26: 1059-1063.
[12] Bloomer RJ, Goldfarb AH, Wideman L, Mckenzie M J, Consitt LA. *J Strength Cond Res* **2005**, 19:276-285.
[13] Khorjahani A, Rahimi R and Jalalvand A. *World Appl Sci J* **2012**, 16 (9): 1250-1255.
[14] Rahimi, R., M. Ghaderi, B. Mirzaei and H. Faraji. *World Applied Sciences Journal*, **2010**, 8(10): 1287-1293.
[15] Bloomer RJ, Goldfarb AH, Wideman L, McKenzie MJ, and Consitt LA, *Journal of Strength and Conditioning Research*, **2005**,19(2):276-285.
[16] Ji, LL. Free radicals and antioxidants in exercise and sports. In: Exercise and Sport Science. W.E. Garrett and D.T. Kirkendall, eds. Philadelphia, PA: Lippincott Williams and Wilkins, **2000**. pp. 299–317.
[17] Hoffman JR, Im J, Kang J, Maresh CM, Kraemer WJ, French D, Nioka S, Kime R, Rundell KW, Ratamess NA, Faigenbaum AD, and Chance B. *Journal of Strength and Conditioning Research*, **2007**, 21(1): 1117-24.
[18] Hudson MB, Hosick PA, McCaulley GO, Schrieber L, Wrieden J, McAnulty SR, Triplett NT, McBride JM, and Quindry JC. *Medicine Science in Sports and Exercise*, **2008**, 40: 542-548.