

## The Effects of Delorme and Oxford Techniques on Serum Cell Injury Indices and Growth Factor in Untrained Women

<sup>1</sup>S. Razmjou, <sup>1</sup>H. Rajabi, <sup>2</sup>M. Jannati, <sup>3</sup>M. Azizi and <sup>4</sup>A.A. Jahandideh

<sup>1</sup>Department of Physical Education, University of Tarbiat Moallem, Iran

<sup>2</sup>Department of Physical Education, University of Tehran, Iran

<sup>3</sup>Department of Physical Education, Islamic Azad University, Karaj Branch, Iran

<sup>4</sup>Department of Physical Education, University of Tarbiat Moallem Sabzevar, Iran

**Abstract:** The aim of this study was to investigate the effects of pyramid and reverse pyramid resistance trainings on serum cell injury indices (CK, AST, ALD) and growth factor (IGF-1) in untrained women. Also, it tried to assess the possible relationship between them. 34 subjects (age  $18.5 \pm 2.20$  years, weight  $56 \pm 9.86$  kg, height  $162 \pm 5.33$  cm) participated in this study and were randomly assigned to pyramid training (PT) (N=12), reverse pyramid training (RPT) (N=12) and control groups (CON) (N=10). The two experimental groups performed resistance training 3 days a week for 6 weeks. The pyramid group started their first set of ten repetitions at 50% of 10 RM, the second set of ten at 75% of 10 RM and the third set of ten at the 100% of 10 RM. The reverse pyramid group performed their sets quite the reverse: 100% of 10 RM, 75% of 10 RM and 50% of 10 RM. Blood samples were gathered before (T1) and 24 hours after (T2) the first training session and before (T3) and 24 hours after (T4) the last training session. ANOVA and t test were used to compare data. At T2, CK increased in both groups with no difference observed among the groups. After 6 weeks, there was no change in AST and ALD but CK increased again and this increase was lower compared to the first session (it was only significant in RP group). In fact, CK increment had a significant decrease in the last session compared to the first session in the PT group, which indicated that adaptations occurred. IGF-1 had no significant change and there was no relationship between growth factor and cell injury indices. After 6 weeks, adaptations occurred in response to CK and muscles underwent little pressure or even injury. These two methods had similar effects on growth factor response, but enzyme response was greater in Oxford method. Both methods were found to be efficient at developing strength.

**Key words:** DeLorme and Oxford resistance training % Cell injury % Growth factor % Adaptation

### INTRODUCTION

Resistive exercise is used extensively in training and rehabilitation situations and is increasingly recognized as an important exercise mode for health-related exercise settings [1]. It produces acute physiological responses and chronic adaptations that are critical to increasing muscular strength and hypertrophy [2]. Resistance training is reported to have many benefits [3]; however, it may increase cellular damage [4]. Mechanical loads impose stress, strain and sometimes damage on working muscles and connective tissues. Following a strenuous resistance exercise, there are marked increments in cellular related serum enzyme activities [5]. Several muscle proteins can act as indicators of increased permeability

of the muscle membrane: examples are Creatine Kinase (CK), Aspartate Aminotransferase (AST), Myoglobin (Mb), Aldolase (ALD) and LDH [6]. The appearance of one or more of these proteins is generally considered to reflect muscle damage [6]. Elevation of CK after exercise is a well known phenomenon [6]. The mechanism of exercise-induced serum enzyme rise has been attributed to either a metabolic effect on muscle fibers, producing increased membrane permeability or a mechanical stress on muscle fibers, resulting in membrane damage and fiber necrosis [5]. There are many studies which show that serum enzyme activities increase after resistance training [3, 4, 7, 8] but to our knowledge few studies have examined acute physiological response and chronic adaptations to different types of resistance training.

Following muscle damage, growth factors are produced [9]; indeed increased mechanical loadings increase growth factors such as IGF-1 (Insulin like growth factor1) [10]. Following resistance exercise, serum IGF-1 elevation has been reported presumably in response to GH-stimulated hepatic secretion. Anabolic hormones such as serum IGF-1 are critical to skeletal muscle growth [2]. IGF-1 is a small polypeptide hormone (70 amino acids) and is known as a positive modulator of muscle growth that is produced and secreted by the liver and skeletal muscles [2, 11]. IGF-1 stimulates protein synthesis and reduces protein breakdown [12] and enhance muscle hypertrophy [2]. It has been shown to have a strong anabolic effect on muscle tissues [1]. Overloaded muscle and subsequent mechanical damage after resistance training appear to be prominent stimuli for circulating IGF-1 secretion, which plays a prominent role during tissue remodeling process [13, 14]. Therefore, the concentration of circulating IGF-1 may increase during repair and growth processes [15]. However, some studies have shown no change in circulating IGF-1 during or following resistance training [11, 13, 16, 17].

Despite the proven effectiveness of resistance training in building strength, uncertainty still exists about the most efficient way to train. The work of DeLorme showed that with training, strength returns more quickly to atrophied muscles and hypertrophy is achieved by 20-30 repetitions [18]. DeLorme defined the ten-repetition maximum as the weight an individual could lift only ten times before temporary failure of the muscle occurred. One of the DeLorme's hypotheses is that the muscle should be warmed up by the time 10RM is reached. Therefore, once the 10RM has been established, the subjects begin sets of training by performing the first set of 10 at 50% 10RM, the second at 75% 10RM and the third at the 10RM [19]. Warm-up lifts did not intend to fatigue the muscle to the point that interfered with the subject's ability to complete the 10RM [19]. Instead these initial lifts were thought to be important in preventing muscle soreness and in teaching the subject how to complete the exercises, thereby permitting maximal exertion by the final set. Zinovieff identified another method to strengthen muscle, the Oxford technique, in which heavy resistance and low repetition was maintained as per DeLorme, but the full 10RM was the first set and was subsequently reduced to 75% and to 50% of 10 RM in the two remaining sets. It was thought that this decrement in resistance would mimic the progressive increase in muscle fatigue [20].

Considering the fact that Oxford (Reverse Pyramid resistance training, RPT) and DeLorme (Pyramid resistance training, PT) resistance training methods are very common in gyms and yet it is not clear which method is associated with more growth factor secretion, more strength gain and less cell injury indices and regarding the fact that data involving healthy subjects using these two methods is very limited and in view of deficiencies described above, the aim of this study is to examine the effect of acute and chronic DeLorme and Oxford resistance training on serum cell injury indices and growth factor in untrained women and also to assess possible relationship between IGF-1 and cell injury indices.

## MATERIALS AND METHODS

**Subjects:** 34 women (age:  $18.5 \pm 2.20$  years, weight:  $56 \pm 9.86$  kg, height:  $162 \pm 5.33$  cm) volunteered to participate in this study. Written consents were filled out by the subjects in accordance with the policy statement regarding the use of human subjects and informed consent of the Sport Science Research Center (SSRC) of Iran. The subjects had not been involved in any regular exercise and had no previous experience of resistance training. They were all healthy and were examined by a physician. Subjects were randomly assigned to pyramid training (DeLorme) (PT) (N=12), reverse pyramid training (Oxford) (RPT) (N=12) and control groups (CON) (N=10). Initially 12 subjects were assigned to the CON group; but as 2 subjects did not participate in the last blood sampling, data of the remaining 10 CON subjects was analyzed. The number of subjects in this study was based on the previous sources [21, 22]. Subjects were matched for physical characteristics before the study started. A minimum of one week was used for the familiarization period to allow subjects to accustom themselves to the exercises and to test procedures before initial data collection. The research project and observance of ethical issues was approved by Physiology Committee of Tarbiat Moallem University and Sport Science Research Center (SSRC) of Iran.

**Body Composition:** Lange skin fold calipers were used to measure body fat percentage. Three-site skin fold Jackson's prediction equation (triceps, suprailiac and thigh skin folds) was used to determine body fat percentage [23].

**Measurement of Strength:** Ten repetition maximum (10RM) was assessed for six exercises (biceps curl, triceps extension, lateral pull down, leg extension, leg curl and leg press) on 0<sup>th</sup>, 3<sup>rd</sup> and 6<sup>th</sup> training week. 10RM was defined as the maximum amount of weight that could be lifted 10 times through a full range of motion. In other words, the resistance used by the subject in a particular exercise was heavy enough to allow only the targeted number of repetitions (10 repetitions) for a given set, thus 10RM was a resistance by which the subject could only perform 10 repetitions [17]. Each subject performed a standard, general warm-up before 10RM testing. For 10RM test, each subject began by lifting a load with an interval of 2 minutes. The load increased at stages so that the 10RM could be determined in 10 attempts. We estimated 1RM from 10RM [24] and computed a net change score by subtracting the initial 1RM from the final 1RM.

**Resistance Training Protocol:** Each subject trained 3 days a week for 6 weeks. On exercise training days, the subjects performed some light stretching and warm-up exercises such as mild walking and jogging for 10-15 minutes. The pyramid (DeLorme) group started their first set of ten repetitions at 50% of 10RM, the second set of ten repetitions at 75% of 10RM and the third set of ten repetitions at the 100% of 10RM. The reverse pyramid (Oxford) group performed their sets quite the reverse: 100% of 10RM, 75% of 10RM and 50% of 10RM. The subjects lifted a weight at a comfortable speed. There was a 2-minute interval between each set. At the end of the third weeks, a new 10RM was established and training continued according to the new 10RM. At the end of the sixth week, the same muscle performance tests of strength were performed to determine a gain in strength.

**Blood Sampling:** Each participant provided a venous blood sample before (T1) 24 hours [4, 22, 23] after (T2) the first training session and before (T3) and 24 hours after (T4) the last training session. Venous blood samples (5ml) were obtained from superficial forearm vein during the same time of the day. Whole blood was centrifuged at 1500 g and the resulting serum was stored at -20°C until it was analyzed. Whole blood was used to determine serum CK using standard colorimetric procedures at 340 nm, serum aspartate aminotransferase

using enzymatic IFCC method and serum aldolase using enzymatic ultraviolet method. Serum IGF-1 concentration was determined using ELISA techniques. (In the present study, total circulating IGF-1 is determined, including hepatic and skeletal muscle).

**Statistical Analysis:** Kolmogorov-Smirnov test was used to check normal distribution. Hormonal and blood variable data were evaluated using an analysis of variance with repeated measures. Bonferroni's post hoc test was used to determine when significance occurred in the time intervals. Since blood samples of the control group were not gathered at T2 and T3, ANOVA with repeated measures was used only for the P and RP groups. Considering the fact that no change was observed in the control group at posttest, it was omitted in the statistical analysis. The independent Student's t-test was used to analyze the differences in muscle strength, hormone and blood variable data and body composition between experimental groups. The multiple regression was used to determine the relationship between growth factor and cell injury indices.  $P < 0.05$  were considered to be statistically significant.

## RESULTS

At baseline, the pyramid and reverse pyramid resistance training and control groups did not significantly differ with respect to age, height, weight, BMI, body fat percentage, circulating IGF-1, AST, ALD, CK or 1RM strength (Table 1, 2).

**Muscular Strength:** Resistance training was associated with increased muscle strength (Table 2,  $P = 0.00$ ). In non-exercising control group, 1RM strength did not change ( $P = 0.92$ ). Strength increases (expressed as kg) were similar in both groups except for the biceps curl which was significantly greater in reverse pyramid group when compared with pyramid group ( $P = 0.04$ ).

**Creatine Kinase:** Resting serum CK is presented in Table 1. CK was significantly elevated at T2 in both groups (Fig.1,  $P = 0.02$  for PT,  $P = 0.004$  for RPT) with no difference observed between the groups ( $P = 0.21$ ). After 6 weeks of resistance training (T4), CK increased again, but this increase was lower compared to the first session and it was only significant in RP group ( $P = 0.02$ ).

Table 1: Baseline values for pyramid (PT), reverse pyramid resistance training (RPT) and control groups

	Control	PT	RPT
Age (yr)	18.57±2.43	18.5±2.27	18.6±2.22
Height (cm)	161±4.18	162±5.04	162±6.58
Weight (Kg)	56.35±10.09	58.55±11.48	53.20±8.09
BMI (Kg/m <sup>2</sup> )	21.69±3.88	21.94±3.97	20.01±2.04
Body fat (%)	25.99±4.46	26.28±6.14	24.82±4.01
Serum IGF-1 (ng.mLG <sup>1</sup> )	567.27±125.65	698.84±143.3	675.11±176.2
CK (U/L)	98.71±35.37	107.20±46.39	101.1±43.61
ALD (U/L)	3.98±1.42	3.31±2.71	4.48±2.06
AST (U/L)	17.57±2.29	17.20±4.46	16.70±2.88

Values are mean± SD. N for control=10, N for PRT=12 and N for RPRT=12

Table 2: Muscular strength change after 6 weeks of training

Exercises	Group	Mean± SD		P-Value	
		Baseline	6 weeks	within group	between groups
Biceps curl (kg)	PT	13.33±3.140	23.47±4.67	0.001*	0.04*
	RPT	13.33±3.140	26.16±3.24	0.001*	
Triceps extension (kg)	PT	16.66±3.510	27.38±5.0	0.001*	0.40
	RPT	15.99±3.440	25.37±2.45	0.001*	
Lateral pull down (kg)	PT	21.99±3.210	31.16±5.27	0.001*	0.73
	RPT	20.66±2.100	30.40±3.31	0.001*	
Leg extension (kg)	PT	30.65±9.000	50.43±10.90	0.001*	0.056
	RPT	30.66±6.440	56.66±7.02	0.001*	
Leg curl (kg)	PT	16.37±7.350	25.16±10.26	0.002*	0.57
	RPT	14.49±6.800	21.96±7.28	0.003*	
Leg press (kg)	PT	85.99±17.05	125.59±17.37	0.001*	0.11
	RPT	75.99±13.03	122.17±13.12	0.001*	

PT= Pyramid Resistance training, RPT= Reverse Pyramid Resistance Training. \* P < 0.05 (significant)

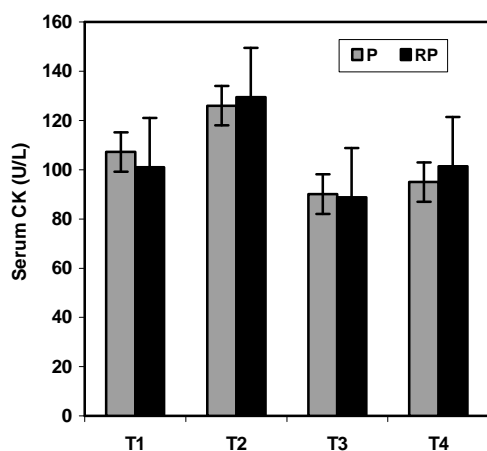


Fig. 1: Effect of PT and RPT resistance training on serum CK. In both groups CK significantly elevated at T2 versus T1. At T4, CK had a significant increase only in RPT group. Also at T4, CK had a significant decrease compared to T2 in PT group. \* P < 0.05 in groups and at p<0.05 between the groups. Values are mean SE, N=12 for each group.

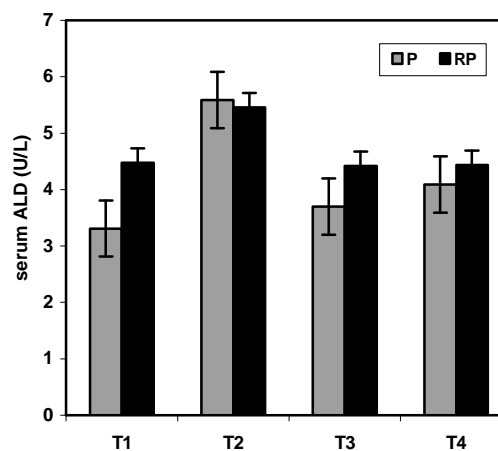


Fig. 2: Effect of PT and RPT resistance training on serum ALD. ALD levels were not significantly affected by training. Values are mean SE, N=12 for each group

In fact, CK increment had a significant decrease in the last session (T4) compared to the first session (T2) in PT group (P=0.027), which showed that adaptations

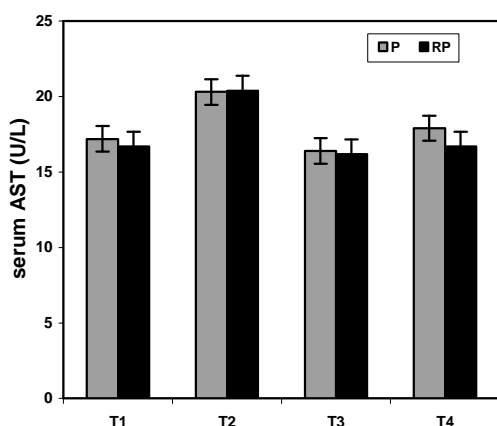


Fig. 3: Effect of PT and RPT resistance training on serum AST. AST levels were not significantly affected by training. Values are mean SE, N=12 for each group

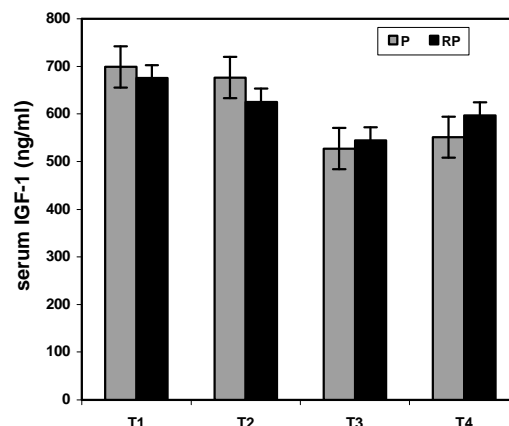


Fig. 4: Effect of PT and RPT resistance training on circulating IGF-1. IGF- 1 levels were not significantly affected by training. Values are mean SE, N=12 for each group

Table 3: P-Value for the relationship between IGF-1 and AST, ALD, CK

Groups	Variables		P-Value at D1	P-Value at D2
PT	IGF-1	AST	0.49	0.78
		ALD	0.63	0.93
		CK	0.22	0.71
RPT	IGF-1	AST	0.13	0.24
		ALD	0.23	0.17
		CK	0.78	0.87

D1: T2 and T1 difference, D2: T4 and T3 difference

Table 4: Body composition change after 6 weeks of resistance training

Parameter	PT	RPT	P-Value	
			PT	RPT
Weight (Kg)	58.45±10.25	53.50±7.76	0.85	0.46
BMI (Kg/m <sup>2</sup> )	21.91±3.48	20.15±2.09	0.87	0.38
Body fat (%)	25.93±5.66	24.93±3.70	0.21	0.64
Arm CIR (cm)	26.60±2.80	25.35±2.47	0.27	0.55
Leg CIR (cm)	51.60±4.45	48.45±3.75	0.18	0.26
Arm skin fold (mm)	15.40±7.39	15.50±5.35	0.51	0.81
Leg skin fold (mm)	31.80±6.81	28.80±5.34	1.00	0.64

PT= Pyramid Resistance training, RPT= Reverse Pyramid Resistance Training  
CIR= circumference

occurred. This decrease was not significant in RP group (P=0.24). In non-exercising control group, blood samples were gathered at the beginning of the study and 6 weeks later. CK did not change in this group (98.71±35.3U/L at 0<sup>th</sup> week and 83.14±15.93U/L at 6<sup>th</sup> week, P=0.21).

**Aldolase:** Neither pyramid nor reverse pyramid groups were associated with changes in ALD at T2 and also after six weeks of resistance training at T4 (Fig. 2, P=0.25 for PT, P=0.32 for RPT). There was no difference between the groups (P=0.48 at T2, P=0.57 at T4). In non-exercising

control group, ALD did not change (3.98±1.42U/L at 0<sup>th</sup> week and 3.99±1.47U/L at 6<sup>th</sup> week, P=0.98).

**Aspartate Aminotransferase:** No significant change was observed in serum AST after one session (T2) or after 6 weeks of resistance training (T4) (Fig. 3, P=0.081 for PT, P=0.23 for RPT). There was no significant difference between the groups either (P=0.85 at T2, P=0.55 at T4). In non-exercising control group, AST did not change. (17.57±2.22U/L at 0<sup>th</sup> week and 18.42±2.57U/L at 6<sup>th</sup> week, P=0.35).

**Serum Insulin-Like Growth Factor:** Resting concentrations of circulating IGF-1 are presented in Table 1. Resistance training was associated with a decrease in circulating IGF-1 after one session (T2), which was not significant (Fig. 4,  $P=1.00$  for PT,  $P=1.00$  for RPT) and it was not significantly different in response to pyramid versus reverse pyramid resistance training ( $P=0.67$ ). After six weeks of resistance training (T4), circulating IGF-1 increased, which was not significant either in groups ( $P=1.00$  for PT,  $P=0.85$  for RPT) or between groups ( $P=0.58$ ). In non-exercising control subjects, serum IGF-1 did not change ( $567.27 \pm 125$  ng.mLG<sup>1</sup> at 0<sup>th</sup> week and  $570.42 \pm 132$  ng.mLG<sup>1</sup> at 6<sup>th</sup> week,  $P=0.64$ ).

No statistically significant relationship was found between serum IGF-1 and cell injury indices (AST, ALD, CK) in both groups (Table 3).

**Body Composition:** No change was observed in body fat percentage, weight, BMI, arm and leg circumferences, arm and leg skin folds (Table 4).

## DISCUSSION AND CONCLUSION

We found that the acute response (T2) to resistance training (DeLorme or Oxford method) is a significant increment in serum CK. Maybe it is a result of increased membrane permeability due to mechanical load or metabolic effects [5] which indicates a possible relationship between muscle damage and strenuous performance. The serum CK level can be raised from the damage of the muscle tissue as a consequence of intense training [25]. Indeed It is established that resistance exercise can damage muscle tissue [26]. Determination of CK is the most common marker to assess skeletal muscle damage [27]. In studies of high force eccentric exercises, a common variable used to estimate the degree of muscle cell injury is the enzyme creatine kinase, which is released from damaged cell and accumulated in the blood [28, 29]. However, measurements of other enzyme levels (ALD, AST) are also indicative markers of skeletal muscle injury [6, 27]. Muscle tissue damage after resistance training and serum enzyme increment have been reported in many studies [7, 8, 30, 31, 32], but to our knowledge, few studies investigated the adaptations occurred in response to a training program in different protocols. In our study, although AST and ALD increased, it was not as significant and dramatic as CK. Since among these three enzymes, only one of them had a significant increase, there is a doubt about the occurrence of muscle damage after these trainings.

At T4, both PT and RPT groups had increment in serum CK (but it was only significant in RPT). Six weeks of resistance training were associated with adaptations, since CK increment at the last session (T4) was lower compared to the first session (T2) and a significant decrement was observed in the pyramid group. Therefore, due to resistance training, adaptations in response occurred and muscles underwent little pressure or even injury. However, it seems that reverse pyramid exercise imposes greater stress on the muscles even after muscular adaptations (strength improvement), which probably relates to the nature of this type of exercise because in this method, the participants should lift the heaviest weight in the first attempt before the muscles become completely prepared.

Circulating IGF-1 concentration had no significant change. Indeed circulating concentrations of IGF-1 seem to respond to exercise in a biphasic manner. Circulating IGF-1 levels have been found to decrease during brief exercise periods in a number of studies and to increase during more chronic periods of exercise [33, 34, 35, 36]. As Raastad *et al.* (2000) found, during a strength training program, circulating IGF-1 decreased after eight days of training [37] while Borst *et al.* (2001) found that circulating IGF-1 had increased after 13 weeks in a 25-week resistance training program [38]. A rapid increase in lifting capacity correlated with this rise. Therefore, the increase in muscles strength at the beginning of the training is due to neural adaptations (when serum IGF-1 decreased) while latter changes have a much higher contribution of structural changes such as muscular hypertrophy [39]. In our study circulating IGF-1 decreased at T2 (not significant) but after 6 weeks of resistance training it increased, though it was not significant. Possibly, there would be a statistically significant increase if our training period lasted more.

An explanation for this biphasic phase of serum IGF-1 response to exercise is the inflammatory hypothesis [40]. Beginning phase of an exercise program leads to a tremendous increase in serum concentrations of various cytokines which increase inflammation. This was observed in a study by Scheet *et al.* (2002) in which they had participants to play intense soccer. Proinflammatory cytokines such as tumor necrosis factor increased and serum IGF-1 decreased with this increase [36]. As training progresses, participants successfully adapt to the training load with subsequent lowering of proinflammatory cytokines. As they lower, serum IGF-1 levels increase.

On the other hand, there are some factors that can affect circulating IGF-1; factors like IGFBPs (IGF binding proteins) have the potential to alter serum IGF-1 action.

Serum IGF-1 binds to as many as 10 distinct serum proteins [38]. Nearly all circulating IGFs are bound to IGF binding proteins. These factors regulate serum IGF availability; indeed they protect serum IGF-1 from degradation and lower the free concentrations of serum IGF-1 [38]. Resistance exercise has been shown to elevate IGF-1 [38]. Resistance exercise has been shown to elevate IGF-1 [38]. Resistance exercise has been shown to elevate IGF-1 [38]. Resistance exercise has been shown to elevate IGF-1 [38]. Resistance exercise has been shown to elevate IGF-1 [38].

Due to lack of a significant relationship between circulating IGF-1 and AST, ALD and CK, it is possible that the amount of muscle tissue damage observed after resistance training does not require alterations in circulating IGF-1. The relatively low AST and ALD response also indicate that training status may influence the amount of muscle tissue disruption and thus types of operational mechanism of remodeling [17]. Kraemer et al. (1995) also found no relationship between serum IGF-1 and CK [17].

This study aimed to determine the optimal method of developing muscle strength between DeLorme and Oxford methods. Determining the best method for building strength is relevant to individuals of all ages and all health levels. Our results showed that both DeLorme and Oxford methods develop strength in healthy girls. Muscle strength improved in all exercises without any change in body composition. Optimal strength development can occur when a muscle contracts against a degree of resistance high enough to reach maximal or near maximal effort [20]. Early gains in strength are attributed to nervous system adaptations and latter strength benefits are attributed to hypertrophy and structural changes [39]. Considering the increment of strength without a change in body composition, we came to the conclusion that strength increment in our study is due to neural adaptations. Kemmler *et al.* (2004) also found that single sets and multiple sets of resistance training increased women strength without any change in body mass and body composition [41].

Strength increases had no significant difference among the groups except for the biceps curl (greater gains were observed in the Oxford group). Although the mechanism is not clear and needs more investigations, it seems that training stress is more expressed in biceps because of its weakness. Older studies found Oxford method more efficient [36] and in recent studies Fish *et al.* (2003) reported no difference between these two methods in developing strength [20].

In conclusion, it should be mentioned that these methods have similar effects on growth factor response,

but enzyme response was greater in Oxford method. And there is no relationship between IGF-1 changes and cell injury indices. Both DeLorme and Oxford resistance training methods were found to be efficient in developing strength and are recommended to those who are eager to improve their physical fitness and strength. Considering the greater increment in CK levels at the beginning of the training, it is recommended that a lower stress should be imposed at the first sessions in untrained subjects.

## ACKNOWLEDGEMENTS

This study was supported in part by a research grant from *Sport Science Research Center (SSRC) of Iran.*

## REFERENCES

1. Kraemer, R.R., J.L. Kilgore, G.R. Kraemer and V.D. Castracane, 1992. Growth hormone, IGF-1 and testosterone responses to resistive exercise. *Med. Sci. Sports Exerc.*, 24(12): 1346-1352.
2. Kraemer, W.J. and N.A. Ratamess, 2005. Hormonal responses and adaptations to resistance exercise and training. *Sports Medicine*, 35(4): 339-61.
3. Guzel, N.A., S. Hazar and D. Erbas, 2007. Effects of different resistance exercise protocols on nitric oxide, lipid peroxidation and creatine kinase activity in sedentary males. *J. Sports Science and Medicine*, 6(4): 417-422.
4. Liu, J.F., W.Y. Chang, K.H. Chan, W.Y. Tsai, C.L. Lin and M.C. Hsu, 2005. Blood lipid peroxides and muscle damage increased following intensive resistance training of female weightlifters. *Ann. N Y Acad Sci.*, 1042: 255-61.
5. Karamizrak, S.O., E. Ergen, I.R. Töre and N. Akgün, 1994. Changes in serum creatine kinase, lactate dehydrogenase and aldolase activities following supramaximal exercise in athletes. *J. Sports Med. Phys. Fitness.*, 34(2): 141-146.
6. Driessen-Kletter, M.F., G.J. Amelink, P.R. Bar and J. Gijn, 1990. Myoglobin is a sensitive marker of increased muscle membrane vulnerability. *J. Neurol.*, 237: 234-238.
7. Dixon, C.B., R.J. Robertson, F.L. Goss, J.M. Timmer, E. Nagle and R.W. Evans, 2003. The Effect of Resistance Training Status on Free Radical Production and Muscle Damage Following an Acute Resistance Exercise Bout. *Med. Sci. Sports Exerc.*, 35(5): s158.

8. Pettersson, J., U. Hindorf, P. Persson, T. Bengtsson, U. Malmqvist, V. Werkstrom and M. Ekelund, 2008. Muscular exercise can cause highly pathological liver function tests in healthy men. *Br. J. Clin Pharmacol.*, 65: 253-9.
9. Goldspink, G., 2001. Method of treating muscular disorders. United States Patent No. US 6,221,842 B1.
10. Adams, G.R., 2002 Invited review: autocrine/paracrine IGF-I and skeletal muscle adaptation. *J. Applied Physiol.*, 93: 1159-1167.
11. Walker, K.S., R. Kambadur, M. Sharma and H.K. Smith, 2004 Resistance Training Alters Plasma Myostatin but not IGF-1 in Healthy Men. *Med. Sci. Sports Exerc.*, 36(5): 787-93.
12. Fryburg, D.A., 1994. Insulin-like growth factor I exerts growth hormone- and insulin-like actions on human muscle protein metabolism. *Am. J. Physiol.*, 267(2 Pt 1): E331-6.
13. Nicklas, B.J., A.J. Ryan, A.J. Treuth, S.M. Harman, M.R. Blackman, B.F. Hurley and M.A. Rogers, 1995. Testosterone, growth hormone and IGF-I responses to acute and chronic exercise in men aged 55-70 years. *Int. J. Sports Med.*, 16: 445-450.
14. Zhang, Y., J. Jiang, R.A. Black, G. Baumann and S.J. Frank, 2000. Tumor necrosis factor-alpha converting enzyme (TACE) is a growth hormone binding protein (GHBP) sheddase: the metalloprotease TACE/ADAM-17 is critical for (PMA-induced) GH receptor proteolysis and GHBP generation. *Endocrinol.*, 141(12): 4342-4348.
15. Hellsten, Y., H.A. Hansson, L. Johnson, U. Frandsen and B. Sjödén, 1996. Increased expression of xanthine oxidase and insulin-like growth factor I (IGF-I) immunoreactivity in skeletal muscle after strenuous exercise in humans. *Acta Physiol. Scand.* 157(2): 191-197.
16. Kraemer, W.J., S.J. Fleck, J.E. Dziados, E.A. Harman, L.J. Marchitelli, S.E. Gordon, R.P. Mello, P.N. Frykman, L.P. Koziris and N.T. Triplett, 1993. Changes in hormonal concentrations after different heavy-resistance exercise protocols in women. *J. Appl. Physiol.*, 75: 594-604.
17. Kraemer, W.J., B.A. Aguilera, M. Terada, R.U. Newton, J.M. Lynch, G. Rosendaal, J.M. McBride, S.E. Gordon and K. Hakkinen, 1995. Responses of IGF-I to endogenous increases in growth hormone after heavy-resistance exercise. *J. Appl. Physiol.*, 79: 1310-1315.
18. DeLorme, T. and A.L. Watkins, 1948. Techniques of progressive resistance exercises. *Arch. Phys. Med.*, 29: 263-273.
19. DeLorme, T. and A.L. Watkins, 1945. Restoration of power by heavy-resistance exercises. *J. Bone Joint Surg*, 27: 645-667.
20. Fish, D.E., B.J. Krabak, D. Johnson-Greene and B.J. DeLateur, 2003. Optimal resistance training: comparison of Delorme with Oxford techniques. *American J. Physical Medicine and Rehabilitation*, 82(12): 903-909.
21. Chen, T.C. and S.S. Hsieh, 2001. Effects of a 7-day eccentric training period on muscle damage and inflammation. *Med. Sci. Sports Exerc.*, 33: 1732-1738.
22. Marx, J.O., N.A. Ratamess, B.C. Nindl, L.A. Gotshalk, J.S. Volek, K. Dohi, J.A. Bush, A.L. Gómez, S.A. Mazzetti, S.J. Fleck, K. Häkkinen, R.U. Newton and W.J. Kraemer, 2001. Low-volume circuit versus high-volume periodized resistance training in women. *Med. Sci. Sports Exerc.*, 33: 635-643.
23. Jackson, A.S., M.L. Pollock and A. Ward, 1980. Generalized equations for predicting body density of women. *Med. Sci. Sport Exerc.*, 12: 175-182.
24. Baechle, T.R. and R.W. Earle, 2000. The essentials of strength training and conditioning. IL: Human Kinetics.
25. Brancaccio, P., N. Maffulli and F.M. Limongelli, 2007. Creatine kinase monitoring in sport medicine. *Br. Med. Bull.*, 81-82: 209-30.
26. Yamamoto, L.M., D.A. Judelson, M.J. Farrell, E.C. Lee, L.E. Armstrong, D.J. Casa, W.J. Kraemer, J.S. Volek and C.M. Maresh, 2008. Effects of hydration state and resistance exercise on markers of muscle damage. *J. Strength and Conditioning Res.*, 22(5): 1387-1393.
27. Bohlmeier, T.J., A.H.B. Wu and M.B. Perryman, 1994. Evaluation of laboratory tests as a guide to diagnosis and therapy of myositis. *Rheum Dis. Clin North Am.*, 20: 845.
28. Clarkson, P., A. Kearns, P. Rouzier, R. Rubin and P. Thompson, 2006. Serum creatinekinase levels and renal function measures in exertional muscle damage. *Pediatric Critical Care Medicine*, 38(4): 623-627.
29. Sewright, K.A., M.J. Hubal, A. Kearns, M.T. Holbrook and P.M. Clarkson, 2008. Sex differences in response to maximal eccentric exercise. *Med. Sci. Sports Exerc.*, 40(2): 242-51.
30. Hayward, R., K.A. Hutcheson and C.M. Schneider, 2003. Influence of acute resistance exercise on cardiac biomarkers in untrained women. *J. Emergency Medicine*, 25(4): 351-356(6).



31. Kuo, Y.C. and J.C. Lin, 2003. Effect of different intensity resistance exercise on creatine kinase and malondialdehyde H-13K free communication/poster exercise training. *Med. Sci. Sports Exerc.*, 35(5) Supplement, 1: S368.
32. Li, B.G., P.O. Hasselgren, C.H. Fang and G.D. Warden, 2004. Insulin-like growth factor-I blocks dexamethasone-induced protein degradation in cultured myotubes by inhibiting multiple proteolytic pathways. *J. Burn. Care Rehabil.*, 25: 112-118.
33. Eliakim, A., J.A. Brasel, S. Mohan, T.J. Barstow, N. Berman and D.M. Cooper, 1996. Physical fitness, endurance training and the growth hormone-insulin-like growth factor I system in adolescent females. *J. Clin Endocrinol. Metab.*, 81: 3986-92.
34. Eliakim, A., J.A. Brasel, S. Mohan, W.L. Wong and D.M. Cooper, 1998. Increased physical activity and the growth hormone-IGF-I axis in adolescent males. *Am. J. Physiol. Regul. Integr. Comp. Physiol.*, 275: R308-14.
35. Eliakim, A., T.P. Scheet, R. Newcomb, S. Mohan, D.M. Cooper, 2001. Fitness, training and the growth hormone:insulin-like growth factor I axis in prepubertal girls. *J. Clin Endocrinol. Metab.*, 86: 2797-802.
36. Scheet, T.P., D. Nemet, J. Stoppani, C.M. Maresh, R. Newcomb and D.M. Cooper, 2002. The effect of endurance-type exercise training on growth mediators and inflammatory cytokines in pre-pubertal and early pubertal males. *Pediatr Res.*, 52: 491-7.
37. Raastad, T., T. Bjoro and J. Hallen, 2000. Hormonal responses to high- and moderate-intensity strength exercise. *European J. Applied Physiol.*, 82: 121-128.
38. Borst, S.E., D.V. De Hoyos, L. Garzarella, K. Vincent, B.H. Pollock, D.T. Lowenthal and M.L. Pollock, 2001. Effects of resistance training on insulin-like growth factor-I and IGF binding proteins. *Med. Sci. Sports Exerc.*, 33(4): 648-53.
39. Fleck, S.J. and W.J. Kraemer, 2004. Designing resistance training programs. Second edition. Champaign, IL: Human Kinetics.
40. Kraemer, W.J., K. Hakkinen, R.U. Newton, B.C. Nindle, J.S. Volek, M. McCormick, L.A. Gotshalk, S.E. Gordon, S.J. Fleck, W.W. Campbell, M. Putukian and W.J. Evans, 1999. Effect of heavy- resistance training on hormonal response patterns in younger vs. older men. *J. Appl. Physiol.*, 87: 982-992.
41. Kemmler, W.K., D. Lauber, K. Engelke and J. Weineck, 2004. Effect of single-vs. multiple-set resistance training on maximum strength and body composition in trained postmenopausal women. *J. Strength and Conditioning Res.*, 18(4): 689-694.