©Journal of Sports Science and Medicine (2014) **13,** 680-688
http://www.jssm.org

Research article

Effects of Resistance Training on Muscle Strength, Endurance, and Motor Unit According to Ciliary Neurotrophic Factor Polymorphism in Male College Students

Ae-Rim Hong [1], Sang-Min Hong [2] and Yun-A Shin [1]✉
[1] Department of Exercise Prescription & Rehabilitation, College of Sports Science, Dankook University, Anseo-dong, Cheonan-si, Chungnam Republic of Korea; [2] Department of Physical Education, College of Education, Dongguk University, Pildong-ro-1-gil, Jung-gu, Seoul, Republic of Korea

Abstract
Changes in muscle mass and strength across the adult age span are variable and related to the ciliary neurotrophic factor (CNTF) genotype. In particular, a single CNTF haplotype (1357 G→A) is important for neuronal and muscular developments and may be associated with muscle strength response to resistance training. We examined whether CNTF genotype differentially influences the effect of resistance training on neuromuscular improvement in male college students. Resistance training of the upper extremities comprised 3 sets at 75%–85% intensity per 1 repetition maximum, 3 times a week, for a total of 8 weeks. We measured isokinetic muscle function of the elbow joint with regard to strength (60°/s) and endurance (180°/s) by using an isokinetic dynamometer. The biceps brachii (BB) and brachioradialis muscles were studied using surface electromyography with spike-triggered averaging to assess surface-detected motor unit potential (SMUP) area. After resistance training, the SMUP of the BB increased significantly at 60°/s ($p < 0.05$), but no difference in the CNTF genotype was observed. The SMUP of the BB at 180°/s increased significantly in the GG/AA genotype group compared with that in the GA genotype group ($p < 0.05$). The average power of the elbow flexor at 180°/s increased significantly after resistance training ($p < 0.05$), but again, no difference in the CNTF genotype was observed. Thus, improvements in muscle strength and endurance may have resulted directly from resistance training rather than from genetic factors related to nerves in muscle tissue.

Key words: Resistance training, muscle strength, muscle endurance, motor unit, ciliary neurotrophic factor genotype.

Introduction

Resistance exercise pertains to a wide range of activities leading to muscle contractions as a response to resistance to an external force. Several previous studies have confirmed the various effects of resistance training; overload stress following resistance training reportedly increases muscle strength and the cross-sectional area of muscle fibers, improves muscle function (Moore et al., 2004), and delays the aging-related process of sarcopenia (Johnston et al., 2008). Therefore, resistance training serves as the driving force for a healthy life and is the main reason for improving athletic performance in various sports (Behringer, 2010). In response to resistance training, muscle strength increases (Bandy et al., 1990; Kraemer et al., 1988; Rose et al., 1982) with increases in motorization of muscle motor units, which are affected by changes in nerve effectiveness (Chestnut and Docherty, 1999; Hakkinen, 1989; Sale, 1988; Wojtys et al., 1996). Muscle contractions are regulated by the central nervous system and influenced by the fast or slow twitch of fibers, or neural reinforcement patterns (Buller et al., 1987). Improvement in muscle function with resistance training is much more effective through activity of the nervous system.

Neurotrophic factors are proteins that act as biochemical change factors to help neuronal existence, growth, division, and protection (Lewin and Barde, 1996); they also play essential roles in various stages of repair of damaged neurons (Connor and Dragunow, 1998). Ciliary neurotrophic factor (CNTF) facilitates motor nerve function (Sendtner et al., 1992). CNTF is also a cytokine belonging to the interleukin (IL)-6 family, and when combined with the CNTF receptor, performs the role of a chemical messenger for target tissues such as motor nerves and skeletal muscles (Ip et al., 1993). Its function as a myotrophic factor has also been investigated (DiStefano et al., 1996; Forger et al., 1993; Helgren et al., 1994), and, according to Guillet et al. (1999), the concentration of CNTF in the sciatic nerve in mice was positively correlated with swimming capacity as well as the twitch and titanic tension of muscles. In addition, when CNTF was exogenously injected in the soleus muscle of elderly mice with underexpression of CNTF, muscle strength and cross-sectional area increased (Fraysse et al., 2000).

Since Takahashi et al. (1994) first reported the substitution of the human CNTF gene 1357 G→A single-nucleotide polymorphism (SNP), several studies have investigated the relationship between CNTF polymorphism and muscle function. Large-scale, cross-sectional studies have demonstrated that individuals possessing CNTF variants showed greater muscle strength than those with the G1357G genotype (Arking et al., 2006; De Mars et al., 2007; Roth et al., 2001). However, the effects of resistance training varied depending on the CNTF gene polymorphism, i.e., women with the GG genotype showed a greater increase in muscle strength after 12 weeks of training than those with the AA genotype, whereas men showed no difference in strength based on genotype (Walsh et al., 2009). Walsh et al. (2009) explained that these sex-related differences may be attributable to hormones such as androgens and suggested the need for further investigation on the effects of resistance training.

Received: 14 February 2014 / Accepted: 16 June 2014 / Published (online): 01 September 2014

It has been suggested that variations in the results of resistance training are due to CNTF polymorphism, which is regulated by hormones and the mobilization of nerve roots. Conwit et al. (2005) reported that an increase in muscle force in participants with the GA genotype, compared with those with the GG genotype, was associated with a more effective force per motor unit size mobilization. During resistance training, the increase in muscle strength in the early phase is attributable to neural adaptation rather than from changes in the muscle size. Any further increase thereafter is mostly attributable to muscular hypertrophy (William et al., 2001). CNTF sustains the survival of motor neurons in vitro and in vivo (Oppenheim et al., 1991), and because differences in muscle function or the effects of resistance training are associated with CNTF polymorphism, it can be assumed that there may be a difference in neural adaptation during the initial stage of resistance training.

Previous studies have compared differences in muscle strength and hypertrophy (Klausen et al., 1981; Roth et al., 2001) as well as nerve root mobilization (Conwit et al., 2005) during resistance training. However, studies on the mechanism of muscle strength based on nerve root mobilization are limited. Therefore, in this study, we conducted an 8-week resistance exercise program in healthy male college students to examine; i) differences in nerve root mobilization units and muscle strength every 2-weeks across the 8-week resistance exercise program, and ii) to determine the role of CNTF polymorphism in muscle strength and endurance and the adaptation of nerve roots.

Methods

Subjects

The study population included 83 healthy male college students in their twenties (average age, 22.56 [SD, 1.38] years). The subjects were recruited after responding to an advertisement in college newspapers in Chungman. After explaining the purpose and procedure of the study, we collected blood samples, which were subjected to CNTF polymorphisms. Of the 83 subjects, 73 (87.9%) exhibited the GG genotype, 9 (10.8%) exhibited the GA genotype, and 1 (1.2%) exhibited the rare AA genotype. Results of this screening were used to classify subjects into the GG genotype group (n = 8) or the GA/AA genotype group (n = 10). We selected participants who showed no differences in age, weight, muscle mass, or body fat percentage. The exclusion criteria used before selection of the participants also included the following: participation in any specific diet, smoking, use of medication, use of steroids, and medical deviations. We then applied resistance training in the 2 groups of subjects (Table 1). To decrease the influence of previous exercise on the training results, we asked subjects to refrain from exercise of any type for 4 weeks prior to the study. It has been reported that the effects of training and muscle capillary density decrease significantly 4 weeks after training cessation (Klausen et al., 1981; Mujika and Padilla, 2001). To confirm that they do not participate in the exercise, we telephoned the subjects every day during those 4 weeks. Following this, during the 8 weeks of the study, subjects were prohibited from engaging in intense physical activity, taking medication, or drinking alcohol. It has been reported that neuromuscular adaptation and muscle fiber size increase significantly after 8 weeks of resistance training (Henneman et al., 1965; Moore et al., 2004). Subjects also were asked to complete a diet questionnaire and maintain their current diet and amount of food consumed. Informed consent was obtained from all the subjects prior to their participation. The present study was approved by the Institutional Ethics Committee of Physical Education of Dankook University.

Table 1. Subjects' physical characteristics. Data are means (±SD).

Variables	Groups		t
	NN (n =8)	NM+MM (n =10)	
Age, y	23.00 (0.76)	22.20 (1.69)	1.341
Height, cm	175.25 (4.86)	175.70 (5.31)	–.185
Weight, kg	73.61 (8.49)	71.41 (8.82)	.535
Muscle mass, kg	59.86 (5.97)	57.69 (6.83)	.708
Fat-free mass, kg	63.63 (6.42)	61.41 (7.40)	.668
Body fat, %	14.10 (2.29)	14.53 (3.40)	–.306
Waist-hip ratio	0.83 (0.02)	0.81 (0.03)	1.241
BMI, kg/m²	23.79 (1.79)	23.02 (2.39)	.752

BMI, body mass index; MM, mutation homozygote; NM, mutation heterozygote; NN, normal homozygote

Anthropometrics

Using bioelectrical impedance (Inbody 7.0; Biospace, Seoul, Korea), we measured weight, muscle mass, body composition, and body fat percentage. Body mass index (BMI) was calculated as the ratio of weight (kg) over height (m^2).

Genotyping

To decrease the influence of the components of blood on genotyping, subjects were asked to fast for 12 h prior to blood collection. After subjects underwent mind and body relaxation for 30 min, approximately 3 mL of blood was drawn from the median antebrachial vein using a disposable vacuum-sterilized syringe. The collected blood was transferred immediately into tubes containing EDTA, gently mixed to avoid breaking blood corpuscles, and stored in a freezer at –70°C until analysis. To extract genomic DNA from monocytes, a blood SV kit (GeneAll Biotechnology, Seoul, Korea) was used. The concentration of the extracted DNA was measured using a spectrophotometer, and the A260/A280 absorbance ratio was calculated. For samples that did not reach the threshold of 1.8–2.0 DNA purity, DNA extraction was repeated. Polymerase chain reaction (PCR) was conducted according to the method described by Takahashi et al. (1994). Briefly, 20 μL of DNA, 28 μL of distilled water, 1 μL of forward primer (50 pmole·μL^{-1}), and 1 μL of reverse primer (50 pmole·μL^{-1}) were mixed with an α-Taq premix (GeneAll Biotechnology). The base sequences of the primers were as follows: forward, 5′-CCTTGGCCAGTGAGATGAG-3′ and reverse, 5′-

CTTGAAGGTTCTCTTGGAGT-3′. We used Multigene and performed 1 cycle for 5 min at 95°C (duration); after that, we conducted 30 cycles of 40 s at 94°C, 2 min at 55°C, 3 min at 72°C (annealing step), and, finally, left it for 1 min at 72°C (final extension) before we stopped the reaction at 4°C. To 10 µL of reaction solution, we added 7 µL of distilled water, 2 µL of 10× M Buffer, and 1 µL of restriction enzyme *Hae*III, and allowed the mixture to develop for 3 h at 37°C. After incubation, 10 µL of reaction solution was loaded onto a 3% agarose gel containing red safelight–emitting solution, and electrophoresis was performed for 70–80 min at 50 V. The PCR products were visualized using a UV transilluminator.

Resistance training program

Because the lower extremities are used daily for walking and supporting the body (Rose and Gamble, 2006), this study implemented exercises that involved only the upper extremities to compare changes in the mobilization of motor units and muscle strength. Subjects participated in resistance training, which was supervised by experienced physical education instructors, for a period of 8 weeks; the training was conducted 3 times a week, on alternate days (Monday, Wednesday, and Friday), at the same time (2 h 30 min after lunch). The 70-min program consisted of warm-up exercises (10 min), resistance exercises (50 min), and cool-down exercises (10 min). Warm-up and cool-down exercises included dynamic and static stretching. After measuring 1 repetition maximum (RM) for each subject according to American College of Sports Medicine (ACSM, 2010) guidelines (Berger, 1962), resistance training was performed at 75%–85% intensity of individual RM in the form of 3 sets of 8–12 repetitions each. The rest time between sets was limited to 3 min (Westcott, 1982). After every 2 weeks of resistance training, 1 RM was measured again, and intensity was re-established. Upper body resistance training involved 8 exercises that included shoulder press, bench press, lat pull-down, arm curl, hammer curl, triceps extension, dips, and crunches.

Surface-detected motor unit potential

An isokinetic device using surface electromyogram sensors attached to the biceps brachii (BB) and brachioradialis muscles was used to measure opposing muscle strength for each angular velocity (°/s) of the elbow joint. After subjecting the raw data to filtering (recursive digital filter, MATLAB elliptic filter; MyoResearch v4.0; Noraxon USA, Inc., Scottsdale, AZ, USA; 350-Hz low pass, 10-Hz high pass) and full-wave rectification using a surface electromyogram analysis program (MyoResearch v4.0), smoothing and analysis were performed.

Isometric strength testing

We measured isokinetic muscle function of the elbow joint using Cybex (Humac Norm; Computer Sports Medicine Inc., Stoughton, MA, USA). We used only the axial joint and examined the skeletal muscle before measuring its activity during simple stretching and warm-up to avoid fatigue after the examination. The chair was located at a 90° angle, the slope at the back of the chair was 180°, and the dynamometer was positioned in front of the face. To obtain maximum activity of the elbow joint, the other upper extremity was fastened to the chair using a connected belt. After gripping the handle, the lateral humeral condyle was aligned to the axis of the dynamometer. We entered the angles after maximum extension and after bending at 140°, and selected the appropriate working range of the joint for each subject. To exclude the influence of the force of gravity, we measured and compensated for gravity effect torque. To measure muscle strength and endurance, 3 rounds of exercises at 60°/s and 26 rounds of exercises at 180°/s were conducted. To regulate intensity before the actual exercise, subjects were asked to perform 3 rounds of practice exercise at the actual exercise speed. To collect exact data, we verbally encouraged the subjects as moral support. For muscle strength, the maximum muscle strength of each weight group was recorded in Nm units, whereas endurance was recorded as the average power in Watts for each weight group.

Statistical analysis

SPSS version 20.0 for Windows (IBM Corporation, Armonk, NY, USA) was used for statistical analysis. Mean and SD of all results was calculated, and, to confirm differences in body composition based on gene polymorphism, an independent *t*-test was conducted. To verify the effects of 8 weeks of resistance training on body composition and muscle function factors based on CNTF polymorphism in each 2-week period, two-way analysis of variance (ANOVA) with repeated measurements was conducted. For post-hoc testing, we used the Tukey test. The level of statistical significance for all results was set at $\alpha = 0.05$.

Results

Body composition

Changes in body composition after 8 weeks of resistance training are presented in Table 2. Before resistance training, baseline body composition values showed no differences between genotype groups ($p > 0.05$). After 8 weeks of resistance training, weight and BMI showed no significant changes based on time, group, or the interaction between time and group ($p > 0.05$). Post-hoc analysis also showed no significant difference in exercise duration ($p > 0.05$).

Muscle mass changed with time and demonstrated significant growth after 8 weeks of resistance training ($p < 0.001$). However, the effect was not different based on the duration of exercise, group, or the combined interaction between duration and group, and no difference based on genotype was observed ($p > 0.05$). Post-hoc analysis showed a significant increase in muscle mass compared with that before resistance training at week 2 ($p < 0.01$), week 4 ($p < 0.001$), week 6 ($p < 0.001$), and week 8 ($p < 0.001$). There was a significant increase from week 2 to week 4 ($p < 0.05$), week 6 ($p < 0.001$), and week 8 ($p < 0.01$). There was also a significant increase from week 4 to week 6 and week 8 (both $p < 0.001$).

Table 2. Changes in body composition after 8 weeks of resistance exercise. Data are means (±SD).

Variables	Groups	Pre	Week 2	Week 4	Week 6	Week 8	Posthoc
Weight, kg	GG	73.68 (8.19)	73.83 (8.63)	74.10 (8.34)	73.54 (8.29)	74.15 (7.65)	NS
	GA/AA	72.25 (9.65)	72.86 (9.70)	72.42 (9.32)	72.94 (9.85)	73.34 (9.67)	
	F	T 1.093 G .066 T×G 2.048					
BMI, kg/m²	GG	23.99 (1.76)	24.00 (1.87)	24.11 (1.83)	23.91 (1.85)	24.11 (1.69)	NS
	GA/AA	23.39 (2.62)	23.53 (2.43)	23.35 (2.31)	23.53 (2.45)	23.62 (2.34)	
	F	T .971 G .282 T×G 1.550					
Muscle mass, kg	GG	58.46 (5.59)	58.80 (6.94)	59.98 (6.59)	61.36 (6.69)	61.51 (7.18)	0<2<4<6,8
	GA/AA	56.11 (6.41)	57.88 (7.25)	58.39 (6.35)	60.36 (6.61)	60.57 (7.77)	
	F	T 34.461 * G .183 T×G 2.195					
Fat-free mass, kg	GG	62.00 (6.26)	62.44 (7.35)	63.84 (6.98)	64.76 (7.50)	64.66 (7.58)	0<2<4<6 0,2<8
	GA/AA	60.04 (7.22)	61.38 (7.63)	61.99 (7.52)	63.88 (7.21)	63.84 (8.11)	
	F	T 19.649 * G .162 T×G .668					
Body fat, %	GG	15.15 (2.93)	15.38 (2.97)	14.33 (2.51)	13.29 (2.19)	13.28 (1.90)	0,2>4>6 0,2>8
	GA/AA	16.27 (3.57)	15.78 (2.73)	14.89 (3.19)	14.16 (3.63)	14.13 (3.34)	
	F	T 12.531* G .266 T×G .337					

* p < 0.001. Abbreviations: 0, pre; 2, week 2; 4, week 4; 6, week 6; 8, week 8; BMI, body mass index; G, group; NS, not significant; T, time.

Fat-free mass also changed over time and improved significantly after 8 weeks of resistance training ($p < 0.001$). However, no interaction effect between duration and group was observed, and no significant difference based on genotype was detected ($p > 0.05$). According to the results of post-hoc testing, significant improvements were observed at week 2 ($p < 0.05$), week 4 ($p < 0.001$), week 6 ($p < 0.001$), and week 8 ($p < 0.001$), compared with that before resistance training. There was a significant increase from week 2 to week 4 ($p < 0.01$), week 6 ($p < 0.001$), and week 8 ($p < 0.01$); there was also a significant increase from week 4 to week 6 ($p < 0.001$).

Body fat percentage also changed over time, showing a significant decrease after 8 weeks of resistance training ($p < 0.001$). However, no interaction between group and duration was detected, and therefore, no change related to genotype was observed ($p > 0.05$). Post-hoc analysis showed a significant decrease in body fat percentage compared with that before resistance training at week 4 ($p < 0.001$), week 6 ($p < 0.001$), and week 8 ($p < 0.05$). There was also a significant decrease at week 4 ($p < 0.01$), week 6 ($p < 0.001$), and week 8 ($p < 0.05$), compared with that at week 2, and a significant decrease at week 6 compared with that at week 4 ($p < 0.05$).

Surface-detected motor unit potential

Changes in surface-detected motor unit potential (SMUP)—an indicator of mobilization units of nerve roots (Stashuk, 1999)—after 8 weeks of resistance training are presented in Table 3. The SMUP of the biceps brachii muscle increased significantly over time at 60°/s ($p < 0.05$). However, no affect based on group, exercise duration, or the interaction between group and duration was observed, and therefore, no difference based on genotype was detected ($p > 0.05$). Post-hoc analysis showed a significant increase at week 2 ($p < 0.05$) and week 6 ($p < 0.05$), compared with that before resistance training. The SMUP of the brachioradialis muscle at 60°/s showed no significant change based on group, exercise duration, or the interaction between group and duration ($p > 0.05$). Post-hoc analysis showed a significant increase at week 2 compared with that before resistance training ($p < 0.05$), and a significant decrease at week 4 compared with that at week 2 ($p < 0.05$). At 180°/s, the SMUP of the biceps brachii demonstrated an interaction effect between group and duration of exercise ($p < 0.05$), but no effect based on group or duration was observed ($p > 0.05$). Post-hoc analysis showed a significant increase at week 2 compared with that before resistance training ($p < 0.05$), as well as a significant decrease at week 6 compared with that at week 2 ($p < 0.05$). The SMUP of the brachioradialis at 180°/s showed no change based on group, exercise duration,

Table 3. Changes in SMUP area after 8 weeks of resistance exercise. Data are means (±SD).

Variables		Groups	Pre	Week 2	Week 4	Week 6	Week 8	Posthoc
60°/sec	BB, μV *sec	GG	9.06 (2.50)	10.05 (2.56)	9.91 (2.70)	9.86 (3.62)	9.85 (2.52)	0<2,6
		GA/AA	7.92 (3.32)	9.95 (4.67)	9.08 (3.01)	10.79 (3.73)	7.80 (1.57)	
		F	T 5.685* G .361 T×G 1.891					
	BR, μV *sec	GG	5.90 (1.26)	6.42 (1.46)	6.03 (1.48)	6.41 (2.32)	6.40 (3.24)	0<2 2>4
		GA/AA	6.37 (1.73)	7.29 (1.79)	6.21 (1.61)	6.11 (1.29)	5.98 (1.09)	
		F	T 1.451 G .001 T×G .286					
180°/sec	BB, μV *sec	GG	39.51 (11.33)	41.40 (13.57)	35.31 (8.85)	32.66 (10.67)	39.46 (11.58)	0<2 2>6
		GA/AA	36.39 (13.62)	44.32 (15.58)	36.90 (9.98)	44.93 (11.66)	39.57 (16.47)	
		F	T 3.812 G .043 T×G 4.188 *					
	BR, μV *sec	GG	23.95 (5.99)	27.82 (10.43)	25.25 (7.57)	26.41 (7.91)	25.51 (15.24)	0<2
		GA/AA	22.43 (7.41)	26.55 (6.83)	24.29 (6.14)	21.75 (4.27)	23.66 (5.62)	
		F	T 1.266 G .378 T×G .211					

* p < 0.05. 0, pre; 2, week 2; 4, week 4; 6, week 6; 8, week 8; BB, biceps brachii; BR, brachioradialis; G, group; SMUP, surface-detected motor unit potential; T, time

Table 4. Changes in muscle strength and endurance after 8 weeks of resistance exercise. Data are means (±SD).

Variables	Groups	Pre	Week 2	Week 4	Week 6	Week 8	Posthoc
Weight, kg	GG	61.00 (6.48)	61.33 (7.00)	62.40 (7.50)	64.00 (7.07)	63.20 (5.45)	0<8
	GA/AA	64.14 (9.70)	65.14 (8.97)	61.86 (6.09)	67.29 (5.19)	71.71 (12.91)	4<6,8
	F	T 2.520 G .772 T×G 1.297					
BMI, kg/m²	GG	44.40 (4.10)	43.60 (4.93)	51.20 (4.66)	59.20 (6.30)	58.20 (7.26)	0<4,6,8
	GA/AA	47.57 (11.50)	54.71 (19.96)	58.43 (13.70)	63.43 (12.31)	65.14 (14.09)	2,4<6,8
	F	T 27.648 *G 1.218 T×G .587					

* $p < 0.001$. Abbreviations: 0, pre; 2, week 2; 4, week 4; 6, week 6; 8, week 8; EF, elbow flexor; G, group; T, time

or the interaction between group and duration ($p > 0.05$). Post-hoc analysis demonstrated a significant increase at week 2 compared with that before resistance training ($p < 0.01$).

Isometric strength testing

Changes in isometric strength following 8 weeks of resistance training are presented in Table 4. The maximum muscle strength of the elbow flexor at 60°/s showed no relationship to group, exercise duration, or the interaction between group and duration, and no correlation with genotype was observed ($p > 0.05$). However, the results of post-hoc testing showed a significant increase at week 8 compared with that before resistance training ($p < 0.05$), as well as a significant increase at week 6 ($p < 0.05$) and week 8 ($p < 0.05$) compared with that at week 4. The average power of the elbow flexor at 180°/s was influenced by exercise duration, and increased significantly after 8 weeks of resistance training ($p < 0.001$). Group and the interaction between group and duration showed no effect, and therefore, no change based on genotype was observed ($p > 0.05$). The results of post-hoc testing showed a significant increase at week 4 ($p < 0.001$), week 6 ($p < 0.001$), and week 8 ($p < 0.001$), compared with that before resistance training. Week 6 ($p < 0.001$) and week 8 ($p < 0.01$) also showed a significant increase compared with that at weeks 2 and 4.

Discussion

The results of this study involving CNTF genotypes demonstrated that body composition, muscle mass, and fat-free mass increased and body fat percentage decreased with resistance training in healthy male college students; however, these effects were independent of differences in the CNTF genotype. Resistance training is characterized by high-intensity movements that stimulate the secretion of growth hormones. When metabolized, these hormones undergo decomposition in the body and increase fat-free mass (Kraemer et al., 2001). In addition, the increased amount of growth hormone in the blood stimulates the release of fatty acids from neutral fats in adipose cells, which imparts a positive effect on lipid metabolism (Møller et al., 1990; Quisth et al., 2005). By week 2 of resistance training, body fat percentages started to decrease, with a significant difference observed at week 4. Resistance training uses muscle glycogen as the main source of energy (Singh et al., 1999), and because the subjects were asked to not change their diet, the decrease in body fat percentage occurred slower than the increases in muscle mass and fat-free mass. In addition, the maintenance of weight and BMI is attributable to the simultaneous increases in muscle mass and fat-free mass and the decrease in body fat percentage. Changes in body composition were not associated with any CNTF genotype, which is consistent with the findings of Walsh et al. (2009), who reported no significant difference in muscle cross-sectional area in relation to genotype after 12 weeks of resistance training. Therefore, the CNTF genotype does not influence changes in body composition factors such as muscle mass and fat-free mass. Although this study did not identify differences in body composition associated with CNTF genotype, we acknowledge that the use of bioelectrical impedance may have been a limitation. However, the measurement of body composition in association with CNTF genotype was not a primary aim of the study. Therefore, further studies evaluating the effect of resistance training on body composition associations with CNTF genotype using gold standards such as DEXA are required.

Changes in motor unit potential and muscle strength and endurance occurred after 8 weeks of resistance training, with the greatest increases observed at the beginning of the exercise program. Both 60°/s peak torque which was the value for muscle strength of the elbow flexor, and 180°/s average power which was the value for endurance, demonstrated peak values at weeks 6 and 8.

Physiological responses to resistance training occur predominantly in the nervous system, which plays an important role in the initial adaptation of muscles (Kraemer, 1988; McCall et al., 1999). The effects and adaptation of nerve roots may be responsible for the successful motorization of motor units, thereby providing more muscle power (Kraemer et al., 1993). The 90% increase in muscle strength during resistance training in the initial 2 weeks and the 40%–50% increase in the following 2 weeks are attributable to neural adaptation (Earle and Baechle, 2004). Even without structural changes in muscle, it is possible to increase muscle strength through neural adaptation (Enoka, 1988), with further increases in strength resulting predominantly from muscular hypertrophy (William et al., 2001). According to Staron et al. (1994), men who participated in 8 weeks of resistance training showed a significant increase in 1 RM, which serves as an indicator of muscle strength, with the largest increase observed 2 weeks after the initiation of training. In the study by Wilmore et al. (2007), the cross-sectional area of muscle tissue did not increase significantly after resistance training, and the increase in muscle strength may be attributable to improvement in nerve adaptation. In this study, which involved 60°/s and 180°/s isokinetic

exercises controlled by a dynamometer, the biceps brachii muscle demonstrated a higher motor unit potential at weeks 2 and 6, whereas the brachioradialis muscle showed the highest potential at week 2. Accordingly, the highest level of nerve adaptation after resistance training was observed at week 2.

Conwit et al. (1999) reported that greater muscle strength during isokinetic knee extension is associated with higher mobilization of motor units. The highest peak torque, which represents maximum muscle strength, was observed during isokinetic 60°/s exercises for 8 weeks, whereas motor unit potential decreased from week 2 to week 6 and from week 4 to week 8, despite the increase in muscle strength. These changes may be attributable to the increase in load after 2 weeks of resistance training; nerve adaptation occurs by week 2, and at week 4, a few motor units exhibit high muscle strength. Nerve adaptation, which is for further increasing exercise load, increases motor unit potential, as supported by measurements of maximum muscle strength at week 6.

The 180°/s exercises, which are for endurance motor unit potential, were the highest at week 2, and the average power steadily increased from week 4 to week 8. During the 180°/s exercises, despite the increase in average power, motor unit potential decreased after week 2, because even if extensive exercise is performed for a long period of time, only a few motor units are mobilized, and their effectiveness increases. Previous research also demonstrated that resistance training increases the capacity of muscle tissue to store energy and increases the secretion of neurotransmitters; in addition, the smoothing of neural facilitation results in an increase in muscle endurance (Fournier et al., 1982; Sale, 1988). The results of this study are consistent with these previous findings.

CNTF is a neurotrophic agent that supports the existence and division of various kinds of neural cells that are part of motor nerves (Sendtner et al., 1994). It has been reported that the CNTF genotype is associated with functional differences in the motorization of nerve roots as well as muscle strength and endurance (Takahashi et al., 1994). However, this study showed that increases in muscle strength and endurance after resistance training were not associated with the CNTF genotype; however, difference in motor unit potential of the biceps brachii muscle based on the CNTF genotype was observed during isokinetic 180°/s exercises.

Roth et al. (2001) reported that women with the GA genotype exhibited more muscle strength in untrained arms at low muscle strength units (3.14 rads/s), but no difference was associated with the CNTF genotype in arms at high muscle strength units (0.52 rads/s). On the other hand, both men and women with trained arms demonstrated no differences in the effects of resistance training based on the CNTF genotype. In this study, peak torque in isokinetic 60°/s exercises was higher in those with the GA or AA genotype than in those with the GG genotype; however, this difference was not significant. Average power in isokinetic 180°/s exercises was also higher in those with the GA or AA genotype than in subjects with the GG genotype; however, this value was also not statistically significant. Guillet et al. (1999) suggested that women with the A allele have lower levels of functional CNTF protein than women with the GG genotype, and hence, the CNTF receptors in muscles have lower activity. Accordingly, in untrained arms, the neurotrophic and myotrophic effects of CNTF were lower, and there was a difference in the extent of increase in muscle strength (Roth et al., 2001).

These neurotrophic and myotrophic roles of CNTF do not present themselves in trained arms. In particular, men showed no differences in terms of the adaptation of muscle characteristics after resistance exercise in both trained and untrained arms (Roth et al., 2001). This result is consistent with the study of Guillet et al. (1999), in which female rats received CNTF treatment; older rats exhibited growth of muscle fiber area, whereas younger rats showed no change in muscle fiber size. CNTF prevents the degeneration of motor nerves (Sendtner et al., 1990; Sendtner et al., 1997); thus, 24-month-old rats responded to CNTF treatment because levels of CNTF decrease with aging, but there was no such effect in 6-month-old rats. Accordingly, the neurotrophic and myotrophic effects of CNTF can be influenced by age or the need for restoring function, and the CNTF genotype may regulate muscle strength and motor unit function in aging; however, they have no influence in young and healthy individuals. Thus, resistance training results in good muscle adaptation capacity.

Conwit et al. (2005) reported that the mobilization of motor units based on muscle strength showed differences in the size of motor units and potential patterns according to the CNTF genotype. It was reported that when subjects with the GA genotype exhibited high muscle strength, motor unit mobilization per level of muscle strength was small, indicating that motor unit function was effective. When performing 60°/s isokinetic exercises in this study, no differences were associated with motor unit potential and the CNTF genotype. On the other hand, when performing 180°/s isokinetic exercises in the biceps brachii muscle, those with the GA or AA genotype had a higher motor unit potential than those with the GG genotype, which is different from the results of a previous study. The discrepancy may be because this study measured changes in muscle strength and endurance following resistance training; the resistance load steadily increased during the training period, resulting in nerve root adaptation and an increase in motor unit potential. If the motor unit potential was measured strictly at the same muscle strength units during training, we believe that it could provide information on the effective functioning of motor units based on the development of nerve adaptation and the mobilization of fewer motor units. Accordingly, during 180°/s isokinetic exercises in the biceps brachii muscle, subjects with the GA or AA genotype exhibited higher motor unit potential than those with the GG genotype; however, this difference was not significant and can be attributed to the fact that this group also exhibited higher average power during resistance training.

In summary, muscle strength factors and motor unit potential improved after 8 weeks of resistance training, but these changes were not associated with CNTF polymorphism. One reason for this could be the small

number of subjects included in the study. Another reason may be the influence of functioning of leukemic inhibitory factor (LIF). The structure and function of LIF are similar to those of CNTF (Haas et al., 1999), and according to the results of in vitro experiments and animal experiments, it is, along with CNTF, a necessary protein for the existence and functioning of motor nerves (Arakawa et al., 1990; Banner and Patterson, 1994; Hughes et al., 1993; Sendtner et al., 1990; Pennica et al., 1996), and plays a major role in motor endplates (Holtmann et al., 2005). Accordingly, even if the level of CNTF is low, LIF, which supplements CNTF, is activated and can have a similar level of adaptation and development of functioning as the nervous system. On the other hand, when performing 180°/s exercises, the motor unit potential of the biceps brachii muscle was related with the interaction between duration of exercise and group after 8 weeks of resistance training. This result is difficult to generalize because it was not observed with any other variable; however, it can be assumed that in the variant group that lacks CNTF, substances such as LIF and similar proteins perform a supplementary function. The other reason may be the influence of motor-related genes. The angiotensin-converting enzyme gene is related to physical strength, and many studies have reported that polymorphisms of this gene are associated with differences in muscle strength, rapidity, and cardiopulmonary endurance (Charbonneau et al., 2008; Giaccaglia et al., 2008; Pescatello et al., 2006; Sahlen et al., 2010; Thompson et al., 2006; Williams et al., 2005). The α-actinin-3 gene also has been suggested to influence muscle strength and rapidity, and plays an important role in the arrangement and functioning of myofibrils (Macarthur and North, 2005). It has been reported that increases and differences in muscle function are associated with polymorphisms of this gene (Zanoteli et al., 2003). Apart from that, there is also the influence of numerous motor-related genes (Rankinen et al., 2004). The same results involving CNTF polymorphism were difficult to verify in this study, and therefore, there is a need to further study CNTF along with related substances and motor-related genes. Finally, we did not measure the difference in CNTF levels before and after resistance training. Guillet et al. (1999) reported that CNTF level was associated with sports performance and muscle strength in rats. Therefore, if the CNTF levels differ between CNTF genotypes after resistance training, it could affect body composition, muscle performance, and motor unit function. Thus, further studies on the effect of resistance training on CNTF level are required.

Conclusion

We conclude that 8 weeks of resistance training resulted in improvements in motor unit potential and muscle strength and endurance, and no differences were associated with the CNTF genotype, except for the biceps brachii muscle during 180°/s exercises. Therefore, improvements in muscle strength and endurance after resistance training in healthy male college students are a direct result of the training program and are not related to a genetic factor involving motor nerves.

Acknowledgements

The authors are grateful to the students who participated in the experiment and Professor Jae Ho Kim who assisted in the experiment.

References

Arakawa, Y., Sendtner, M. and Thoenen, H. (1990) Survival effect of ciliary neurotrophic factor (CNTF) on chick embryonic motoneuron in culture: comparison with other neurotrophic factors and cytokines. *Journal of Neuroscience* **10**, 3507-3515.

Arking, D.E., Fallin, D.M., Fried, L.P., Li, T., Beamer, B.A., Xue, Q.L., Chakravarti, A. and Walston, J. (2006) Variation in the ciliary neurotrophic factor gene and muscle strength in older Caucasian women. *Journal of the American Geriatrics Society* **54**, 823-826.

Bandy, W.D., Lovelace-Chandler, V. and McKitrick-Brandy, B. (1990) Adaptation of skeletal muscle to resistance training. *Journal of Orthopaedic and Sports Physical Therapy* **12**, 248-255.

Banner, L.R. and Patterson, P.H. (1994) Major changes in the expression of the mRNAs for cholinergic differentiation factor/leukemia inhibitory factor and its receptor after injury to adult peripheral nerves and ganglia. *Proceedings of the National Academy of Sciences of the United States of America* **91**, 7109-7113.

Behringer, M., Vom Heed, A., Yue, Z. and Mester, J. (2010) Effects of resistance training in children and adolescents: a meta-analysis. *Pediatrics* **126**, 1199-1210.

Berger, R.A. (1962) Effect of varied weight training programs on strength. *Research Quarterly. American Association for Health, Physical Education and Recreation* **33**, 168-181.

Buller, A.J., Kean, C.J. and Ranatunga, K.W. (1987) Transformation of contraction speed in muscle following cross-reinnervation: dependence on muscle size. *Journal of Muscle Research and Cell Motility* **8**, 504-516.

Charbonneau, D.E., Hanson, E.D., Ludlow, A.T., Delmonico, M.J., Hurley, B.F. and Roth, S.M. (2008) ACE genotype and the muscle hypertrophic and strength responses to strength training. *Medicine and Science in Sports and Exercise* **40**, 677-683.

Chestnut, J.L. and Docherty, D. (1999) The effects of 4 and 10 repetition maximum weight-training protocols on neuromuscular adaptations in untrained men. *Journal of Strength and Conditioning Research* **13**, 353-359.

Connor, B. and Dragunow, M. (1998) The role of neuronal growth factors in neurodegenerative disorders of the human brain. *Brain Research Reviews* **27**, 1-39.

Conwit, R.A., Ling, S., Roth, S., Stashuk, D., Hurley, B., Ferrell, R. and Metter, E.J. (2005) The relationship between ciliary neurotrophic factor (CNTF) genotype and motor unit physiology: preliminary studies. *BMC Physiology* **5**, 15.

Conwit, R.A., Stashuk, D., Tracy, B., McHugh, M., Brown, W.F. and Metter, E.J. (1999) The relationship of motor unit size, firing rate and force. *Clinical Neurophysiology* **110**, 1270-1275.

De Mars, G., Windelinckx, A., Beunen, G., Delecluse, C., Lefevre, J. and Thomis, M.A. (2007) Polymorphisms in the CNTF and CNTF receptor genes are associated with muscle strength in men and women. *Journal of Applied Physiology* **102**, 1824-1831.

DiStefano, P.S., Boulton, T.G., Stark, J.L., Zhu, Y., Adryan, K.M., Ryan, T.E. and Lindsay, R.M. (1996) Ciliary neurotrophic factor induces down-regulation of its receptor and desensitization of signal transduction pathways in vivo: non-equivalence with pharmacological activity. *Journal of Biological Chemistry* **271**, 22839-22846.

Earle, R.W. and Baechle, T.R. (2004) *NSCA's essentials of personal training*. Human Kinetics, Champaign.

Enoka, R.M. (1988) *Neuromechanical basis of kinesiology*. Human Kinetics, Champaign.

Forger, N.G., Roberts, S.L., Wong, V. and Breedlove, S.M. (1993) Ciliary neurotrophic factor maintains motoneurons and their target muscles in developing rats. *Journal of Neuroscience* **13**, 4720-4726.

Fournier, M., Ricci, J., Taylor, A.W., Ferguson, R.J., Montpetit, R.R. and Chaitman, B.R. (1982) Skeletal muscle adaptation in adolescent boys: sprint and endurance training and detraining. *Medicine and Science in Sports and Exercise* **14**, 453-456.

Fraysse, B., Guillet, C., Huchet-Cadiou, C., Camerino, D.C., Gascan, H. and Leoty, C. (2000) Ciliary neurotrophic factor prevents unweighting-induced functional changes in rat soleus muscle.

Journal of Applied Physiology **88**, 1623-1630.

Giaccaglia, V., Nicklas, B., Kritchevsky, S., Mychalecky, J., Messier, S., Bleecker, E. and Pahor, M. (2008) Interaction between angiotensin converting enzyme insertion/deletion genotype and exercise training on knee extensor strength in older individuals. *International Journal of Sports Medicine* **29**, 40-44.

Guillet, C., Auguste, P., Mavo, W., Kreher, P. and Gascan, H. (1999) Ciliary neurotrophic factor is a regulator of muscular strength in aging. Journal of Neuroscience **19**, 1257-1262.

Haas, C.A., Hofmann, H.D. and Kirsch, M. (1999) Expression of CNTF/LIF-receptor components and activation of STAT3 signaling in axotomized facial motoneurons: evidence for a sequential postlesional function of the cytokines. *Journal of Neurobiology* **41**, 559-571.

Hakkinen, K. (1989) Neuromuscular and hormonal adaptations during strength and power training. A review. *Journal of Sports Medicine and Physical Fitness* **29**, 9-26.

Helgren, M.E., Squinto, S.P., David, H.L., Parry, D.J., Boulton, T.G., Heck, S.C., Zhu, Y., Yancopoulos, G.D., Lindsay, R.M. and DiStefano, P.S. (1994) Trophic effect of ciliary neurotrophic factor on denervated skeletal muscle. *Cell* **76**, 493-504.

Henneman, E., Somjen, G. and Carpenter, D.O. (1965) Functional significance of cell size in spinal motoneurons. *Journal of Neurophysiology* **28**, 560-580.

Holtmann, B., Wiese, S., Samsam, M., Grohmann, K., Pennica, D., Martini, R. and Sendtner, M. (2005) Triple knock-out of CNTF, LIF, and CT-1 defines cooperative and distinct roles of these neurotrophic factors for motoneuron maintenance and function. *Journal of Neuroscience* **25,** 1778-1787.

Hughes, R.A., Sendtner, M. and Thoenen, H. (1993) Members of several gene families influence survival of rat motoneurons in vitro and in vivo. *Journal of Neuroscience Research* **36**, 663-671.

Ip, N.Y., McClain, J., Barrezueta, N.X., Aldrich, T.H., Pan, L., Li, Y., Wiegand, S.J., Friedman, B., Davis, S. and Yancopoulos, G.D. (1993) The alpha component of the CNTF receptor is required for signaling and defines potential CNTF targets in the adult and during development. *Neuron* **10**, 89-102.

Johnston, A.P., De Lisio, M. and Parise, G. (2008) Resistance training, sarcopenia, and the mitochondrial theory of aging. *Applied Physiology Nutrition and Metabolism* **33,** 191-199.

Klausen, K., Andersen, L.B. and Pelle, I. (1981) Adaptive changes in work capacity, skeletal muscle capillarization and enzyme levels during training and detraining. *Acta Physiologica Scandinavica* **113**, 9-16.

Kraemer, W.J. (1988) Endocrine responses to resistance exercise. *Medicine and Science in Sports and Exercise* **20**, S152-S157.

Kraemer, W.J., Deschenes, M.R. and Fleck, S.J. (1988) Physiological adaptations to resistance exercise. Implications for athletic conditioning. *Sports Medicine* **6**, 246-256.

Kraemer, W.J., Dudley, G.A., Tesch, P.A., Gordon, S.E., Hather, B.M., Volek, J.S. and Ratamess, N.A. (2001) The influence of muscle action on the acute growth hormone response to resistance exercise and short-term detraining. *Growth Hormone and IGF Research* **11**, 75-83.

Kraemer, W.J., Fleck, S.J., Dziados, J.E., Harman, E.A., Marchitelli, L.J., Gordon, S.E., Mello, R., Frykman, P.N., Koziris, L.P. and Triplett, N.T. (1993) Changes in hormonal concentrations after different heavy-resistance exercise protocols in women. *Journal of Applied Physiology* **75***,* 594-604.

Lewin, G.R. and Barde, Y.A. (1996) Physiology of the neurotrophins. *Annual Review of Neuroscience* **19**, 289-317.

Macarthur, D.G. and North, K.N. (2005) Genes and human elite athletic performance. *Human Genetics* **116**, 331-339.

McCall, G.E., Byrnes, W.C., Fleck, S.J., Dickinson, A. and Kraemer, W.J. (1999) Acute and chronic hormonal responses to resistance training designed to promote muscle hypertrophy. *Canadian Journal of Applied Physiology* **24**, 96-107.

Møller, N., Jørgensen, J.O., Alberti, K.G., Flyvbjerg, A. and Schmitz, O. (1990) Short-term effects of growth hormone on fuel oxidation and regional substrate metabolism in normal man. *Journal of Clinical Endocrinology and Metabolism* **70**, 1179-1186.

Moore, D.R., Burqomaster, K.A., Schofield, L.M., Gibala, M.J., Sale, D.G. and Phillips, S.M. (2004) Neuromuscular adaptations in human muscle following low intensity resistance training with vascular occlusion. *European Journal of Applied Physiology* **92**, 399-406.

Mujika, I. and Padilla, S. (2001) Muscular characteristics of detraining in humans. *Medicine and Science in Sports and Exercise* **33**, 1297-1303.

Oppenheim, R.W., Prevette, D., Yin, Q.W., Collins, F. and MacDonald, J. (1991) Control of embryonic motoneuron survival in vivo by ciliary neurotrophic factor. *Science* **251**, 1616-1618.

Pennica, D., Arce, V., Swanson, T.A., Vejsada, R., Pollock, R.A., Armanini, M., Dudley, K., Phillips, H.S., Rosenthal, A., Kato, A.C. and Henderson, C.E. (1996) Cardiotrophin-1, a cytokine present in embryonic muscle, supports long-term survival of spinal motoneurons. *Neuron* **17**, 63-74.

Pescatello, L.S., Kostek, M.A., Gordish-Dressman, H., Thompson, P.D., Seip, R.L., Price, T.B., Angelopoulos, T.J., Clarkson, P.M., Gordon, P.M., Moyna, N.M., Visich, P.S., Zoeller, R.F., Devaney, J.M. and Hoffman, E.P. (2006) ACE ID genotype and the muscle strength and size response to unilateral resistance training. *Medicine and Science in Sports and Exercise* **38**, 1074-1081.

Quisth, V., Enoksson, S., Blaak, E., Hagstrom-Toft, E., Arner, P. and Bolinder, J. (2005) Major differences in noradrenaline action on lipolysis and blood flow rates in skeletal muscle and adipose tissue in vivo. *Diabetologia* **48**, 946-953.

Rankinen T, Perusse L, Rauramaa R, Rivera MA, Wolfarth B, Bouchard C. (2004) The human gene map for performance and health-related fitness phenotypes: the 2003 update. *Medicine in Science and Sports Exercise* **36**, 1451-1469.

Rose, J. and Gamble, J.G. (2006) *Human walking.* 3rd edition. Lippincott Williams & Wilkins, Philadelphia.

Rose, S.J. and Rothstein, J.M. (1982) Muscle mutability. Part 1. General concepts and adaptations to altered patterns of use. *Physical Therapy* **62**, 1773-1787.

Roth, S.M., Schrager, M.A., Ferrell, R.E., Riechman, S.E., Metter, E.J., Lynch, N.A., Lindle, R.S. and Hurley, B.F. (2001) CNTF genotype is associated with muscular strength and quality in humans across the adult age span. *Journal of Applied Physiology* **90**, 1205-1210.

Sahlen, A., Shahgaldi, K., Aminoff, A., Aagaard, P., Manouras, A., Winter, R., Ehrenborg, E. and Braunschweig, F. (2010) Effects of prolonged exercise on left ventricular mechanical synchrony in long-distance runners: importance of previous exposure to endurance races. *Journal of the American Society of Echocardiography* **23**, 977-984.

Sale, D.G. (1988) Neural adaptation to resistance training. *Medicine and Science in Sports and Exercise* **20**, S135-S145.

Sendtner, M., Carroll, P., Holtmann, B., Hughes, R.A. and Thoenen, H. (1994) Ciliary neurotrophic factor. *Journal of Neurobiology* **25**, 1436-1453.

Sendtner, M., Gotz, R., Holtmann, B. and Thoenen, H. (1997) Endogenous ciliary neurotrophic factor is a lesion factor for axotomized motoneurons in adult mice. *Journal of Neuroscience* **17**, 6999-7006.

Sendtner, M., Kreutzberg, G.M. and Thoenen, H. (1990) Ciliary neurotrophic factor prevents the degeneration of motor neurons after axotomy. *Nature* **345**, 440-441.

Sendtner, M., Schmalbruch, H., Stockli, K.A., Carroll, P., Kreutzberg, G.W. and Thoenen, H. (1992) Ciliary neurotrophic factor prevents degeneration of motor neurons in mouse mutant progressive motor neuronopathy. *Nature* **358**, 502-504.

Singh, M.A., Ding, W., Manfredi, T.J., Solares, G.S., O'Neill, E.F., Clements, K.M., Ryan, N.D., Kehayias, J.J., Fielding, R.A. and Evans, W.J. (1999) Insulin-like growth factor I in skeletal muscle after weight-lifting exercise in frail elders. *American Journal of Physiology - Endocrinology and Metabolism* **277**, E135-E143.

Staron, R.S., Karapondo, D.L., Kraemer, W.J., Fry, A.C., Gordon, S.E., Falkel, J.E., Hagerman, F.C. and Hikida, R.S. (1994) Skeletal muscle adaptations during early phase of heavy-resistance training in men and women. *Journal of Applied Physiology* **76**, 1247-1255.

Stashuk, D.W. (1999) Decomposition and quantitative analysis of clinical electromyographic signals. *Medical Engineering and Physics* **21**, 389-404.

Takahashi, R., Yokoji, H., Misawa, H., Hayashi, M., Hu, J. and Deguchi, T. (1994) A null mutation in the human CNTF gene is not causally related to neurological diseases. *Nature Genetics* **7**, 79-84.

Thompson, P.D., Tsongalis, G.J., Ordovas, J.M., Seip, R.L., Bilbie, C., Miles, M., Zoeller, R., Visich, P., Gordon, P., Angelopoulos, T.J., Pescatello, L. and Moyna, N. (2006) Angiotensin-converting enzyme genotype and adherence to aerobic exercise

training. *Preventive Cardiology* **9**, 21-24.
Walsh, S., Kelsey, B.K., Angelopoulos, T.J., Clarkson, P.M., Gordon, P.M., Moyna, N.M., Visich, P.S., Zoeller, R.F., Seip, R.L., Bilbie, S., Thompson, P.D., Hoffman, E.P., Price, T.B., Devaney, J.M. and Pescatello, L.S. (2009) CNTF 1357 G -> A polymorphism and the muscle strength response to resistance training. *Journal of Applied Physiology* **107**, 1235-1240.
Westcott, W.L. (1982) *Strength fitness: physiological principles and training techniques.* 1st edition. Allyn & Bacon, Boston.
William, D.M., Frank, I.K. and Victor, L.K. (2001) *Exercise physiology: energy, nutrition, and human performance.* 5th edition. Lippincott Williams & Wilkins, Baltimore.
Williams, A.G., Day, S.H., Folland, J.P., Gohlke, P., Dhamrait, S. and Montgomery, H.E. (2005) Circulating angiotensin converting enzyme activity is correlated with muscle strength. *Medicine and Science in Sports and Exercise* **37**, 944-948.
Wilmore, J.H., Costill, D.L. and Kenney, L.W. (2007) *Physiology of sport and exercise.* 4th edition. Human Kinetics, Champaign.
Wojtys, E.M., Huston, L.J., Taylor, P.D. and Bastian, S.D. (1996) Neuromuscular adaptations in isokinetic, isotonic, and agility training programs. *American Journal of Sports Medicine* **24**, 187-192.
Zanoteli, E., Lotuffo, R.M., Oliveira, A.S., Beggs, A.H., Canovas, M., Zatz, M. and Vainzof, M. (2003) Deficiency of muscle alpha-actinin-3 is compatible with high muscle performance. *Journal of Molecular Neuroscience* **20**, 39-42.

Key points

- Resistance training improves muscle strength and endurance in young men.
- This improvement in muscular strength and endurance is irrespective of CNTF genotypes.

AUTHORS BIOGRAPHY

Ae-Rim HONG
Employment
Department of Exercise Prescription & Rehabilitation, College of Sports Science, Dankook University, Republic of Korea
Degree
MSc
Research interests
SNP, muscle strength, resistance training, muscle activation
E-mail: aerim_926@naver.com

Sang-Min HONG
Employment
Adjunct Professor, Department of Physical Education, Dongguk University, Republic of Korea
Degree
PhD
Research interests
Exercise training, sports performance.
E-mail: rippey5070@naver.com

Yun-A SHIN
Employment
Professor, Department of Exercise Prescription & Rehabilitation, College of Sports Science, and Department of Kinesiologic Medical Science, Graduate School, Dankook University, Republic of Korea
Degree
PhD
Research interests
Obesity, aging, SNP, training, adipocytokines
E-mail: shinagel@dankook.ac.kr

✉ **Yun-A Shin**
Department of Exercise Prescription & Rehabilitation, College of Sports Science, and Department of Kinesiologic Medical Science, Graduate School, Dankook University, San 29, Anseo-dong, Cheonan-si, Chungnam 330-714, Republic of Korea