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ORIGINAL PAPER

Effects of detraining on motor unit potential area, muscle function and physical performance based on CNTF gene polymorphism

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Sang Min Hong, Ae Rim Hong and Yun A Shin. Effects of detraining on motor unit potential area, muscle function and physical performance based on CNTF gene polymorphism. *JENB*., Vol. 18, No. 2, pp.151-160, 2014 **[Purpose]** The purpose of this study was to identify the effect of detraining on motor unit potential area (SMUP), muscular function and physical performance, according to CNTF gene polymorphism. **[Methods]** For this study, GG (normal homozygote, n = 8) group and GA + AA (mutation heterozygote and homozygote, n = 10) group were divided by CNTF gene polymorphism and both groups were performed detraining for 4 weeks. The data was analyzed by two-way repeated measures ANOVA for verifying the differences between two groups and interaction using *SPSS* (ver. 20.0) statistical program. **[Results]** The results were as follows. First, changes in body composition were measured but there was no significant interaction effect between time and group. Seconds, changes in SMUP were measured by SEMG. Interaction effect between time and group was found lateral vastus during isokinetic exercise of 180°/sec ($p < .05$). Third, changes in isokinetic muscle strength of 60°/sec and 180°/sec were measured but there was no significant interaction effect. Fourth, significant statistical differences were not showed changes of sports performance after detraining. **[Conclusion]** In conclusion, there were no significantly differences between GG and GA + AA group after detraining, therefore, further study will be considered a matter in various its interventions such as serum levels of CNTF and changes in receptors and muscle fiber types. **[Keyword]** detraining, motor unit potential area, muscular function, physical fitness, CNTF gene polymorphism

INTRODUCTION

One of the most significant characteristics of skeletal muscle is its dynamic nature. skeletal muscle tissue is high in plasticity and thus it induces adaptation of muscle performance (such as muscle endurance) in a response to outside stress and also it shows change of functional characteristics of muscle and muscle tissue structure in an adaptive response to the activation of muscle-nerve system and hormone reaction [1,2]. Stress coming from regular exercises improves the function of skeletal muscle to resist stress and thus induces adaptation of skeletal muscle such as muscle hypertrophy and muscle strength increase [3,4].

Also when detraining occurs, its function decreases rapidly within a short period of time and it goes back to the state before the training, opposing to the adaptation phenomenon to the training [5]. Skeletal muscle-change per detraining occurs even after a short period of time such as 2 weeks and muscle size and strength decrease occur based on the change of muscle-nerve and hormones [6]. But one study [7] reported that hormone-change per detraining is actually increasing anabolic hormones such as growth hormone and testosterone during the period of rest in order to prevent catabolism of muscle and prevents the decrease of muscle tissue and muscle strength and thus hormones' effects on the decrease of muscle strength and size are uncertain.

On the other hand, it was reported that motor nerve function decrease per detraining decreases motor unit mobilization and coordination functions of skeletal muscle and thus induces muscle strength decrease even without the size change of

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muscle tissue [8]. Gondin *et al*. (2006) [9] reported that after 4 weeks of detraining, knee extensor's strength and muscle activity had significantly decreased and such was the result from the nerve change per detraining. Therefore, motor nerve shows rapid change even in a short period time based on detraining and it affects much on the change of the performance of muscle functions.

Such motor nerve has neurotrophic factor called CNTF (ciliary neurotrophic factor) and it combines with CNTFR (CNTF receptor), which is autoreceptor, and thus expresses in target tissues such as motor nerve and skeletal muscle [10,11] and therefore said to be helping the function of motor nerve. CNTF gene polymorphism (1357 G (GG) → A (GA/AA) single- nucleotide polymorphism) of the Japanese was reported for the first time by Takahashi *et al*. (1994) [12] and it stated that a mutation group with homozygous deficient allele (AA type) is relatively low in its function of CNTF compared to a normal group with homozygous insertion allele (GG type).

But after the publication of Tankahashi *et al*. (1994) [12], Arking *et al*. (2006) [13] and Roth *et al*. (2001) [14], on their studies to reveal the muscle strength difference per CNTF gene polymorphism, reported that a GA type mutation group had higher muscle strength compared to a GG type normal group. Also it was reported that the effect of gene polymorphism per resistance training showed the change that for female, a GG type normal group had more muscle strength increase after 12 weeks of resistance training compared to an AA type mutation group but for male, there was no difference per gene polymorphism [15].

Also from the study of comparing mobilization capacity of motor units per CNTF gene polymorphism, Conwit *et al*. (2005) [15] reported that a GA type mutation group uses smaller motor-units when exerting the low-level strength and thus had a better motor-function for using muscle strength compared to a GG type normal group and thus the results of existing studies showed difference outcomes in terms of the comparison of muscle strength per CNTF gene polymorphism and mobilization capacity of motor unit and the change per resistance training.

With their study on the period of detraining, So & Seo (2004) [17] reported that when 8 weeks of resistance training was applied to 15 healthy male college students, fat free mass at the second week of training was increased compared to the before-training period and body fat was significantly decreased compared to the before-training period, when checking the results such as the changes of body composition, growth hormone and IGF-1 (insulin-like growth factor-1) at the second week and the fourth week of detraining.

After that, at the fourth week of detraining, fat free mass was decreased compared to the second week although it was not significant and also body fat was increased although it was not significant as well. It was reported that such changes of fat free mass and body fat occurred because the effects of growth hormone per training lasted up to the second week of detraining but it would decrease as the period of detraining gets longer as shown with the anabolic hormone decrease at the fourth week and thus detraining period should not be longer than 2 weeks. Also, long term detraining such as longer than 8 weeks increases cortisol level compared to testosterone and thus induces a great decrease of muscle strength [18]. Therefore, it was suggested that the 4 weeks of time is proper in order to get an accurate measurement of muscle function-change per the change of nerve-related factors, excluding the effects of anabolic hormones such as growth hormone per detraining [19].

Therefore, this study was designed to study the effect of muscle-nerve on muscle function by using 20 healthy male college students, majoring in sports, practicing resistance training regularly (at least 2 times per week), and they were categorized into a normal group (GG type) and mutation groups (GA/AA type) based on CNTF gene polymorphism and then applied 4 weeks of detraining period and checked the differences of decreasing motor unit potential area among groups and the change of muscle function and physical performance.

METHODS

Study subject

83 healthy male college students in their 20's, without medical issue or a problem in musculoskeletal system, performing resistance training for at least 2 times per week for at least 6 months of period, were selected. After giving the explanation on goal and process of this study to the subjects, blood sample was taken and analysis was done to categorize CNTF gene polymorphism.

Ultimately, voluntary participants were selected and they were confirmed by categorizing them into a GG (normal homozygote) group (8 people) and GA/AA (mutated heterozygote/ mutated homozygote) groups (10 people).

The average period of majoring the weight training for the subjects was 2.5 ± 1.5 years and they did at least 2 times a weeks, 100 minutes per session of resistance training.

For resistance training for the students majoring in weight training, the weight of 70~100% 1RM was used for 3~5 sets

Table 1. The physical characteristics of subjects

Variables	Group		t
	GG (N = 8)	GA/AA (N = 10)	
Age (years)	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 percentage (%)	14.10 ± 2.29	14.53 ± 3.40	-.306
BMI (kg/m²)	23.79 ± 1.79	23.02 ± 2.39	.752

on main major muscle groups including upper limbs and lower limbs and abdominal muscle.

Table 1 is the physical characteristics of the subjects and there was no inter-groups difference from all the variables of physical characteristics.

Assessing tool and method

Body composition

For body composition, bioelectrical resistance analyzer (Inbody 7.0, Biospace, Korea) was used to assess weight, muscle mass, fat free mass and body fat (%) and body mass index (kg/m^2) was obtained using the weight measured with the square of height (m) and Inbody 7.0. All the subjects were instructed to have assessment with light clothing and medical history was asked and consent was obtained before the assessment.

Motor unit potential area

When measuring the relative maximum strength of knee joint per angular velocity (°/sec) using isokinetic equipment, surface electromyogram (SEMG) sensor was applied to knee extensor (rectus femoris, vastus medialis and vastus lateralis) and elbow flexor (biceps brachii muscle and brachioradialis muscle) and the signals from them were obtained at the same time.

Raw SEMG data from the experiment was filtered (with Recursive digital filter, Matlab Elliptic filter, 350 Hz low pass, 10 Hz high pass) using EMG analysis program (MyoResearch v4.0, NORAXON Co.) and then full wave rectification and smoothing was done and surface motor unit potential area (SUMP area) analysis was followed.

Also, the average score of 3 sets of each exercise was calculated and the score obtained with integrated EMG (Root mean square, RMS) from EMG waveforms was standardized with MVIC value and then calculated by %MVIC [20].

Isokinetic muscle function

Isokinetic muscle function of knee joint and elbow joint was measured with Cybex (Humac norm, USA).

Only pivot joint was measured and also simple stretching and warm-up for 10 minutes were provided before the assessment to prevent injury.

When assessing the muscle function of knee joint, the position of a chair was set at 90°, the back-pad of the chair was set at 85° and a measuring device was set at 90° and also the body was fixated with a belt, attached to the chair to prevent other body parts' involvement and the outer phase of the femur, which is the axis of rotation of knee, was located on the same position with the axis of the measuring device.

Individualized range of motion for the subjects were set by inputting the angles after maximum extension and 90° flexion and GET (gravity effect torque) was measured and readjusted to eliminate the effect of gravity.

At the starting point of flexion, 3 times of 60°/sec exercises were done to assess muscle strength and 26 times of 180°/sec exercises were done to assess muscle endurance and 3 sets of practices were done to be ready for the intensity of actual exercise. Shouting was provided to increase the motivation of the subjects.

Physical performance

Reaction speed

This was assessed as an indicator of motor nerve and the subjects were instructed to stand with slightly flexed knees on a mattress with reaction speed sensor and to watch the light reaction of the machine, from 1~1.5 m distance, and jump immediately as soon as the light comes on.

The time from the point of coming of light to the point of the detachment of feet of the subjects from the mattress was recorded by second.

A total of 3 measurements was done and the average value was used.

Vertical jump

This was assessed as an indicator of reflex response and we had the subjects standing on the jump-mattress with a measuring device attached to their waist and had them maximal vertical jump using bounce.

The recording was done by cm and unnecessary actions potentially affecting the results were restricted. The assessments were done 2 times and the higher score was used.

Side step

This was assessed as an indicator of agility and with a

central sensor as the center-point, 2 sensors were installed to the right and the left, 120 cm distance apart from the center.

The subjects did side-steps for 30 seconds in front of the central sensor while lowering the center of body weight by lowering the waist and knees.

The number of occasion of feet passing each sensor was recorded and it was done once.

50m running

This was assessed as an indicator of speed and sensors were installed at a starting line and a finishing line and the subjects initiated running voluntarily as soon as they are ready at the starting point.

The time taken to reach the 50m finishing line was recorded by seconds and it was done once.

Gene polymorphism analysis

We had the subjects refraining from eating for 12 hours to restrict the factors affecting blood components and after having them 30 minutes of rest, 3㎖ of blood was obtained by inserting a disposable sterile syringe into median forearm vein.

Obtained blood was moved to a tube treated with EDTA and carefully mixed not to break hemocyte and then it was stored in a -70℃ freezer. Blood SV kit of GeneAll Biotechnology (Korea) was used to extract Genomic DNA of monocyte. Spectrophotometer A260/A280 density was assessed for the extracted DNA and if the sample is below the standard of 1.8~2.0, then DNA was extracted again and the density was checked. Polymerase chain reaction was assessed by the method used by Takahashi *et al*. (1994) [12]. DNA 20 μl, distilled water 28 μl, Forward primer (50 pmole/μl) 1μl and Reverse primer (50 pmole/μl) 1 μl were put into α-Taq premix from GeneAll Biotechnology (Korea) and mixed together. Primer sequences was Forward 5'-CCTT GGCCAGTG AGATGAG-3', Reverse 5'- CTTGAAGGTTC TC TTGGAGT-3'.

1 cycle was done for 5 minutes of duration at 95℃ using Multigene and then 30-cycles were done at 94℃ (40 seconds), 55℃ (2 minutes) and 72℃ (3 minutes) (annealing step) and then finally, after leaving it for 1 minutes at 72℃ (final extension), we put a stop on the reaction at 4℃. Obtained reaction solution of 10μl was treated with distilled water 7 μl, 10 × M Buffer 2 μl and restriction enzyme Hae Ⅲ 1 μl and cultured at 37℃ for 3 hours. After that, 10 μl of obtained solution from the process was added to 3% Agarose gel with red safe emitting solution and electrophoresis was done for 70~80 minutes with 50 V and then it was observed with ultraviolet (UV) projector. CNTF gene allele frequency of this study was categorized into GG 65 people (78.3%), GA 17 people (20.5%), AA 1 people (1.2%) from a total of 83 people and analysis showed that Hardy-Weinberg equilibrium was maintained ($\chi^2 = 0.009$, $df = 1$, $p = .996$).

CNTF gene polymorphism analysis for Korean people [16] showed that genotype frequencies were, from a total of 187 people (100.0%), GG 138 people (73.8%), GA 47 people (25.1%), AA 2 people (1.1%) (in a sequence of GG > GA > AA) and also our study's genotype frequencies were, from a total of 83 people (100.0%), GG 65 people (78.3%), GA 17 people (20.5%), AA 1 people (1.2%) (in a sequence of GG > GA > AA) and thus showed the same result with each other.

Detraining

We had our study subjects on detraining for 4 weeks after gene polymorphism analysis. For the duration of 4 weeks of detraining, resistance training and daily activities of medium-to-high level intensity were restricted by using daily phone call asking for activity patterns and exercises and other than that, normal daily activities were allowed to continue.

Regarding health supplements, the supplements related to muscle formation such as protein powder or steroids were restricted for 4 weeks by using phone call asking for their use and drinking pattern and other than that, normal diet was allowed to continue.

Data processing method

Window SPSS Ver. 20.0 program was used for the data processing of this study. Mean and standard deviation were calculated from the results of measurement items and independent t-test was done to confirm the previous difference of body composition per gene polymorphism.

Two-way ANOVA with repeated measures was done to confirm the effects of 4 weeks of detraining on body composition, motor unit potential area, muscle function and physical performance variables based on CNTF gene polymorphism and the statistical significance level of all the results was set at $\alpha = .05$.

RESULTS

Body composition's change

Body composition's change after 4 weeks of detraining is listed in Table 2. There was no significant difference of

weight for both groups ($p > .05$).

Body mass index ($p < .01$), muscle mass ($p < .001$), fat free mass ($p < .001$), percentage of body fat ($p < .01$) were showed significant differences per detraining but there was no interaction effect from inter-groups and between group and detraining.

Motor unit potential area's change

Surface motor unit potential area's change during isokinetic 60°/sec exercise

Surface motor unit potential area's change during isokinetic 60°/sec exercise following 4 weeks of detraining is listed in Table 3. Interaction effect was not shown from biceps brachii muscle, brachioradialis muscle and vastus lateralis based on detraining, inter-groups, between group and detraining. Rectus femoris and vastus medialis showed significant change per detraining ($p < .05$) but it did not show interaction effect based on inter-groups and between group and detraining.

Surface motor unit potential area's change during isokinetic 180°/sec exercise

Surface motor unit potential area's change during isokinetic 180°/sec exercise following 4 weeks of detraining is listed in Table 4. There was no interaction effect from inter-groups

Table 2. Changes of body composition after detraining

Variables	Group	Pre	Post	△%		p
Weight (kg)	GG	73.61 ± 8.49	73.68 ± 8.18	0.1	Time	.213
					Group	.671
	GA/AA	71.41 ± 8.82	72.25 ± 9.65	1.2	T*G	..281
BMI (kg/m²)	GG	23.79 ± 1.79	23.99 ± 1.76	0.8	Time	.002**
					Group	.525
	GA/AA	23.02 ± 2.39	23.39 ± 2.62	1.6	T*G	.299
Muscle mass (kg)	GG	59.86 ± 5.97	58.46 ± 5.59	-2.3	Time	.000***
					Group	.456
	GA/AA	57.69 ± 6.83	56.11 ± 6.41	-2.7	T*G	.749
Fat free mass (kg)	GG	63.63 ± 6.42	62.00 ± 6.26	-2.6	Time	.000***
					Group	.531
	GA/AA	61.41 ± 7.40	60.04 ± 7.22	-2.2	T*G	.648
Body fat percentage (%)	GG	14.10 ± 2.29	15.15 ± 2.93	7.4	Time	.003**
					Group	.597
	GA/AA	14.53 ± 3.40	16.27 ± 3.57	12.0	T*G	.400

**$p < .01$, ***$p < .001$, △% changes of between pre and post

Table 3. Changes of SMUP at isometric 60°/sec after detraining (μV*sec)

Variables		Group	Pre	Post	△%		p
Elbow flexion	biceps brachii	GG	11.01 ± 3.78	9.06 ± 2.50	-17.7	Time	.089
						Group	.402
		GA/AA	8.99 ± 4.30	7.92 ± 3.32	-11.9	T*G	.598
	brachioradialis	GG	5.82 ± 1.39	5.90 ± 1.26	1.4	Time	.939
						Group	.544
		GA/AA	6.38 ± 2.28	6.37 ± 1.73	-0.2	T*G	.933
Knee Extension	Retus Femoris	GG	2.23 ± 9.33	3.28 ± 1.45	47.1	Time	.023*
						Group	.685
		GA/AA	2.49 ± 8.34	3.45 ± 1.27	38.6	T*G	.912
	Vastus medialis	GG	1.55 ± 0.59	2.35 ± 1.20	51.6	Time	.038*
						Group	.208
		GA/AA	1.96 ± 0.77	2.86 ± 0.92	45.9	T*G	.902
	Vastus lateralis	GG	1.58 ± 0.31	2.20 ± 0.66	39.2	Time	.262
						Group	.113
		GA/AA	2.71 ± 6.08	2.84 ± 1.19	4.8	T*G	.065

*$p < .05$, △% changes of between pre and post

and between group and detraining on biceps brachii muscle and rectus femoris. Brachioradialis muscle and vastus medialis showed significant change per detraining ($p < .01$) but there was no interaction effect from inter-groups and between group and detraining. Vastus lateralis showed a tendency of change per detraining ($p = .05$) and significant interaction effect was shown from between group and detraining ($p < .05$).

Change of muscle function

Change of maximum muscle strength during isokinetic 60°/sec exercise

Change of relative maximum muscle strength during isokinetic 60°/sec exercise and relative average muscle strength during isokinetic 180°/sec exercise per 4 weeks of detraining is listed in Table 5. Elbow flexor showed significant change per detraining during isokinetic 60°/sec exercise ($p < .05$) and there was no interaction effect from inter-groups and between group and detraining. Knee extensor showed no interaction effect from detraining, inter-groups, between group and detraining.

Muscle endurance's change during isokinetic 180°/sec exercise

Elbow flexor showed a tendency of change during isokinetic 180°/sec exercise per 4 weeks of detraining but there was no statistical significance ($p = .059$). Regarding knee extensor, there was no interaction effect from detraining, inter-groups,

Table 4. Changes in SMUP area at isometric 180°/sec after detraining (μN*sec)

Variables		Group	Pre	Post	△%		p
Elbow flexion	biceps brachii	GG	36.67 ± 11.02	39.51 ± 11.33	7.7	Time	.684
						Group	.582
		GA/AA	40.11 ± 12.95	36.39 ± 13.62	-9.3	T*G	.072
	brachioradialis	GG	25.52 ± 5.84	23.95 ± 5.99	-6.2	Time	.007**
						Group	.689
		GA/AA	25.32 ± 7.55	22.43 ± 7.41	-11.4	T*G	.249
Knee Extension	Retus Femoris	GG	12.44 ± 1.77	12.58 ± 4.40	1.1	Time	.379
						Group	.439
		GA/AA	23.28 ± 29.84	11.45 ± 2.28	-50.8	T*G	.368
	Medial Vastus	GG	11.17 ± 4.10	10.64 ± 4.21	-4.7	Time	.003**
						Group	.768
		GA/AA	12.14 ± 4.45	11.06 ± 4.51	-8.9	T*G	.097
	Lateral Vastus	GG	10.56 ± 3.62	10.37 ± 3.20	-1.8	Time	.050
						Group	.468
		GA/AA	12.34 ± 3.56	10.54 ± 2.99	-14.6	T*G	.045*

*$p < .05$, **$p < .01$, △% changes of between pre and post

Table 5. Changes in muscle strength and endurance at isometric 60°/sec and 180°/sec after detraining

Variables		Group	Pre	Post	△%		P
60°/sec (Nm)	Elbow flexor	GG	62.33 ± 4.68	61.00 ± 6.48	-2.1	Time	.026*
						Group	.504
		GA/AA	66.57 ± 8.75	64.14 ± 9.70	-3.7	T*G	.425
	Knee extensor	GG	245.83 ± 32.14	240.33 ± 29.08	-2.2	Time	.083
						Group	.240
		GA/AA	263.29 ± 25.39	259.86 ± 21.99	-1.3	T*G	.667
180°/sec (Watts)	Elbow flexor	GG	43.33 ± 8.50	43.67 ± 4.08	0.78	Time	.059
						Group	.404
		GA/AA	49.57 ± 11.55	47.57 ± 11.50	-4.03	T*G	.552
	Knee extensor	GG	144.17 ± 7.08	151.67 ± 10.37	5.20	Time	.861
						Group	.548
		GA/AA	149.43 ± 24.47	149.57 ± 24.03	0.05	T*G	.457

*$p < .05$, **$p < .01$, △% changes of between pre and post

Table 6. Changes of sports performance after detraining

Variables	Group	Pre	Post	△%		P
Reaction velocity (sec)	GG	0.40 ± 0.06	0.39 ± 0.04	-2.5	Time	.192
					Group	.540
	GA/AA	0.42 ± 0.07	0.40 ± 0.07	-4.8	T*G	.535
Vertical jump (cm)	GG	62.88 ± 4.09	57.25 ± 5.26	-9.0	Time	.000***
					Group	.864
	GA/AA	62.50 ± 2.99	58.20 ± 4.05	-6.9	T*G	.532
Side step (n)	GG	73.38 ± 3.78	70.63 ± 4.69	-3.7	Time	.066
					Group	.242
	GA/AA	70.50 ± 5.97	68.80 ± 3.94	-2.4	T*G	.648
50m run (sec)	GG	7.00 ± 0.23	7.12 ± 0.23	1.7	Time	.924
					Group	.934
	GA/AA	7.09 ± 0.58	6.99 ± 0.61	-1.4	T*G	.105

*$p < .05$, **$p < .01$, △% changes of between pre and post

between group and detraining.

Physical performance's change

The change of physical performance related to exercise per 4 weeks of detraining is list in Table 6. Regarding reaction speed, side step and 50m running, there was no interaction effect from detraining, inter-groups, between group and detraining. Vertical jump showed significant change per detraining ($p < .001$), but there was no interaction effect from inter-groups and between group and detraining.

DISCUSSION

CNTF is cytokine, one of neurotrophic peptide to maintain smooth motor nerve, and it promotes differentiation and survival of various nerve cells such as sensory, sympathetic and ciliary cells [21] and it brings the deterioration and weakening of motor nerve in animal models with neurodegenerative diseases [22] and it is also reported to have nerve-protecting effect [23] that promotes the survival of nerve. On the other hand, many reports came from studies related to CNTF and body composition and it is reported that CNTF affects weight by the activation of intracellular signaling pathways (janus kinases and signal transducers and activators of transcription 3, JAK/STAT3) such as leptin in hypothalamic nuclei which controls appetite and weight and also it is related to anorexia and weight reduction [24,25].

Regarding studies on the difference of body composition per CNTF gene polymorphism, O'Dell *et al*. (2002) [26] reported that when they were categorized 575 elderly men aged between 59 to 73 based on CNTF gene polymorphism and assessed, compared and analyzed them, a mutated homozygote AA type group showed significantly higher score of weight and body mass index compared to a normal homozygote GG type group and a mutated heterozygote GA type group. Compared to that, a longitudinal study [24] consisted of 422 male and female adults aged between 19 to 90 and a longitudinal study [26] consisted of 286 elderly men reported that there was no significant difference of body composition in all variables based on gene polymorphism.

In this study, there was no significant difference of 2 groups in terms of body composition-change per 4 weeks of detraining and there was significant decrease of muscle mass ($p < .001$) and fat free mass ($p < .001$) and significant increase of body mass index ($p < .01$) and body fat ($p < .01$).

But since there was no interaction effect per between group and detraining, there was no difference of body composition per detraining based on CNTF genotype. Such results are believed to suggest that since CNTF has more of localized activation in neuromuscular junction instead of general activation in central nervous system or tissue system, it just affects motor nerve without affecting body composition per gene polymorphism [27] and CNTF is existing in schwann cell and astrocyte [28,29] and its secretion does not increase in a normal state where there is no neurological disease and neuromuscular deterioration.

In the beginning stage of resistance training, muscle strength increases even without muscle hypertrophy and such is possible due to neural adaptation [30]. Also, even for a short term detraining, it was reported that neural factors are affecting the early stage muscle strength loss [6]. As such, the process of gaining or losing of muscle strength is highly related to neuromuscular function.

Therefore, it is expected that if there is a change of CNTF gene, working to maintain and differentiate the survival capacity of various nerve cells including motor nerve, the

deterioration of muscle function per detraining would occur more rapidly.

After 4 weeks of detraining in this study, for upper limbs, there was no significant change of motor unit potential area of biceps brachii muscle and brachioradialis muscle per detraining during isokinetic 60°/sec exercise but elbow flexor showed significant reduction ($p < .05$). Brachioradialis muscle's motor unit potential area was significantly reduced during isokinetic 180°/sec exercise ($p < .01$) and elbow flexor's muscle endurance showed a trend of decrease per detraining ($p = .059$). Such results indicate that this study, which was done on young male adults, is different than the study of Arking *et al*. (2006) [13] since the former did not showed difference of the change of motor unit potential area and elbow flexor per detraining based on CNTF gene-type and the latter reported that when there is gene-mutation of CNTF gene polymorphism, the function of maintaining nerve cell and preventing its deterioration was decreased and thus there was a correlation between CNTF gene polymorphism and muscle strength for the elderly population.

Such differences are believed to be the results of the adaptation differences of serum levels of CNTF and CNTF receptor (CNTFR) based on age [31,32] (Guillet *et al*., 1999; Helgren *et al*., 1994) and also further studies will be necessary for the factors of non-use of muscle based on old age. But the result of this study, where it shows the decrease of brachioradialis muscle's motor unit potential area and elbow flexor's muscle endurance during isokinetic 180°/sec exercise, is believed to support a previous study [6] which reported that the loss of muscle strength is related to the change of motor-nerve and also it is estimated that 4 weeks of detraining decreased muscle function of using motor-units and thus decreased muscle endurance.

For lower limbs, motor unit potential area decreased significantly in rectus femoris and vastus medialis during isokinetic 60°/sec exercise ($p < .05$) but relative maximum strength of knee extensor showed no significant change. Also, there was no interaction effect of between group and detraining and thus no difference from CNTF gene polymorphism is shown. Vastus medialis' motor unit potential area was significantly reduced during isokinetic 180°/sec exercise ($p < .01$) and vastus lateralis showed a tendency of decrease ($p = .050$) per detraining and interaction effect of between group and detraining ($p < .05$) but there was no decrease of muscle endurance per knee extensor's detraining. The result of non-change in lower limb strength was explained by the theory that lower limbs are getting its stimulus while doing daily activities such as walking and thus lower limbs are not so severely affected by detraining [33].

On the other hand, in the case of a GG group, motor unit potential area showed a tendency of increase or very little decrease while maintaining the same level of muscle strength and in the case of vastus lateralis, it showed interaction effect of between group and detraining ($p < .05$) and it can be assumed that such was the result of other type of compensation functions of CNTF nerve cell-maintaining role of a GG group ($p < .05$) [17]. On the other hand, significant decrease of vastus lateralis' motor unit potential area had occurred, even without any significant change of lower limbs' maximum strength and muscle endurance and it supports previous studies that motor nerve function is the first one to decrease from the mechanism of muscle strength decrease due to detraining and it can be explained that GA/AA groups did not get CNTF's protective function unlike the GG group.

Exercise performance showed that vertical jump was significantly decreased in both groups ($p < .001$) but there was no interaction effect of between group and detraining and thus it showed no difference per CNTF gene polymorphism. Exercise performance is considered high when the mechanism between strength of muscle contraction and function of power works effectively [34] and thus the significant change of motor unit potential area of rectus femoris and vastus medialis suggested decrease of the efficiency of the unit mobilization for exercise functions since even without change of muscle strength from detraining, still more units had to be mobilized and thus it can be said that such results induced the decrease of exercise performance.

CONCLUSION

This study showed that there was no difference per detraining for young male adults regardless of CNTF gene mutation which is involved with sustaining and differentiating the survival of various nerve cells including motor nerve.

But it is difficult to confirm with limited variables since other factors such as the change of muscle fiber-ratio, serum levels of CNTF and CNTF receptor were not assessed. Therefore, we suggest that additional studies on serum levels of CNTF, CNTF receptor and the change of muscle fiber should be conducted so that the change of muscle functions can be proven.

Also, this study had a limitation that it could not control all the factors that might affect the change of physical functions per detraining including athletic career, physical activity level, dietary habits, season, duration and type of prior training and motivation.

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