Neuromuscular adaptations induced by adjacent joint training

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Funding information
JSPS KAKENHI, Grant/Award Number: JP15J08355

Effects of resistance training are well known to be specific to tasks that are involved during training. However, it remains unclear whether neuromuscular adaptations are induced after adjacent joint training. This study examined the effects of hip flexion training on maximal and explosive knee extension strength and neuromuscular performance of the rectus femoris (RF, hip flexor, and knee extensor) compared with the effects of knee extension training. Thirty-seven untrained young men were randomly assigned to hip flexion training, knee extension training, or a control group. Participants in the training groups completed 4 weeks of isometric hip flexion or knee extension training. Standardized differences in the mean change between the training groups and control group were interpreted as an effect size, and the substantial effect was assumed to be ≥0.20 of the between-participant standard deviation at baseline. Both types of training resulted in substantial increases in maximal (hip flexion training group: 6.2% ± 10.1%, effect size = 0.25; knee extension training group: 20.8% ± 9.9%, effect size = 1.11) and explosive isometric knee extension torques and muscle thickness of the RF in the proximal and distal regions. Improvements in strength were accompanied by substantial enhancements in voluntary activation, which was determined using the twitch interpolation technique and RF activation. Differences in training effects on explosive torques and neural variables between the two training groups were trivial. Our findings indicate that hip flexion training results in substantial neuromuscular adaptations during knee extensions similar to those induced by knee extension training.

KEYWORDS
electromyography, hip flexion, isometric training, knee extension, maximal strength, rate of torque development, rectus femoris muscle, training specificity

INTRODUCTION

Neuromuscular improvements as a result of resistance training occur within a few weeks. Enhancing the force generated with maximal effort and enhancing the force generated with the intent to move rapidly are the main purposes of resistance training programs. Strength during maximal voluntary contraction (MVC) and/or strength during explosive voluntary contraction (EVC) have been increased as a result of isometric resistance training for 4-6 weeks. These improvements in strength were accompanied by corresponding changes in agonist muscle activation and/or muscle size (hypertrophy).

When interpreting training-induced effects on the neuromuscular performance of a muscle group, training specificity must be considered. Performance is not necessarily improved for a task during which the joint angle and/or muscle contraction type are different from those during the training task but involve the same muscle group. In contrast, if we focus on training specificity for a muscle rather than for a muscle group, then joint action during training can be considered. For muscles that
generate more than one joint action as an agonist (ie, multiarticular muscles), it is poorly understood whether resistance training affects the joint action and the neuromuscular function of the muscle based on the tasks involved during training or if the training changes the function of the muscle during other joint actions. A 4-week wrist abduction training program increased the wrist abduction MVC force by 11% but did not change wrist extension, despite involving muscles that act as agonists during both joint actions of the forearm. The previous finding suggests that strength gains and neural enhancement of the multiarticular muscles are limited to those during joint actions that are involved during the training. In contrast, Lee et al found that voluntary activation (VA%) determined using the twitch interpolation technique was relatively high (~95% or more) during MVC of both wrist abduction and extension before training. The VA% at baseline was associated with the training response in MVC of both wrist abduction and extension before training. Therefore, the relatively high activation may have made it difficult to detect significant changes in wrist extension strength. Because neuromuscular improvements such as enhanced strength and agonist muscle activations during joint actions have resulted without the intent to move the joint (eg, by neuromuscular electrical stimulation), the previously observed training specificity was possibly related to the magnitude of the responsiveness of a muscle to improve with training rather than to the specificity of joint actions during training. This notion can be tested by an interventional study specifically focused on neuromuscular adaptations of the muscle that activates differently during different joint actions.

This study examined the effects of short-term isometric hip flexion training on neuromuscular performance during maximal and explosive isometric contractions of the knee extensors, which have important roles during exercise performance. Compared to other muscle groups, it is difficult to fully activate the knee extensor quadriceps femoris during isometric MVC, which implies that there is room for improvement in neuromuscular performance with isometric strength training. In addition, the magnitude of activation of the biarticular rectus femoris (RF), which crosses the knee and hip joints, was lower during hip flexor isometric MVC than during knee extensor isometric MVC. Therefore, we hypothesized that neuromuscular adaptation of RF during isometric knee extension is induced by isometric hip flexion training as well as by isometric knee extension training; consequently, isometric knee extension performance is substantially improved after isometric hip flexion training. We expected to provide beneficial practical information and greatly improve the understanding of training specificity.

2 | METHODS

2.1 | Participants

Thirty-eight healthy young men who had not performed lower body resistance training for at least 2 years were recruited. The sample size was calculated (G*Power 3.1.7; Kiel University, Kiel, Germany) for an expected effect size (medium, $f = 0.25$) for knee extensor MVC torque with $\alpha$ of 0.05, power of 0.90, and correlation among repeated measures of 0.8. The correlation was input based on a previous interventional study. Estimation revealed that at least 8 participants per group were required to detect the different changes in MVC torque over time among groups. Participants were randomly assigned to hip flexion training (HF group), knee extension training (KE group), or the control (CON) group so that there would not be significant group differences in age, height, and body mass. Considering potential dropouts, we assigned more participants to the training groups than to the CON group. One participant who was assigned to the KE group withdrew due to personal reasons. Thirty-seven participants (HF group: $n = 14$, 22 $\pm$ 2 years, 169 $\pm$ 3 cm, 61 $\pm$ 8 kg; KE group: $n = 12$, 22 $\pm$ 3 years, 170 $\pm$ 4 cm, 61 $\pm$ 7 kg; CON group: $n = 11$, 22 $\pm$ 4 years, 170 $\pm$ 7 cm, 62 $\pm$ 6 kg, mean $\pm$ standard deviation [SD]) completed the study. No participant had a previous or current knee or hip injury. The baseline magnitude of physical activity was assessed using the long version of the International Physical Activity Questionnaire, which was validated in Japanese. No main group effect was observed (HF group, 2443 $\pm$ 1703 metabolic equivalent [MET] min/wk; KE group, 2534 $\pm$ 2233 MET min/wk; CON group, 2210 $\pm$ 2252 MET min/wk). This study was approved by the Ethics Committee of the Shibaura Institute of Technology. The study was performed in accordance with the Declaration of Helsinki. Each participant was informed of the study’s purpose and potential risks and provided written informed consent.

2.2 | Torque recordings

Participants sat on a chair with a dynamometer. The dynamometer was attached to the knee joint at 90° and hip joint at 80° (anatomical position = 0°) during both measurement and training sessions. We used different dynamometers between measurements before and after the intervention (CON-TREX MJ; PHYSIOMED, Schnaittach, Germany) and training (VTK-002; VINE, Tokyo, Japan). The participant’s pelvis and torso were secured to the dynamometer with a non-elastic strap or seat belt. The centers of rotation of the knee/hip joints and dynamometer were carefully aligned. An ankle strap for knee extension measurements was attached slightly proximal to the lateral malleolus, and a pad to evaluate hip flexion strength was positioned approximately 5 cm proximal from the upper border of the patella. The relative position of the participant in relation to the dynamometer and the strap or pad position were carefully matched using a scale between measurements before and after the intervention and throughout the training period. Torque signals were acquired at 4 kHz for the
measurements and at 1 kHz for training data using an A/D converter (PowerLab16/35; ADInstruments, Bella, Vista, Australia) and transferred to a computer. Analyses were performed using LabChart software (ADInstruments). During offline analysis, the torque data were low-pass-filtered at 500 Hz.

2.3 Training

Participants completed 4 sets of 10 unilateral (right leg) isometric hip flexor or knee extensor MVCs 3 times per week with 1-2 days between sessions for 4 weeks. After completing a warm-up procedure comprising submaximal contractions at 30%, 50%, 70%, and 100% of MVCs, the participants flexed their hips (HF group) or extended their knees (KE group) as fast and forcefully as possible and maintained contractions for 3 seconds. The contractions were repeated every 20 seconds (ie, 17-second rest between contractions), with a 2-minute rest period between sets. Verbal encouragement was provided to the participant throughout the training sessions. To examine the effects of the training volume on strength improvement, we determined the training volume during each session as the sum of 40 MVC time-torque integrals and divided it by the sum of the integrals of the first session. If a participant generated the same integral during the baseline measurement and the inferior border of the iliac crest using an anode midway between the superior aspect of the greater trochanter and the inferior border of the iliac crest. For the RF, because of the muscle architecture of the RF and of the vastus lateralis (VL) was measured using ultrasonography.

2.4 Experimental protocol

Participants completed a familiarization session (3-7 days before the baseline measurement) and a measurement session before (5-7 days before the first training session) and after (2-4 days after the last training session) a 4-week training or control period. All measurements and training sessions were conducted for the right leg. Evoked quadriceps femoris twitch responses were first measured. Thereafter, voluntary contraction strength was evaluated. Knee joint strength and hip joint strength were measured randomly. Measurements of knee joint strength were performed in the following order: knee extensor MVC, VA% during knee extensor MVC assessment, knee extensor EVC, and knee flexor MVC. Regarding hip joint strength, hip flexor MVC and EVC measurements were performed. In addition, the muscle architecture of the RF and of the vastus lateralis (VL) was measured using ultrasonography.

2.4.1 Electromyography (EMG) recording

Surface EMG signals were recorded from the RF, VL, and biceps femoris long head (BF) using bipolar Ag/AgCl electrodes (Blue Sensor N-00-S; Ambu A/S, Ballerup, Denmark) with an interelectrode distance of 20 mm. The EMG signals were amplified with a bioamplifier system (MEG-6108; Nihon Kohden, Tokyo, Japan; low-cut filter, 5 Hz; high-cut filter, 1 kHz). The muscle belly and fascicle longitudinal directions were confirmed using real-time B-mode ultrasonography (ACUSON S2000; Siemens Medical Solutions, Malvern, PA, USA). After the areas under the electrodes were shaved, rubbed with sandpaper, and cleaned with alcohol, the electrodes were placed over the belly of the three muscles at 50% of the thigh length, which was determined as the length from the popliteal crease to the greater trochanter. For the RF, because regional differences in muscle activation during knee extension and/or hip flexion have been observed, EMG signals were also obtained from a more proximal region (70% of the thigh length). A reference electrode was placed over the left lateral malleolus. The EMG signals were recorded at a sampling frequency of 4 kHz using the same A/D converter and computer software as those used for the torque signals to synchronize the data. During offline analysis, EMG data were band-pass-filtered between 6 and 500 Hz, except when EMG onset was detected (band-pass filter was off).

2.4.2 Evoked twitch contractions

The quadriceps femoris twitch responses were obtained using a constant current variable voltage stimulator (DS7A; Digitimer Ltd, Hertfordshire, UK). The femoral nerve was stimulated percutaneously with rectangular pulses of 1 ms in the femoral triangle using a cathode (2 × 2 cm) and midway between the superior aspect of the greater trochanter and the inferior border of the iliac crest using an anode (4 × 5 cm). The supramaximal stimulus intensity was determined by increasing the current intensity until plateaus in the twitch torque and peak-to-peak compound muscle action potential amplitude (M max) occurred. Thereafter, five supramaximal twitch responses at a higher current (≥20%) were obtained every 10 seconds. The peak twitch torque, time-to-peak torque, torque at 50 milliseconds from contraction onset, and M max were determined and averaged across five contractions. Contraction onset was manually identified (Figure 1) according to previously described procedures. Briefly, the torque signal was initially viewed with an x-axis scale of ~400 milliseconds and a y-axis scale of ~1 Nm, and the signal onset was approximately determined. Thereafter, the signals were viewed with a higher resolution, and the onset was validated as the last peak or trough within the baseline noise envelope.

2.4.3 Maximal voluntary strength

After performing warm-up procedures consisting of submaximal contractions at intensities of 30%, 50%, and 80% of
MVCs, participants extended the knee and flexed the hip and knee twice as forcefully as possible. Verbal encouragement was provided during contractions. The peak torque of each contraction was defined as MVC torque. If the difference in peak torque between the two contractions was more than 10%, then additional contractions were performed; a 1-minute rest was allowed between contractions. The root mean square values of EMG signals (RMS-EMGs) during MVC were determined over a 0.5-second period around the maximal torque and normalized to Mmax (normalized RMS-EMG) for RF and VL to reduce the effects of peripheral factors on the interpretation of EMG amplitudes. In contrast, the influence of cross talk on RF EMG data from the adjacent muscles was not excluded. The RF EMG amplitude in the proximal region in particular may have been due to cross talk from the two adjacent hip flexor muscles (ie, the sartorius and tensor fasciae latae) because the surface area of RF below the skin should not be large in the proximal region. Therefore, we determined RMS-EMG of RF during knee flexor MVC relative to that during knee extensor MVC. It was 6.5% ± 2.2% in the distal region and 6.5% ± 4.3% in the proximal region, and these values were smaller than those of VL (13.1% ± 5.3%). Therefore, cross talk was unlikely to substantially influence RF EMG. The RMS-EMG of BF during knee extensor MVC was normalized to that during knee flexor MVC. The mean values of the two contractions were used for later analyses. Furthermore, the RMS-EMG ratio during hip flexor MVC to that during knee extensor MVC of RF was calculated.

2.4.4 VA% during knee extensor MVC
After knee extensor MVC testing, an additional two knee extensor MVC measurements were performed using the twitch interpolation technique to assess VA%. Supramaximal triplet stimulations were performed (100 Hz, 1 mseconds duration) during and after knee extensor MVC. The VA% was determined using the following formula: \( \text{VA}\% = (1 - \frac{\text{superimposed triplet torque}}{\text{potentiated resting triplet torque}}) \times 100 \). The mean of the two contractions was used for later analyses. Due to discomfort, VA% of one participant in the KE group could not be obtained.

2.4.5 Explosive voluntary strength
Participants performed 10 explosive voluntary knee extension and hip flexion contractions. The participants were encouraged to perform each contraction as fast and forcefully as possible for approximately 1 second every 20 seconds, with an emphasis on fast. Contractions with an unstable baseline of >0.3 Nm at 200 mseconds before contraction onset due to countermovement or pre-tension were excluded from the analyses. The three contractions with the largest peak slopes (ie, the peak value of the first derivative of the time-torque curve) and peak torque >80% of MVC torque were used for later analyses. As an index of voluntary explosive strength, the rate of torque development (RTD) was defined as the slope of the time-torque curve using three 50-mseconds time windows of 0-50, 50-100, and 100-150 mseconds after torque onset; these were determined in the same way as evoked contractions were determined. The RMS-EMGs of RF, VL, and BF were calculated during the same time periods of RTD from EMG amplitude onset. They were normalized to Mmax for RF and VL and to RMS-EMG during knee flexor MVC for BF. The EMG onset was manually determined in a manner similar to that used to determine torque onset (initial scale with an x-axis of ~400 mseconds and y-axis of 0.02 mV). Furthermore, the electromechanical delay of RF (EMDRF) was determined as the time difference between knee extension or hip flexion torque onset and EMG onset of RF. A negative correlation was shown between EMD of the muscle and tendon stiffness. Furthermore, some studies have indicated a positive correlation between tendon/aponeurosis stiffness and EVC strength. Based on these findings, we considered that EMDRF indirectly indicated the mechanical properties of the tendon.

2.4.6 Muscle architectural measurement
The muscle thickness and pennation angle of RF and VL were measured using B-mode ultrasonography (ACUSON S2000; Siemens Medical Solutions, Malvern, PA, USA) with a 45-mm-wide linear array probe. Participants were in...
the supine position with their legs fully extended on a bed to relax the muscles. The measurement regions were carefully matched before and after the intervention. First, the region was determined along the thigh length. Thereafter, the mediolateral width of each muscle was measured over the skin by identifying the boundaries between adjacent muscles. Finally, the measurement regions were determined as 50% of the thigh length from the popliteal crease to the greater trochanter and 80% of the mediolateral width from the medial boundary for RF (distal region), 70% of the thigh length and 65% of the mediolateral width from the medial boundary for RF (proximal region), and 55% of the thigh length and 55% of the mediolateral width from the medial boundary for VL. We measured these regions because architectural changes in the regions and regional differences in RF hypertrophy were observed due to resistance training with high measurement repeatability, and the muscle thickness changes were accompanied by corresponding changes in anatomical cross-sectional areas of the muscles. Therefore, possible changes in muscle thickness suggested muscle hypertrophy rather than deformation of the muscle architecture obtained by ultrasound imaging. The RF is a bipennate muscle, and the image was obtained from the lateral region by adjusting the probe angle relative to the skin under which the fascicles were visualized. The three longitudinal images in each region were scanned approximately 20 minutes after the participant was supine, with water-soluble transmission gel to avoid pressure-induced depression of the skin surface. Muscle thickness was determined as the mean of the distances between the deep and superficial aponeuroses at both ends of the ultrasound image. The pennation angle was measured as the angle between the fascicle and deep aponeurosis. We did not determine fascicle lengths due to potential errors with a limited probe width. The investigator was blinded to the time, and analyses were performed using ImageJ software (National Institute of Health, Bethesda, MD, USA). The measurements were repeated three times, and the mean values were used for further analyses.

2.5 Statistical analyses

Statistical analyses were conducted using SPSS version 22 (IBM, Armonk, NY, USA). Regarding RMS-EMG of RF in the proximal region, it was difficult to detect \( M_{\text{max}} \) for several participants. Therefore, we only used the data for the RMS-EMG ratio, which did not require \( M_{\text{max}} \) for interpretation. Because of poor repeatability, we did not report RMS-EMGs of VL during hip flexor EVC or BF during EVCs. As an index of the repeatability of measurements, the mean coefficient of variation (CV) was reported for the CON group. Normality of the data distribution was investigated using the Kolmogorov-Smirnov test. If the distribution was skewed, then the data were log-transformed. To facilitate interpretation, all data are presented as mean ± SD of raw data. An independent \( t \) test was used to examine whether the training volume differed between the HF and KE groups. A two-way or three-way analysis of variance (ANOVA) with the between-group factor (group [HF, KE, and CON]) and the within-group factor (time [before and after the intervention] and region [distal and proximal]) was used for dependent variables. Regarding variables of VL, we did not expect the changes that occurred after HF training. Therefore, effects of time and group and their interaction with VL variables were examined between the HF group and CON group and between the KE group and CON group, respectively. When a significant interaction or main effect of time was observed, follow-up ANOVAs with Bonferroni multiple comparisons were performed. A one-sample \( t \) test was performed to examine whether the RMS-EMG ratio of RF before the intervention was different from 1 for each group. Pearson’s product-moment correlation coefficients were determined to examine the relation between training-induced changes in the variables. Statistical significance was set at \( P < .05 \).

To examine whether a significant change in dependent variables was substantial, the standardized difference in the mean change, the difference in mean change between each training group and CON group divided by between-participant SD at baseline, and its uncertainty (90% of the confidence limits [CLs]) were determined and used as an effect size (ES) index. We used 90% CLs to evaluate the training effects. The thresholds for interpreting ES were 0.20, 0.60, and 1.20 for small, moderate, and large effects, respectively. We considered the training effects on variables that changed significantly to be substantial if ES was ≥0.20, and the lower limit of the 90% CLs of ES was ≥−0.20. The magnitude of the group differences in the training effects was interpreted based on the standardized difference in the mean change between the HF and KE groups and their 90% CLs. If the difference was ≥0.20 and if the lower limit was ≥−0.20, then the hip flexion training effects were interpreted as beneficial compared to the knee extension training effects.

3 RESULTS

3.1 Repeatability of dependent variables

The CVs of strength variables for the CON group during the 4-week control period, except for hip flexor RTD during the time window of 0-50 milliseconds (19.1%), were 1.7%-14.5% (ie, <15%); in biological science, this value is considered to indicate borderline acceptable repeatability.7 Regarding EMG variables, most of the CVs were ≤15%, whereas those of the normalized RMS-EMGs of VL during hip flexor MVC and BF during MVCs were 17.1%-18.5%. Poor repeatability was shown (CVs >20%) for RMS-EMGs of VL during hip flexor EVC and for BF during knee extensor and hip
flexor EVCs; therefore, we excluded these variables. The CVs of the architectural parameters were 1.7%-3.4%.

3.2 | Training volume

The total (12 sessions) training volumes (time-torque integral) relative to those of the first session were 13.4 ± 1.6 and 13.6 ± 1.3 for the HF and KE groups, respectively. No significant difference was found between the two groups.

3.3 | Intrinsic contractile properties

The evoked twitch response is shown in Table 1. There was no significant main effect of time or interaction of group × time.

3.4 | Voluntary strength

Figure 2 shows knee extensor and hip flexor MVC torques before and after the intervention. No differences were found among the three groups at baseline for any variables. There was a significant group × time interaction for knee extensor (P < .001) and hip flexor (P < .001) MVC torques. The knee extensor MVC torque significantly increased by 6.2% ± 10.1% (P = .018) and 20.8% ± 9.9% (P < .001) in the HF and KE groups, respectively; however, no significant change occurred in the CON group. A significant increase in the hip flexor MVC torque was shown for the HF group (22.4% ± 12.8%; P < .001), but not for KE group or CON group. No change in knee flexor MVC torque was indicated for any groups.

The RTDs during the time intervals 0-50, 50-100, and 100-150 mseconds from torque onset are shown in Figure 3. There were no baseline differences in variables among the three groups. For knee extensor RTD, the main effect of time was significant during 0-50 mseconds (P < .001) and 50-100 mseconds (P = .001), but not during 100-150 mseconds. The group × time interaction was also significant during 0-50 mseconds (P = .020). For both the HF and KE groups, knee extensor RTDs during the 0- to 50-mseconds and 50- to 100-mseconds time windows significantly increased.

### TABLE 1  Study variables measured before and after the intervention

| Variable                        | HF group (N = 14) | KE group (N = 12) | CON group (N = 11) |
|---------------------------------|-------------------|-------------------|-------------------|
|                                | Before            | After             | Before            | After             | Before            | After             |
| Involuntary strength variables  |                   |                   |                   |                   |                   |                   |
| Twitch                          |                   |                   |                   |                   |                   |                   |
| Peak torque, Nm                 | 28 ± 10           | 29 ± 9            | 29 ± 7            | 31 ± 7            | 25 ± 6            | 25 ± 7            |
| Time-to-peak torque, ms         | 95 ± 7            | 93 ± 7            | 94 ± 7            | 94 ± 5            | 94 ± 9            | 97 ± 8            |
| Torque at 50 ms, Nm             | 19 ± 6            | 18 ± 6            | 19 ± 4            | 20 ± 5            | 17 ± 5            | 15 ± 5            |
| Maximal strength variables      |                   |                   |                   |                   |                   |                   |
| Voluntary activation, %         | 90.3 ± 7.4        | 93.3 ± 5.9*       | 89.4 ± 7.0        | 92.5 ± 6.4*       | 91.2 ± 7.8        | 90.8 ± 9.4        |
| EMG variables                   |                   |                   |                   |                   |                   |                   |
| Electromechanical delay of RF   |                   |                   |                   |                   |                   |                   |
| KE, ms                          | 32 ± 11           | 31 ± 8            | 32 ± 11           | 27 ± 6            | 33 ± 9            | 30 ± 9            |
| HF, ms                          | 40 ± 17           | 29 ± 11           | 31 ± 10           | 31 ± 13           | 34 ± 11           | 34 ± 14           |
| Muscle architecture             |                   |                   |                   |                   |                   |                   |
| RF in the distal region         |                   |                   |                   |                   |                   |                   |
| Muscle thickness, mm            | 17.7 ± 3.2        | 19.4 ± 3.3*       | 18.5 ± 2.2        | 21.1 ± 2.5*       | 17.3 ± 2.5        | 17.1 ± 2.7        |
| Pennation angle, °              | 13.9 ± 2.0        | 14.1 ± 2.8        | 14.5 ± 2.5        | 15.8 ± 1.7*       | 14.2 ± 2.0        | 14.2 ± 2.0        |
| RF in the proximal region       |                   |                   |                   |                   |                   |                   |
| Muscle thickness, mm            | 21.9 ± 3.2        | 24.2 ± 4.1*       | 22.3 ± 2.9        | 23.8 ± 3.4*       | 22.4 ± 2.6        | 22.1 ± 2.7        |
| Pennation angle, °              | 17.3 ± 1.7        | 17.5 ± 1.4        | 17.7 ± 2.8        | 17.4 ± 2.6        | 18.4 ± 2.6        | 18.3 ± 2.6        |
| VL                              |                   |                   |                   |                   |                   |                   |
| Muscle thickness, mm            | 22.6 ± 3.2        | 22.6 ± 3.4        | 21.9 ± 2.6        | 22.6 ± 2.5*       | 22.0 ± 2.3        | 21.9 ± 2.9        |
| Pennation angle, °              | 19.8 ± 2.4        | 19.9 ± 2.3        | 19.0 ± 2.5        | 19.9 ± 2.3        | 19.6 ± 2.5        | 19.3 ± 2.6        |

BF, biceps femoris; CON group, control group; HF group, hip flexion training group; KE group, knee extension training group; M_max, peak-to-peak compound muscle action potential amplitude; MVC, maximal voluntary contraction; RF, rectus femoris; RMS-EMG, root mean square of electromyogram; VL, vastus lateralis.

Data are mean ± standard deviation of raw data.

*Indicates a significant change after the intervention.
FIGURE 2 Peak torque during maximal voluntary contraction (MVC) of knee extension (A) and hip flexion (B) Data are shown as mean ± standard deviation. *Indicates a significant change after the intervention. CON, control group; HF, hip flexion training group; KE, knee extension training group

FIGURE 3 Rate of torque development (RTD) of knee extension (left) and hip flexion (right) before and after the intervention. The RTD was defined as the slope of the time-torque curve during time intervals of 0–50 ms (A and B), 50–100 ms (C and D), and 100–150 ms (E and F) from the onset of knee extension or hip flexion. Data are shown as mean ± standard deviation. *Indicates a significant change after the intervention. CON, control group; HF, hip flexion training group; KE, knee extension training group
In contrast, no variables changed in the CON group. Regarding hip flexor RTD, the main effect of time ($P = .003$) and the group × time interaction ($P < .001$) were significant only during 50-100 mseconds. Follow-up analyses demonstrated that hip flexor RTD during 50-100 mseconds significantly increased in the HF group ($P < .001$) but not in the KE group or CON group.

### 3.5 EMG variables

The RMS-EMGs of each muscle during MVC are shown in Figure 4. No baseline differences in variables were found among the groups. Regarding RMS-EMGs during knee extensor MVC, a significant main effect of time ($P = .006$) without a significant group × time interaction was found for RF. The RMS-EMG of RF significantly increased in the HF ($P = .013$) and KE ($P = .013$) groups, but it did not change in the CON group. The two-way ANOVAs and follow-up analyses indicated that RMS-EMG of VL increased only in the KE group ($P = .006$). The RMS-EMG of BF did not change for any groups. For RMS-EMG during hip flexor MVC, the main effect of time ($P = .002$) and group × time interaction ($P = .006$) were significant for RF but not for VL or BF. A significant increase in RMS-EMG of RF was found only in the HF group ($P < .001$). There was a significant correlation between the relative change in the RF RMS-EMG during knee extensor MVC and that during hip flexor MVC in the HF group ($r = .768; P = .001$).

**FIGURE 4** The root mean square values of the electromyogram (RMS-EMG) during maximal voluntary contractions (MVCs) of the rectus femoris (RF; A and B), vastus lateralis (VL; C and D), and biceps femoris long head (BF; E and F) before and after the intervention. The RF and VL values were normalized to the peak-to-peak compound muscle action potential amplitude ($M_{\text{max}}$), and those of BF were normalized to those during knee flexor MVC. Data are shown as mean ± standard deviation. *Indicates a significant change after the intervention. †Denotes a significant difference in the RMS-EMG of RF before the intervention between knee extension and hip flexion. CON, control group; HF, hip flexion training group; KE, knee extension training group.
Regarding the RMS-EMG ratio for RF, the ratio before the intervention was significantly <1 for all groups ($P \leq .001$-.005), indicating that the RMS-EMG for RF during hip flexor MVC was significantly lower than that during knee extensor MVC. The main effect of region ($P < .001$) and the group $\times$ time interaction ($P = .040$) was significant for the ratio. A significant simple main effect of time was shown only for the HF group ($P = .010$), indicating a significant increase in the ratio (distal: $0.65 \pm 0.20$ [before] and $0.84 \pm 0.26$ [after]; proximal: $0.72 \pm 0.24$ [before] and $0.91 \pm 0.34$ [after]). The ratio for the distal region before the intervention was negatively correlated with the relative change in RMS-EMG for RF during hip flexor MVC ($r = -.568$; $P = .034$). In contrast, no simple main effect of time was shown for the KE group or the CON group.

For RMS-EMG during knee extensor EVC (Figure 5), the two-way ANOVA showed a significant group $\times$ time interaction ($P = .005$-.047) for the RMS-EMG for RF during 0-50 and 50-100 ms. The RMS-EMG for RF significantly increased during the two phases in the HF and KE groups ($P \leq .001$-.017). The RMS-EMG for VL during knee extensor EVC did not change during any phases for all the groups.

**FIGURE 5** The root mean square of the electromyogram (RMS-EMG) during explosive voluntary contractions of knee extension and hip flexion of the rectus femoris (left and center) and vastus lateralis (right) before and after the intervention. The values were normalized to peak-to-peak compound muscle action potential amplitude ($M_{\text{max}}$). The RMS-EMGs were determined for time intervals of 0-50 ms (A-C), 50-100 ms (D-F), and 100-150 ms (G-I) from the onset of the electromyogram. Data are shown as mean $\pm$ standard deviation. *Indicates a significant change after the intervention. CON, control group; HF, hip flexion training group; KE, knee extension training group.
Regarding the RMS-EMG for RF during hip flexor EVC, significant increases were observed during 50-100 milliseconds ($P = .001$) and 100-150 milliseconds ($P = .001$) in the HF group. No main effects or interactions were observed for EMDRF (Table 1).

### 3.6 VA%

The results of VA% are shown in Table 1. A significant main effect of time ($P = .009$) with a group × time interaction ($P = .048$) was shown. Follow-up analyses revealed that VA% significantly increased in the HF ($P = .007$) and KE groups ($P = .015$); however, it did not change significantly in the CON group. The relative VA% change was positively correlated with the relative change in the knee extensor MVC torque in the HF and KE groups (Figure 6).

### 3.7 Muscle architecture

The architectural parameters appear in Table 1. A significant interaction of group × time × region was shown for RF muscle thickness ($P = .023$) and pennation angle ($P = .036$). In the CON group, no change was observed for any variable. In the HF group, the RF thickness significantly increased in the distal (10.3% ± 8.7%; $P < .001$) and proximal (10.1% ± 6.6%; $P < .001$) regions. In the KE group, significant increases were shown in the two regions (distal: 14.3% ± 6.9%, $P < .001$; proximal: 6.7% ± 4.9%, $P < .001$), and the relative change was significantly greater in the distal region than the proximal region according to a paired $t$ test ($P < .001$). Regarding the RF pennation angle, an increase (10.7% ± 12.2%; $P < .001$) was observed only in the distal region of participants in the KE group, and the relative change in the distal region was significantly greater than that in the proximal region ($P = .005$). For VL parameters, VL thickness significantly increased by 3.4% ± 4.6% ($P = .011$) in the KE group; however, it did not change in the HF or CON group. No change in VL pennation angle was observed in any groups.

### 3.8 Interpretation of the training effects

Regarding the variables that changed significantly in each training group, the summary of the mean and its uncertainty of ES is shown in Figure 7. Training effects on the variables were interpreted as substantial for all variables. Figure 8 indicates the group differences in the training effects on the variables that changed substantially in at least one training group. The HF and KE groups showed beneficial effects compared to the other training group for some variables, respectively.

### 4 DISCUSSION

The current study demonstrated that substantial improvements in maximal and explosive knee extension strengths and RF activation were induced after 4 weeks of isometric hip flexion as well as knee extension training. Thus, our hypothesis was supported. The group differences in training effects on knee extensor RTD and RF activation during knee extensor MVC and EVC were trivial. Because the total training volume was not different between the two groups, the observed similarities in group responses were not due to the differences in training volume. Based on the training specificity, it is reasonable that the effects of hip flexion training on knee extension strength and neuromuscular function of RF are much smaller than those of knee extension training. Therefore, the present findings raise questions about training specificity and indicate that neuromuscular adaptations are induced by adjacent joint training.

Substantial improvements in the activation of trained muscles during MVC were observed in each group (Figure 4). The results suggest that neural adaptation of the agonist muscles was a major factor of MVC torque enhancement in the two training groups. Supporting this notion, there were positive correlations between the relative changes in knee extensor MVC torque and VA% in the two training groups (Figure 6). In contrast, because no significant change was shown in the knee flexor MVC torque, BF activation during MVC, or evoked quadriceps femoris torques in any group, a substantial influence of change in antagonist activation and muscle hypertrophy of agonist muscles on MVC torques enhancement was unlikely to have occurred. It should be noted that the lack of change in the evoked response in the HF group might have involved the influence of the training response in the sartorius, which acts as a knee flexor as well as a hip flexor while being innervated by the femoral nerve. Therefore, the force exerted by the sartorius can negatively affect the evoked knee extension torque. However, because the volume of the
sartorius is less than half of the RF volume,\textsuperscript{24} substantial effects are less likely.

Contrary to the training specificity, hip flexion training had substantial effects on RF activation during knee extensor MVC, with a trivial group difference in the effect as compared to knee extension training as well as during hip flexor MVC. In contrast, the substantial effect of knee extension training on RF activation was limited to that during knee extensor MVC. The threshold of the hip flexion training effect on RF activation during hip flexor MVC was large, whereas a moderate effect on RF activation during knee extensor MVC was shown following knee extension training (Figure 7). Therefore, a large effect of training may be required to transfer the effect to adjacent joint action.

In the current study, RF activation during hip flexor MVC was lower than that during knee extensor MVC before the intervention (Figure 4). Moreover, for the HF group, there was a negative correlation between the RMS-EMG ratio of RF at baseline and the relative change in RF activation during hip flexor MVC and a positive correlation between the relative changes in RF activation during hip flexor MVC and during knee extensor MVC. These findings suggest that greater improvements in RF activation following hip flexion training resulted from a lower magnitude of RF activation during hip flexion rather than training specificity: The training effects on the neural performance of a muscle are not limited to the joint that the muscle influences during training. Instead, the effects are likely to be related to the activation level of muscle before training.

The knee extensor RTD was notably enhanced during the early phases (ie, $\sim$100 milliseconds) after both hip flexion training and knee extension training. It has been shown that the EVC force during the early phase was mainly determined by twitch force and muscle activation of the agonist muscles.\textsuperscript{25} In addition, the training-induced increase in early-phase knee extensor EVC force was accompanied by a corresponding increase in the quadriceps femoris EMG activity.\textsuperscript{26} Because the previous study\textsuperscript{26} averaged EMG values across the three (RF, VL, and vastus medialis) muscles, the effect of the neural adaptation of individual muscles on the EVC force was unclear. In our study, twitch torque did not change in either training group. The muscle activation of RF increased in both training groups, with a trivial group difference in the magnitude of training effect; however, VL activation did not change in either training group. These results suggest that neural adaptation of the RF rather than the vasti muscles is a mechanism of RTD improvement; therefore, the group differences in the training effects on RTD were trivial (Figure 8). In contrast, other factors such as muscle architecture\textsuperscript{27,28} and tendon or aponeurosis mechanical properties\textsuperscript{18,19} are possibly related
to EVC strength. In our study, the increase in RF muscle thickness and the corresponding change in the pennation angle were observed only in the distal region of participants in the KE group. We did not find any changes in intrinsic contractile properties (Table 1), and an increase in the pennation angle was negatively associated with the EVC strength change. Considering these points, the effects of changes in muscle architecture seem to have little influence on EVC strength changes. Similarly, the lack of a significant change in EMDRF in both training groups suggested that a substantial change in tendon mechanical properties was unlikely to occur. Altogether, improvements in neural activation were the major contributors to RTD enhancement in the two training groups.

To the best of our knowledge, the current study is the first to compare the architectural adaptation of the muscle using two different joint action strength training programs as an agonist. Both training programs substantially increased the RF thickness. The results are consistent with those of a previous study that observed muscle hypertrophy after only 20 days of resistance training intervention. The preferential RF thickness increase in the distal region following isometric knee extension training was in line with that of previous studies that used dynamic knee extension training. Therefore, inhomogeneous quantitative changes in the RF along the length may occur following knee extension training, irrespective of the type of muscle contraction. The mean values of the relative changes in the two regions were similar between the two groups. The magnitude of hypertrophy may be large considering the short-term training in the present study. This might have been due to the effect of muscle swelling. However, ultrasound scans were performed 2-4 days after the last training session. Participants were relaxed in the supine position approximately 20 minutes before the scan. None of the participants reported muscle pain. Therefore, the aforementioned effect was likely to be small. Although the training volume was comparable between the two training groups, mechanical stress on the RF during training should have been larger in the KE group than in the HF group because the RMS-EMG ratio of RF was <1. Myofibrillar protein synthesis after training involving volume-matched resistance exercise was dependent on exercise intensity and reached a plateau at 60% of a one-repetition maximum load. In our study, at baseline, the RMS-EMG ratio of the RF in the HF group was 0.65 in the distal region and 0.72 in the proximal region; these values
increased after the intervention. Therefore, the magnitude may be sufficient to induce quantitative adaptation of the RF after HF training with maximal effort.

There were some limitations to our study. Overall, group differences (HF vs. KE groups) in training effects on knee extension strength and neural adaptation of the RF during knee extension contractions were considered trivial based on the standardized difference in the mean change, except for knee extensor MVC torque (Figure 8). Considering the uncertainties of the standardized difference in the mean change, however, it is difficult to conclude that the hip flexion training effects on these variables are equivalent to those of knee extension training. It is important to note that the superiority of knee extension training compared to hip flexion training was not obvious. Furthermore, the transfer of strength gained from hip flexion training to knee extensor force production may involve neural effects other than RF neural improvement (e.g., the participants learned motivation to apply force). The magnitude of change in knee extensor MVC torque after hip flexion training (6.2%) as compared to that following knee extension training (20.8%) is considered to be a conceivable value based on the relative contribution of RF (approximately 24%) to knee extension strength. Moreover, hip flexion training did not induce significant change in VL activation during knee extensor MVC. Therefore, the aforementioned effects on the present findings would be small.

In conclusion, the present study revealed that short-term isometric hip flexion training, similar to knee extension training, induces substantial improvement of maximal and explosive knee extension performance and neuromuscular enhancement of the RF. Neural adaptation of the RF was considered a major factor for the increase in knee extension strength, but not for musculotendinous adaptations, irrespective of the training modality. Before the intervention, the RF activation was lower during hip flexions than knee extensions. The magnitude of neural adaptation of the RF during knee extensions following hip flexion training was not substantially different from that after knee extension training, which is not in line with previous knowledge of training specificity. Our findings suggest that the magnitude of neuromuscular improvements in muscles after training is not specific to the joint action during training but is related to the responsiveness of the muscle to training.

5 | PERSPECTIVES

The present study provides evidence that the intent to extend the knee is not necessarily needed to improve maximal and explosive knee extensor performances. This is because hip flexion training as well as knee extension training induces a substantial RF neuromuscular adaptation during knee extension. Maximal and explosive hip flexion strengths and RF neural activation during hip flexion were improved by hip flexion training but not by knee extension training. Therefore, a 4-week period of isometric hip flexion training could be beneficial compared to isometric knee extension training for enhancing the RF neuromuscular function. Hip flexion training may be useful for individuals, such as those who have knee pain; thus, knee extension is difficult to perform during training and rehabilitation.

ACKNOWLEDGEMENTS

This work was supported by JSPS KAKENHI Grant Number JP15J08355. We thank the members of Japan Institute of Sports Sciences for their help with data acquisition.

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REFERENCES

1. Del Balso C, Cafarelli E. Adaptations in the activation of human skeletal muscle induced by short-term isometric resistance training. J Appl Physiol. 2007;103:402-411.
2. Noorkõiv M, Nosaka K, Blazevich AJ. Neuromuscular adaptations associated with knee joint angle-specific force change. Med Sci Sports Exerc. 2014;46:1525-1537.
3. Tillin NA, Pain MT, Folland JP. Short-term training for explosive strength causes neural and mechanical adaptations. Exp Physiol. 2012;97:630-641.
4. Kitai TA, Sale DG. Specificity of joint angle in isometric training. Eur J Appl Physiol Occup Physiol. 1989;58:744-748.
5. Sale DG, Martin JE, Moroz DE. Hypertrophy without increased isometric strength after weight training. Eur J Appl Physiol Occup Physiol. 1992;64:51-55.
6. Lee M, Gandevia SC, Carroll TJ. Short-term strength training does not change cortical voluntary activation. Med Sci Sports Exerc. 2009;41:1452-1460.
7. Gondin J, Guette M, Ballay Y, Martin A. Electromyostimulation training effects on neural drive and muscle architecture. Med Sci Sports Exerc. 2005;37:1291-1299.
8. Thorpe SK, Li Y, Crompton RH, Alexander RM. Stresses in human leg muscles in running and jumping determined by force plate analysis and from published magnetic resonance images. J Exp Biol. 1998;201:63-70.
9. Behm DG, Whittle J, Button D, Power K. Intermuscle differences in activation. Muscle Nerve. 2002;25:236-243.
10. Miyamoto N, Wakahara T, Kawakami Y. Task-dependent inhomogeneous muscle activities within the bi-articular human rectus femoris muscle. PLoS ONE. 2012;7:e34269.
11. Ema R, Wakahara T, Miyamoto N, Kancheha H, Kawakami Y. Inhomogeneous architectural changes of the quadriceps femoris induced by resistance training. Eur J Appl Physiol. 2013;113:2691-2703.
12. Craig CL, Marshall AL, Sjöström M, et al. International physical activity questionnaire: 12-country reliability and validity. *Med Sci Sports Exerc.* 2003;35:1381-1395.

13. Murase N, Katsumura T, Ueda C, Inoue S, Shimomitsu T. Validity and reliability of Japanese version of International physical activity questionnaire. *J Health Welfare Statist.* 2002;49:1-9.

14. Balshaw TG, Massey GJ, Maden-Wilkinson TM, Tillin NA, Folland JP. Training-specific functional, neural, and hypertrophic adaptations to explosive- vs. sustained-contraction strength training. *J Appl Physiol.* 2016;120:1364-1373.

15. Watanabe K, Saito I, Akagi R. In vivo measurement of human rectus femoris architecture by ultrasonography: validity and applicability. *Clin Physiol Funct Imaging.* 2013;33:267-273.

16. Hopkins WG. Spreadsheets for analysis of controlled trials, with adjustment for a subject characteristic. *Sportsscience.* 2006;10:46-50.

17. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

18. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

19. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

20. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

21. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

22. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

23. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

24. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

25. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

26. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

27. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

28. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

29. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

30. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

31. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

32. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

33. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

34. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

35. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

36. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

37. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

38. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

39. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

40. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

41. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

42. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

43. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

44. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

45. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

46. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

47. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

48. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

49. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

50. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

51. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

52. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

53. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

54. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

55. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

56. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

57. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

58. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

59. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.

60. Hopkins WG, Marshall SW, Batterham AM, Hanin J. Progressive statistics for studies in sports medicine and exercise science. *Med Sci Sports Exerc.* 2009;41:3-13.