Neural adaptations after 4 years vs. 12 weeks of resistance training vs. untrained

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Title page

Title:
NEURAL ADAPTATIONS AFTER 4 YEARS VS. 12 WEEKS OF RESISTANCE TRAINING VS. UNTRAINED

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Abbreviated title for running head:
NEURAL ADAPTATIONS TO RESISTANCE TRAINING

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Abstract

The purpose of this study was to compare the effect of resistance training (RT) duration, including years of exposure, on agonist and antagonist neuromuscular activation throughout the knee extension voluntary torque range. Fifty-seven healthy men (untrained [UNT] *n*=29, short-term RT [12WK] *n*=14, and long-term RT [4YR] *n*=14) performed maximum and sub-maximum (20-80% maximum voluntary torque [MVT]) unilateral isometric knee extension contractions with torque, agonist and antagonist surface EMG recorded. Agonist EMG, including at MVT, was corrected for the confounding effects of adiposity (i.e. muscle-electrode distance; measured with ultrasonography). Quadriceps maximum anatomical cross-sectional area ($QACSA_{MAX}$; via MRI) was also assessed. MVT was distinct for all three groups (4YR +60/+39% vs. UNT/12WK; 12WK +15% vs. UNT; $0.001<P\leq0.021$), and $QACSA_{MAX}$ was greater for 4YR (+50/+42% vs. UNT/12WK; [both] $P<0.001$). Agonist EMG at MVT was +44/+33% greater for 4YR/12WK ([both] $P<0.001$) vs. UNT; but did not differ between RT groups. The torque-agonist EMG relationship of 4YR displayed a right/down shift with lower agonist EMG at the highest common torque (196 Nm) compared to 12WK and UNT ($0.005\leq P\leq0.013$; Effect size [ES] $0.90\leq ES\leq1.28$). The torque-antagonist EMG relationship displayed a lower slope with increasing RT duration (4YR<12WK<UNT; $0.001<P\leq0.094$; $0.56\leq ES\leq1.31$), and antagonist EMG at the highest common torque was also lower for 4YR than UNT (-69%; $P<0.001$; ES=1.18). In conclusion, 4YR and 12WK had similar agonist activation at MVT and this adaptation may be maximised during early months of RT. In contrast, inter-muscular coordination, specifically antagonist co-activation was progressively lower, and likely continues to adapt, with prolonged RT.

Introduction

Regular resistance training (RT) leads to increases in maximum strength, the maximum force/torque that can be produced by the trained musculature. These improvements in strength can increase the mobility of older adults [1], enhance athletic performance [2], reduce injury risk [3], and may decrease the likelihood of developing musculoskeletal disorders, such as osteoarthritis [4]. Consequently, regular and persistent RT is recommended for athletes, the general population, and older adults alike [5,6]. Neural adaptations have been widely documented to contribute to the increases in strength following short-term RT (up to 16 weeks in duration; [7-13]). It has often been assumed that changes in neuromuscular activation primarily occur in this initial short-term period of RT with no/minimal further adaptations thereafter [14], although there is evidence that extensive neuroplasticity can occur in response to long-term RT [15-17]. At present, however it is unclear if adaptations in neuromuscular activation continue to occur with prolonged RT (i.e. over several years) and might contribute to changes in function (strength).

Surface electromyography (EMG) measurements have generally demonstrated increased neuromuscular activation of the agonist muscles at maximum torque after relatively short-term RT [8-13], although whether these adaptations in maximum agonist activation continue over longer periods of RT remains largely unexplored. Two longitudinal RT interventions with already highly resistance-trained men found no [18] or marginal [19] improvements in activation during isometric actions. Furthermore, Moritani and deVries [20] first proposed that changes in the full range of the torque-agonist EMG relationship may reveal useful information regarding the nature of underpinning physiological adaptations following RT. They hypothesised that changes in the position and extension of the torque-agonist EMG relationship after training could indicate neural (Fig. 1A), morphological (Fig.

1B), or a combination of both neural and morphological adaptations (Fig. 1 C) [20]. Existing literature suggests an extension of the torque-agonist EMG relationship following 4 wk of RT [11,21] (e.g. Fig. 1A, neural adaptation), but a shift to the right with no extension after 8 wk to 6 mo of RT [22-24] (e.g. Fig. 1B, morphological, but no neural adaptation) which represents an incongruous time course of neural adaptations (increase and then decrease). Whilst it is likely the classic Moritani and DeVries model over simplifies the relative contribution of neural and morphological adaptations, the influence of more prolonged RT (i.e. for multiple years) on the torque-agonist EMG relationship has not been examined, and thus the time course of any changes remains unknown. In the first few months of RT the changes in strength appear to be primarily dependent on neural adaptations [25] and thus a similar, but extended, torque-agonist EMG relationship may be expected (Fig. 1A). Whereas with years of RT substantial hypertrophic, in addition to neural, adaptations are thought to occur [26] and the relationship may be expected to shift down/right, whilst extending to a similar maximum EMG as observed after short-term RT (Fig. 1C).

Antagonist co-activation at maximum torque has been found to be unchanged [27], increased [28], and decreased [29] following short-term RT studies. These differences may be because antagonist co-activation is positively related to both torque and agonist activation, such that increased maximum torque and agonist activation after RT tend to cause an increase in antagonist co-activation, even if antagonist co-activation at the same torque has decreased [11]. In which case the relationships between antagonist co-activation and torque/agonist activation may provide a more complete assessment. In fact, Tillin et al [11] found that after short-term RT co-activation was reduced at any absolute level of torque/agonist activation, even though co-activation at maximum torque was unchanged, likely due to the increase in torque/agonist activation. However, whether prolonged RT, leads to further potentially more

substantial adaptations in antagonist co-activation, beyond these short-term changes, is unclear.

Overall, it is unknown whether several years of RT causes continued adaptations in agonist activation and antagonist co-activation, and specifically changes the nature of the inter-relationships between these neural variables and torque, beyond those documented by short-term intervention studies. Whilst a longitudinal study of several years duration may be impractical, a comparison of groups with distinct durations of RT experience may facilitate investigation of these issues. Therefore, the purpose of the current investigation was to conduct a detailed comparison between untrained (UNT) vs. short-term RT (12 weeks [12WK]) vs. long-term RT (average 4 years [4YR]) men for agonist and antagonist neuromuscular activation throughout the voluntary torque range. The position of the torque-agonist EMG and torque-antagonist EMG relationships were assessed by determining their slope and also EMG amplitude at the highest common torque (i.e. the highest torque achieved by all participants). Based on limited existing evidence it was hypothesised that maximum agonist activation would be greater for 12WK vs. UNT, but with no further difference between 12WK and 4YR. It was also hypothesised that the torque-agonist EMG relationship, would: have a similar position, for 12WK vs. UNT (Fig. 1A); have a down/rightwards position for 4YR vs. UNT and 12WK (Fig. 1C). The final hypothesis was that antagonist co-activation at the same torque/agonist activation would progressively decrease with greater training duration (i.e. lower position of the torque-antagonist EMG relationship; 4YR<12WK<UNT).

Materials and Methods

Participants

A total of fifty-seven young, healthy, asymptomatic, males provided written informed consent prior to participation in this study that was approved by the Loughborough University Ethical Advisory Committee. Physical activity levels of all participants were assessed with the International Physical Activity Questionnaire [IPAQ, short format [30]]. The UNT group consisted of 29 participants (IPAQ: 2358 ± 1476 metabolic equivalent min/wk) who had not completed lower-body RT for >18 months and were not involved in systematic physical training. The 12WK group comprised 14 participants (IPAQ: 2097 ± 1303 metabolic equivalent min/wk) measured within a week of completing 12-weeks of supervised isometric knee extension RT (3 x/wk, 40 reps of 3 s at 75% maximum voluntary torque [MVT]), who were originally recruited from an identical population to the UNT group (i.e. no RT for >18 months and not involved in any systematic physical training). Finally, the 4YR group consisted of 14 participants (IPAQ: 5568 ± 1457 metabolic equivalent min/wk) who reported (via a detailed questionnaire and follow-up oral discussion) systematic, progressive heavy RT of the quadriceps (i.e. completion of several knee extensor exercises within an individual session ~3 x/wk typically consisting of: squat, lunge, step-up, and leg press) for ≥3 years (mean ± SD, 4 ± 1 years; range, 3-5 years) with the primary aim of developing maximum strength. The RT of this group had not been experimentally supervised although some of these participants had received variable coaching (technique and programming) support. Use of androgenic-anabolic steroids was an exclusion criterion for all participants. Many individuals in the 4YR group reported regular use of nutritional supplements (e.g. whey protein and creatine).

Overview

UNT, 12WK, and 4YR participants visited the neuromuscular laboratory for a familiarisation session involving isometric voluntary maximum and sub-maximum

contractions. Thereafter, two duplicate neuromuscular measurement sessions were conducted on the dominant leg (7-10 days apart). Finally, MRI and ultrasound scans were performed within 7 days of the second neuromuscular measurement session. Neuromuscular measurement sessions were at a consistent time of day for each individual and started between 12:00-19:00. These sessions involved recordings of isometric knee extension/flexion torque and surface EMG of the superficial quadriceps and hamstrings muscles during voluntary maximum and a range of sub-maximum contractions (20, 40, 60 and 80%). The primary outcome measures were maximum torque and simultaneous agonist and antagonist EMG, as well as the position of the torque-agonist EMG and torque-antagonist EMG relationships assessed by both relationship slope and EMG at the highest common torque. Whilst not primary outcomes the following measurements were also completed: (i) muscle size, assessed with a T1-weighted 1.5T MRI scan of the thigh of each participant's dominant leg (see "*Muscle size*" below), as an additional index of training status and morphological differences between the three groups; and (ii) muscle-electrode distance (MED) using B-mode ultrasonography at the sites where quadriceps EMG sensors were placed to correct for the pronounced, confounding influence of subcutaneous tissue thickness, primarily body fat, on voluntary EMG amplitude [31].

12WK supervised resistance training intervention

All training sessions involved the same dynamometer and configuration used for the measurements (see below). After a brief warm-up of sub-maximum contractions of both legs, participants completed four sets of ten sustained unilateral isometric knee extension contractions of each leg; with sets alternating between dominant and non-dominant legs until 4 sets per leg had been completed. Each set took 60 s with 2 min between successive sets on the same leg. This training model has been described extensively elsewhere [8] and was

selected for the 12WK group within the current study as it has shown to produce increases in maximum strength during short-term RT interventions [11,32]. Briefly, participants were presented with a target torque trace (on a computer monitor in front of them) 2 s before every contraction and were instructed to match this target trace, which increased torque linearly from rest to 75%MVT over 1 s before holding a plateau at 75%MVT for a further 3 s. 12WK participants performed three maximum voluntary isometric contractions (MVCs; see below) at the start of each training week to re-establish MVT and prescribe training torques. 12WK participants were instructed to maintain their habitual physical activity and diet throughout the 12-week training period.

Torque and EMG recording

Measurements were completed in a rigid custom-made isometric dynamometer with knee and hip angles of 115° and 126° (180° = full extension), respectively (as shown in Fig. 6B of reference [33]). Adjustable straps were tightly fastened across the pelvis and shoulders to prevent extraneous movement. An ankle strap (35 mm width reinforced canvas webbing) was placed ~15% of tibial length (distance from lateral malleolus to knee joint space), above the medial malleolus, and positioned perpendicular to the tibia and in series with a calibrated S-beam strain gauge (Force Logic, Swallowfield, UK). The analogue force signal from the strain gauge was amplified (x370) and sampled at 2,000 Hz using an external A/D converter (Micro 1401; CED Ltd., Cambridge, UK) and recorded with Spike 2 computer software (CED Ltd., Cambridge, UK). In offline analysis, force data were low-pass filtered at 500 Hz using a fourth-order zero-lag Butterworth filter, gravity corrected by subtracting baseline force, and multiplied by lever length, the distance from the knee joint space to the centre of the ankle strap, to calculate torque values.

Surface EMG was recorded from the superficial quadriceps (agonist EMG: rectus femoris [RF]; vastus lateralis [VL]; vastus medialis, [VM]) and hamstring muscles (antagonist EMG: biceps femoris [BF] and semitendinosus [ST]) using a wireless EMG system (Trigno; Delsys Inc., Boston, MA). Before single differential Trigno Standard EMG sensors (Delsys Inc., Boston, MA; fixed 1-cm interelectrode distance) were positioned skin preparation (shaving, abrading, and cleansing with 70% ethanol) was conducted. Individual sensors were attached (using adhesive interfaces) at six separate sites over the superficial quadriceps muscles at set percentages of thigh length (above the superior border of the patella) as follows: RF 65 and 55%; VL 60 and 55%; VM 35 and 30%. Similarly, individual sensors were placed on the BF and ST at 45% of thigh length above the popliteal fossa. Sensors were placed parallel to the presumed orientation of the underlying fibres. EMG signals were amplified at source (x300; 20- to 450-Hz bandwidth) before further amplification (overall effective gain, x909), and sampled at 2,000 Hz via the same A/D converter and computer software as the force signal, to enable data synchronization. In offline analysis, EMG signals were corrected for the 48-ms delay inherent to the Trigno EMG system.

Measurement sessions

Following a brief warm-up of the dominant leg (3 s knee extension contractions at 50% [x3], 75% [x3], and 90% [x1] of perceived maximum) measurements were completed in the following order.

Knee extension maximum voluntary contractions

Participants performed 3-4 MVCs and were instructed to “push as hard as possible” for 3-5 s and rest for ≥30 s between efforts. A torque-time curve with a horizontal cursor

indicating the greatest torque obtained within that session was displayed for biofeedback and verbal encouragement was provided during all MVCs [11,34]. Knee extensor MVT was the greatest instantaneous torque achieved during any MVC during that measurement session. Root mean square (RMS) EMG for a 500 ms epoch at MVT (250 ms either side) was calculated for each electrode site. RMS EMG from each quadriceps site was then averaged to provide an overall quadriceps EMG measurement during MVT production (agonist EMG_{MVT}). RMS EMG from each of the hamstring sites during knee extension MVT (antagonist EMG_{MVT}) was normalised to that measured during knee flexion MVT (knee flexion EMG_{MAX}; see below) and then averaged across the two hamstring sites.

Knee extension sub-maximum voluntary contractions

Horizontal cursors indicating four sub-maximum target torque levels were placed on the screen displaying the real-time torque-time curve and participants were instructed to gradually increase torque (over ~1 s) and match the prescribed torque level for ~5 s at 20, 40, 60 or 80%MVT, performed in this order, with ≥30 s between efforts. From each recorded contraction, a 500 ms period of stable torque at approximately the prescribed level was identified and used to calculate mean knee extension torque. RMS EMG of each quadriceps and hamstring EMG site was measured for each of these epochs.

Knee flexion maximum voluntary contractions

Knee flexion MVCs were performed in the same manner as knee extension, except participants performed a series of sub-maximum knee flexion efforts to warm-up and were instructed to "pull as hard as possible" for 3-5 s, rather than "push" [11,34]. Knee flexion MVT was the greatest instantaneous torque achieved during any MVC during that measurement

session. RMS hamstring EMG for a 500 ms epoch at knee flexion MVT (250 ms either side) was analysed for each site (knee flexion EMG_{MAX}).

Muscle size

A 1.5T MRI scan of the dominant leg was made in the supine position at a knee joint angle of ~163° using a receiver 8-channel whole body coil (Signa HDxt, GE). T1-weighted axial slices (5 mm thick, 0 mm gap) were acquired from the anterior superior iliac spine to the knee joint space in two overlapping blocks. Oil filled capsules placed on the lateral side of the participants' thigh allowed alignment of the blocks during analysis. MR images were analysed by two trained investigators using Osirix software (version 6.0, Pixmeo, Geneva, Switzerland). The quadriceps (RF, VL, VM, and vastus intermedius; VI) muscles were manually outlined in every third image (i.e. every 15 mm) starting from the most proximal image in which the muscle appeared. The image with the largest anatomical cross-sectional area (ACSA) was defined as the maximum ACSA for each individual quadriceps muscle and the sum of the muscles was defined as maximum quadriceps ACSA ($QACSA_{MAX}$).

Muscle-electrode distance and correction of agonist EMG.

Images of the distance between the skin surface and the peripheral surface of the muscle at each of the six sites where quadriceps EMG sensors were positioned (i.e. muscle-electrode distance, MED) were collected using a B-mode ultrasonography machine (EUB-8500, Hitachi Medical Systems UK Ltd, Northamptonshire, UK) with a 9.2 cm wide linear-array transducer (EUP-L53L), sampling at 32 Hz, interfaced with a personal computer operating ezcap video capture software. The transducer was coated with water-soluble transmission gel and placed perpendicular to the skin over the RF, VL, and VM at the percentages of thigh length listed above for each quadriceps EMG sensor. Images were later

imported in to a public domain software (Tracker version 4.92; www.cabrillo.edu/~dbrown/tracker) and MED was measured by one trained investigator.

When agonist EMG data for all participants was pooled (i.e. n= 57) there were inverse relationships between absolute EMG amplitude and MED for all sensor locations and all types of contraction (maximum and all sub-maximum levels; Pearson's product moment bivariate correlations, $-0.670 \leq r \leq -0.394$; $0.001<P\leq0.002$). Additionally, when comparing the three groups MED differed or tended to differ (One-way ANOVA $0.014\leq P\leq0.070$) with several "Moderate" to "Large" effect sizes ($0.71\leq ES\leq1.03$) between groups at three out of six sites. Consequently, all individual agonist EMG measurements were corrected for MED at the corresponding site, using the quadratic relationship between agonist EMG amplitude and MED at that specific measurement site. Correcting EMG amplitude measurements for the amount of subcutaneous tissue at the recording site [35] or using MED as a covariate within statistical testing [36] are approaches that have previously been employed. The MED correction in the current study involved summating the individual's residual absolute agonist EMG amplitude, in comparison to the cohort relationship with MED (e.g. agonist EMG amplitude vs. MED), with the pooled group mean for absolute agonist EMG amplitude [37]. Overall corrected agonist EMG during all maximum and sub-maximum contractions was then calculated by averaging the corrected EMG amplitude measurements from each EMG recording site.

Data analysis and statistics

All torque and EMG measurements from the two neuromuscular measurement sessions were averaged to produce criterion values. Bivariate relationships were then analysed with Pearson's product moment correlation. Only agonist EMG values corrected for

MED were used for establishing relationships. Three relationships were plotted and assessed for each individual participant: knee extension torque vs. corrected agonist EMG; knee extension torque vs. normalised antagonist EMG; and normalised antagonist EMG vs. corrected agonist EMG. Relationships were fitted with linear functions, but not forced through zero as forcing the function through zero significantly reduced the R^2 values of all three relationships (paired t-test, [all] $P<0.001$). The position of the torque-agonist EMG and torque-antagonist EMG relationships were assessed with: (i) relationship slope ('m' constant of the linear function) and (ii) EMG at the highest common torque achieved by all participants (196 Nm, equivalent to the MVT of the weakest participant and indicated as a vertical dotted line in Fig. 3A and B). EMG values for each participant at the highest common torque were derived by solving the individual linear function (for an x axis value of 196 Nm) for the relationship between isometric knee extension torque and either agonist or antagonist EMG. The slope of the antagonist EMG-agonist EMG relationship was also calculated.

All statistical analyses were performed using SPSS Version 24.0 (IBM Corp., Armonk, NY). Data are reported or displayed as means ± SD, except in figures displaying EMG relationships where for presentation purposes data points with x and y error bars to the far right of the figure display average SD for the five load increments across the voluntary torque range for each group. One-way ANOVAs were conducted as the main statistical tests to assess if differences existed between groups for: descriptive characteristics (i.e. age, height, and body mass); agonist EMG_{MVT}; antagonist EMG_{MVT}; the slope derived from the relationships (i.e. torque-agonist EMG, torque-antagonist EMG, antagonist EMG-agonist EMG); and agonist EMG and antagonist EMG at the highest common knee extension torque. When one-way ANOVAs displayed $P<0.05$, a combined post-hoc criteria involving both a least significant difference (LSD) P value of <0.10 and an effect size (ES) >0.50 were

required for there to be considered good evidence of between-group differences. LSD P values were stepwise corrected for multiple comparisons [38] and ES was calculated as previously detailed for between-subject study designs [39] and classified as follows: <0.20 "Trivial," 0.20 – 0.49 "Small," 0.50 – 0.80 "Moderate," or >0.80 "Large".

Between-test session reliability of key measurements was assessed by pooling all three groups of participants (i.e. *n*= 57) using: (i) within-participant coefficient of variation (CV_W, [SD/mean] × 100) as a measure of absolute reliability; and (ii) intra-class correlation coefficient (ICC; two-way mixed, absolute agreement) to assess relative reliability. CV_W values were interpreted as "acceptable" <12%, "intermediate" 12–20%, or "unacceptable" >20% [40]. ICC values were interpreted as ''very high'' 0.90–1.00, ''high'' 0.70–0.89, "moderate" 0.50–0.69, "low" 0.30–0.49, "negligible" 0.00–0.29 [41].

Results

Between-test session reliability

Knee extension and knee flexion MVT returned mean CV_W values of 2.7% and 10.8% and ICC values of 0.980 and 0.889, respectively. Absolute agonist and antagonist EMG at knee extension MVT demonstrated mean CV_W values of 8.8% and 17.8% and ICC values of 0.937 and 0.774, respectively. Agonist EMG corrected for MED and normalised antagonist EMG (both) at knee extension MVT produced mean CV_W values of 8.3% and 25.8% and ICC values of 0.876 and 0.790, respectively.

Descriptive characteristics, muscle strength and size

Age, height, and body mass were similar for 12WK (24 ± 2 y; 1.75 ± 0.08 m; 70 ± 9 kg) and UNT (25 ± 2 y; 1.76 ± 0.07 m; 74 ± 10 kg) groups ([all variables] LSD P≥0.247). The 4YR group were younger (22 ± 2 y), taller (1.84 ± 0.06 m), and heavier (92 ± 10 kg) than

the other two groups ([all variables] LSD P≤0.004). Knee extension MVT of 12WK (293 ± 49 Nm) was 15% greater than UNT (255 ± 42 Nm; LSD P=0.021; ES=0.86 "Large"), whilst MVT of 4YR (407 ± 63 Nm) was 60% greater than UNT and 39% greater than 12WK ([both] P<0.001; 2.02≤ES≤3.07 [both] "Large"; Fig. 2A). Knee flexion MVT of 4YR (104 ± 21 Nm) was 72% greater than UNT (61 ± 23 Nm; LSD P<0.001; ES=1.91 "Large") and 63% greater than 12WK (64 ± 15 Nm; P<0.001; ES=2.19 "Large"), but no differences in knee flexion MVT occurred between 12WK and UNT (P=0.636; ES=0.16 "Trivial"). $QACSA_{MAX}$ of 4YR was 50% greater than UNT and 42% greater than 12WK ([both] LSD P<0.001; 2.78≤ES≤3.63 [both] "Large"); but did not differ between 12WK and UNT (P=0.204; ES=0.42 "Small"; Fig. 2B).

Absolute agonist and antagonist EMG at knee extension maximum voluntary torque

Absolute agonist EMG_{MVT} was greater for 4YR (+66%; LSD P<0.001; ES= 1.53 "Large") and 12WK (+32%; P=0.050; ES=0.78 "Moderate") compared to UNT (Table 1). In addition, absolute agonist EMG_{MVT} was greater for 4YR than 12WK (+25%; LSD P=0.048; ES=0.72 "Moderate"; Table 1). In contrast, there were no statistical differences between groups for absolute antagonist EMG_{MVT} (Table 1).

Corrected agonist and normalised antagonist EMG at knee extension maximum voluntary torque

Agonist EMG_{MVT} (corrected for MED) was greater for both 4YR (+44%; LSD P<0.001; ES=1.73 "Large") and 12WK (+33%; P<0.001; ES=1.29 "Large") than UNT; but was not statistically different between the two RT groups (P=0.281; ES=0.35 "Small"; Fig. 2C and Table 1). Normalised antagonist EMG at knee extension MVT was not different between groups (Table 1).

Knee extension torque-agonist EMG relationship

The torque-agonist EMG relationship was well represented by a linear function with high R^2 values for all three groups regardless of RT experience (4YR, 0.978 ± 0.026; 12WK, 0.970 ± 0.054; UNT, 0.972 ± 0.066). There were no differences between the groups for slope of the torque-agonist EMG relationship (Table 1; Fig. 3A). However, agonist EMG at the highest common torque (196 Nm) was lower for 4YR compared to UNT (-24%; LSD P=0.013; ES=0.90 "Large") and 12WK (-30%; P=0.005; ES=1.28 "Large") indicating a downward shift in the torque-agonist EMG relationship (Fig. 4A). No differences in agonist EMG at 196 Nm occurred between 12WK and UNT (LSD P=0.296; ES=0.35 "Small"; Fig. 4A).

Knee extension torque-antagonist EMG relationship

The torque-antagonist EMG relationship was well represented by a linear function with high R^2 values regardless of RT experience (4YR, 0.971 ± 0.020; 12WK, 0.952 ± 0.043; UNT, 0.894 ± 0.111). The slope of the knee extension torque-antagonist EMG relationship differed between all three groups (Table 1), being lower for 12WK than UNT (-30%; LSD P=0.061; ES=0.56 "Moderate"), and lower for 4YR than 12WK (-52%; P=0.094; ES=1.22 "Large") or UNT (-66%; P<0.001; ES=1.31 "Large"; Fig. 3B and Table 1). Antagonist EMG at 196 Nm of knee extension torque was lower for 4YR compared to UNT (-69%; LSD P<0.001; ES=1.18 "Large"; Fig. 4B and Table 1) but not vs. 12WK (P=0.108; ES=1.23 "Large"). No differences in antagonist EMG at 196 Nm occurred for 12WK compared to UNT (LSD P=0.120; ES=0.46 "Small"; Fig. 4B).

Antagonist EMG-Agonist EMG relationship during knee extension contractions

The slope of the antagonist EMG-agonist EMG relationship was lower for both 4YR (-59%; LSD P<0.001; ES=1.22 “Large”) and 12WK (-37%; P=0.028; ES=0.77 “Moderate”) vs. UNT (Fig. 5 and Table 1). The 12WK group appeared to occupy an intermediate position between the other two groups although there was no difference between the slopes of the two trained groups (12WK vs. 4YR; LSD P=0.202; ES=0.69 “Moderate”; Fig. 5). High R^2 values for the antagonist EMG-agonist EMG relationship were displayed for all groups regardless of RT experience (4YR, 0.982 ± 0.012; 12WK, 0.972 ± 0.031; UNT, 0.892 ± 0.135).

Discussion

The purpose of this study was to compare neuromuscular activation of the agonist and antagonist musculature during knee extension contractions throughout the voluntary torque range between long-term RT (4YR), short-term RT (12WK) and untrained (UNT) groups. In agreement with our hypothesis, maximum agonist activation (corrected for MED) was higher for both RT groups than UNT, but did not differ between 12WK and 4YR. As hypothesised, the torque-agonist EMG relationship for 12WK had a similar position to UNT (slope and agonist EMG at 196 Nm), for 4YR occupied a lower position than the other two groups (lower agonist EMG at 196 Nm), although the slope was similar for all groups. The position of the torque-antagonist EMG relationship also showed distinct differences between groups with lower slope according to RT duration (4YR<12WK<UNT) and lower antagonist EMG at the highest common knee extension torque for 4YR vs. UNT. Based on these findings it appears that changes in maximum agonist activation predominantly occur in the first weeks of RT, but not substantially thereafter, although the 4YR RT group displayed a down/rightwards position of the torque-agonist EMG relationship presumably due to substantial hypertrophy. In contrast, the lower antagonist co-activation for 4YR than 12WK,

evidenced by differences in the slope of the torque-antagonist EMG relationship, suggests that inter-muscular co-ordination may be the primary long-term neural adaptation to RT.

The greater agonist activation (both absolute EMG and corrected for MED) at maximum voluntary torque of both RT groups compared to the untrained cohort supports numerous previous reports that agonist activation increases following RT [8-13], although some older studies have reported no change in agonist EMG amplitude after RT [22,23]. Moreover, the greater neuromuscular activation (maximum agonist EMG) of 12WK vs. UNT, coupled with the similar muscle size of these groups supports the concept that strength gains following short-term RT result predominantly from neural adaptations [14,20]. In fact, we recently found the largest determinant of the change in strength following 12-weeks of RT to be the increase in agonist neuromuscular activation (EMG), explaining 30.6% of the variance in strength gains [25]. The greater agonist neuromuscular activation of 12WK vs. UNT may be due to increased motor unit firing rate [42] and/or recruitment of additional motor units [43], but these mechanisms were not discernible from the current EMG amplitude measurements. Whilst absolute agonist EMG also showed differences between the two RT groups (12WK vs. 4YR) this appeared to be in part due to the lower MED of the 4YR group, as there were no differences in agonist EMG amplitude between the two RT cohorts, once corrected for MED. Overall, these findings suggest that maximum agonist activation increases in the first 12 weeks of RT, but does not continue to adapt beyond 12 weeks of RT (up to ~4 years).

The consistent position, but extended, torque-agonist EMG relationship of 12WK compared to UNT in the current cross-sectional study was in agreement with short-term longitudinal RT studies [11,21]. The lower agonist EMG at the highest common torque of the 4YR group (vs. UNT or 12WK) confirmed the visual impression that the torque-agonist

EMG relationship was positioned further to the right for this group, despite the observation that the slope of the torque-agonist EMG relationship was similar for all three groups. The longest intervention studies we are aware of reported a qualitative reduction in the slope of the torque-agonist EMG relationship after 6 months of RT [23,24], which is broadly supportive of the observation that the position of the relationship is adaptable and shifts down/right with prolonged RT. A logical explanation of the 4YR group's lower agonist EMG amplitude to produce the same knee extension torque, and thus the subsequent down/rightwards position of the torque-agonist EMG relationship of this group, is substantial hypertrophy [20] and/or possible greater neuromuscular efficiency [44]. Indeed, the 4YR group had considerably larger quadriceps than the other two groups (+42/+50% greater $QACSA_{MAX}$ vs. 12WK/UNT). Hypertrophied muscle would be expected to require activation of fewer, but larger, fibres to achieve the same torque production and hence lower agonist EMG.

Antagonist EMG amplitude at knee extension MVT was not different between groups, despite 4YR having 28-38% lower normalised antagonist EMG than the other groups. Measurements of antagonist EMG amplitude at MVT are likely confounded by the differences in MVT between groups, which demonstrably effects antagonist EMG via the extremely strong torque-antagonist EMG relationship we have described (R^2>0.89), as well as the large variability in this measurement [11], and these issues probably explain the confused findings for antagonist co-activation at MVT within the literature [27-29]. In this case the position of the relationships between antagonist EMG and torque/agonist EMG or antagonist EMG at a common torque level may be a more reliable and meaningful measures of antagonist co-activation. In fact, the slope of the torque-antagonist EMG relationship was distinct between all three groups (4YR<12WK<UNT), being ~two-thirds less steep for 4YR than UNT. Similarly, antagonist EMG at the highest common knee extension torque was also

substantially lower for 4YR than UNT (-69%), and even though not significantly different comparably large differences were demonstrated between 4YR vs 12WK (-57%). Finally, the agonist-antagonist activation relationship appeared visually to show distinct and progressively lower positions according to RT duration, but only revealed differences for both RT groups vs. UNT. Overall, these findings provide convincing evidence that antagonist co-activation shows substantial scope for continued adaptation beyond the first 12 weeks of training, and thus may be the primary long-term neural adaptation to RT.

The finding that antagonist co-activation was progressively lower as a function of RT duration (i.e. slope of torque-antagonist EMG relationship: UNT>12WK>4YR), despite there being no difference in maximum agonist activation between groups with 12 weeks or ~4 years of RT experience, indirectly supports cortical excitability and spinal reflex response research suggesting that agonist and antagonist activation are modulated by different supraspinal mechanisms [45]. Whilst our understanding of the precise mechanisms (i.e. supraspinal and/or spinal) that modulate antagonist co-activation is still incomplete [46], the progressive decrease in antagonist co-activation across the three groups with increasing RT experience in the current study indicates that with prolonged RT (up to ~4 years) antagonist co-activation likely contributes to increased strength due to reduced antagonist knee flexion torque. It would be highly interesting to be able to accurately translate these apparent changes in antagonist neuromuscular activation to quantitative changes in antagonist torque, however this is problematic for several reasons: we have assessed co-activation of only two of nine knee flexor muscles; such a calculation would require the agonist EMG-knee flexion torque relationship of all these muscles; which is itself confounded by antagonist quadriceps activation. Nonetheless, on a relatively simplistic level, assuming a linear knee flexion torque-agonist EMG relationship in order to calculate antagonist knee flexion torque (i.e. %

normalised antagonist EMG x knee flexion MVT) at the common knee extensor torque of 196 Nm, reveals antagonist torque for UNT of 11.6 Nm (19.0% x 61 Nm), 8.8 Nm for 12WK (13.7% x 64 Nm) and 6.1 Nm for 4YR (5.9% x 104 Nm). The observation that maximum agonist neuromuscular activation was similar for 12WK compared to 4YR, whilst antagonist co-activation showed some marked but functionally small differences between these groups, strongly supports the notion that other adaptations, primarily morphological changes, such as the 4YR group's substantial hypertrophy ($QACSA_{MAX}$: +42% vs. 12WK), is the primary explanation for their much greater strength (MVT: +114 Nm vs. 12WK).

Given the practical issues with implementing supervised RT interventions for multiple years the results of the current study provide novel insight in to how the human neuromuscular system likely adapts with continued RT. Nonetheless it is important to consider the limitations of the current study. The cross-sectional design clearly provides a weaker level of evidence than longitudinal intervention studies, and make it impossible to fully discern the contribution of selection (i.e. innate differences [nature]), as opposed to the influence of RT (nurture), which is the primary question of this research. For example, it is conceivable that individuals attracted to regular prolonged RT (i.e. the 4YR group), are innately stronger, perhaps due to specific neuromuscular differences conceivably including better inter-muscular co-ordination, than the normal population. Nonetheless, whilst the 4YR group were clearly selected for their characteristic RT history, and thus were by definition distinct from the normal population, this was not the case for the 12WK group that were initially recruited from an identical population as the UNT group with only a minor proportion randomly assigned to the 12WK RT intervention. Thus, within the current findings when there is a clear progression across the groups (i.e. UNT>12WK>4YR as for the slope of the torque-antagonist EMG relationship) we can be more confident that this was

not due to selection bias, but in all probability due to the duration of RT. In addition, when there are no differences between groups (e.g. 4YR vs. 12WK as for corrected agonist EMG during MVT) it seems likely that RT duration does not have a pronounced effect.

Although both the 12WK and 4YR groups were performing heavy RT, the contraction modes employed were different between the two groups (i.e. 12WK: isometric RT; 4YR: concentric and eccentric RT). It is possible that the task specific training of the 12WK group (isometric training specific to isometric testing) might have produced task-specific neural adaptations [8,47], which was not the case for the 4YR group, and thus accentuated task-specific adaptations of 12WK may have minimised the neural differences between these two groups. In this case the neural, and potentially also strength, differences that we have documented between 12WK and 4YR may well have been more pronounced given identical RT modes. Nonetheless the pronounced differences in strength between these groups appeared to be primarily due to morphological differences, rather than similar corrected agonist activation or functionally small differences in antagonist co-activation between the groups. Moreover, multiple-year longitudinal RT intervention studies employing contemporary EMG techniques, careful antagonist EMG-torque/agonist EMG relationships, and measurements at multiple intervals throughout the course of the intervention are required to confirm the findings we report here. Whilst we considered including the interpolated twitch technique in the current study, it was excluded due to several studies demonstrating its limited sensitivity to detect changes in activation after RT [11,48-50].

It should also be noted that the results of the present investigation could be specific to the knee joint and the open kinetic chain knee extension task that was used. Further thorough investigations of agonist, antagonist, and stabiliser muscle activation during other single-joint

and also multiple-joint and/or multiplanar strength tasks are necessary to gain greater understanding of the nature of neural adaptations to long-term RT. The limitations of surface EMG measurements have been widely documented and may not provide an ideal index of the neural drive to the muscle [51]. For example, the size of the surface action potential has been found to be only moderately associated with motor unit size [52]. In addition, it is possible that amplitude cancellation (i.e. when positive and negative phases of concurrent action potentials overlap and reduce the sum of the surface EMG measurement) could have influenced the results of the current study [53], especially given there is evidence that increased motor unit synchronisation can occur following RT [15,54]. Therefore, it is recommended that future work utilise other techniques (e.g. EMG decomposition, transcranial magnetic stimulation) to better understand the changes in muscle activation with prolonged RT.

In conclusion, it appears that maximum agonist activation changes predominantly occur in the first weeks of RT (up to 12 wk), but not substantially thereafter, although long-term RT (up to ~4 years) led to a rightwards shift of the torque-agonist EMG relationship presumably due to the substantial hypertrophy of these participants. Interestingly, antagonist co-activation was progressively lower according to RT duration suggesting that inter-muscular co-ordination may be the primary long-term neural adaptation to RT. Multiple-year longitudinal RT intervention studies employing appropriate neural measurements at multiple intervals throughout the course of the intervention are required to confirm the cross-sectional findings observed in the present investigation.

Perspectives

Prior to this study it was largely unexplored whether several years of resistance training (RT) causes continued adaptations in agonist activation and antagonist co-activation

beyond short-term RT, although Moritani & DeVries (1979) hypothesised specific changes in the torque-agonist EMG relationship according to neural (short-term) and hypertrophic (long-term) adaptations. Differences in agonist activation were broadly as speculated by Moritani & DeVries (1979) with greater maximum agonist activation after short-term, but without further changes after long-term RT, and a rightwards shift in the torque-agonist activation relationship only after long-term RT. In addition, it was particularly interesting that there was lower co-activation of the antagonist muscles according to RT duration that suggests continued improvements in inter-muscular co-ordination. The findings of the current investigation have potential implications for the practices and physiological understanding of individuals prescribing and/or undertaking long-term RT. Future research in this area is clearly warranted to investigate the influence of long-term (multiple-year) RT interventions on agonist, antagonist, and stabiliser neuromuscular activation during diverse mechanical tasks/conditions (including isometric and isoinertial, and single- and multiple-joint and/or multiplanar strength tasks).

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None to declare

Conflict of interest

The authors declare that there is no conflict of interest, that no companies or manufacturers will benefit from the results of the study, and that the results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation.

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FIGURE CAPTIONS

Fig. 1 Illustration of pre- to post-training changes in the torque-agonist EMG relationship considered to be representative of: (A) exclusively neural adaptations (unchanged slope but extended relationship with a right and upward shift of the maximum point; (B) exclusively hypertrophic adaptations (lower slope with the maximum point shifted to the right); (C) a combination of neural and hypertrophic adaptations (lower slope, but with a maximum point shifted to the right and upwards). Adapted from Moritani and deVries [20].

Fig. 2 (A) Knee extension maximum voluntary torque; (B) quadriceps maximum anatomical cross-sectional area ($QACSA_{MAX}$); and (C) agonist EMG amplitude (corrected for muscle-electrode distance) of untrained (UNT), short-term resistance-trained (12WK), and long-term resistance-trained (4YR) groups. Data are mean ± SD. Symbols indicate differences between groups: * greater than UNT; † greater than 12WK.

Fig. 3 The relationship between torque and (A) agonist EMG amplitude (corrected for muscle-electrode distance) and (B) normalised antagonist EMG throughout the knee extension voluntary torque range for untrained (UNT), short-term resistance-trained (12WK), and long-term resistance-trained (4YR) groups. Data points to the far right of Fig. 3A and B display x and y error bars that are the mean SD for torque (x error bars) and EMG amplitude (y error bars) for the five load increments across the voluntary torque range for each group. Knee Flexion EMG_{MAX}, agonist EMG during isometric knee flexion maximum voluntary torque.

Fig. 4 (A) Agonist EMG amplitude (corrected for muscle-electrode distance); and (B) normalised antagonist EMG at the highest common isometric knee extension torque achieved by all participants (196 Nm; derived by solving individual linear equations) for untrained (UNT), short-term resistance-trained (12WK), and long-term resistance-trained (4YR) groups. Data are mean ± SD. Symbols indicate differences between groups: * lower than UNT; † lower than 12WK. Knee Flexion EMG_{MAX}, agonist EMG during isometric knee flexion maximum voluntary torque.

Fig. 5 The relationship between agonist EMG amplitude (corrected for muscle-electrode distance) and normalised antagonist EMG throughout the knee extension voluntary torque range for untrained (UNT), short-term resistance-trained (12WK), and long-term resistance-trained (4YR) groups. Data points to the far right of the figure display x and y error bars that are the mean SD for agonist EMG (x error bars) and antagonist EMG (y error bars) amplitude for the five load increments across the voluntary torque range for each group. Knee Flexion EMG_{MAX}, agonist EMG during isometric knee flexion maximum voluntary torque.

TABLES

Table 1. Agonist and antagonist surface EMG amplitudes and the slope of the relationship between agonist EMG and torque/antagonist EMG for untrained (UNT), short-term resistance-trained (12WK), and long-term resistance-trained (4YR) groups.

	UNT (*n*=29)	12WK (*n*=14)	4YR (*n*=14)	ANOVA P value
Activation at MVT:				
Absolute agonist EMG (mV)	0.182 ± 0.073	0.241 ± 0.081*	0.301 ± 0.087*†	<0.001
Absolute antagonist EMG (mV)	0.019 ± 0.009	0.020 ± 0.007	0.016 ± 0.008	0.540
Corrected agonist EMG (mV)	0.192 ± 0.042	0.255 ± 0.061*	0.277 ± 0.060*	<0.001
Normalised antagonist EMG (% Knee flexion EMG_{MAX})	23.1 ± 14.0	20.0 ± 10.0	14.4 ± 10.9	0.107
Activation at highest common torque (196 Nm)				
Corrected agonist EMG (mV)	0.151 ± 0.039	0.164 ± 0.039	0.116 ± 0.040*†	0.004
Normalised EMG (% Knee flexion EMG_{MAX})	19.0 ± 13.2	13.7 ± 7.8	5.9 ± 4.5*	0.001
Slope of linear relationship:				
Torque (Nm)-Corrected agonist EMG (mV)	$8.163 \pm 2.186 \times 10^{-4}$	$9.280 \pm 2.578 \times 10^{-4}$	$7.507 \pm 2.461 \times 10^{-4}$	0.138
Torque (Nm)-Normalised antagonist EMG (% Knee flexion EMG_{MAX})	0.109 ± 0.065	0.077 ± 0.041*	0.037 ± 0.023*†	<0.001
Normalised antagonist EMG (% Knee flexion EMG_{MAX})-Corrected agonist EMG (mV)	132 ± 72	83 ± 41*	54 ± 43*	<0.001

Data are mean ± SD. One-way ANOVAs and subsequent post-hoc tests were used to establish differences between groups. Symbols indicate differences between groups: * Different than UNT; † Different than 12WK. Knee flexion EMG_{MAX}, agonist EMG during isometric knee flexion maximum voluntary torque. Corrected agonist EMG was calculated by using the quadratic relationship between EMG amplitude and muscle-electrode distance for all participants (*n*=57) on a sensor and contraction intensity specific basis before averaging across sites to derive overall agonist EMG measurements.

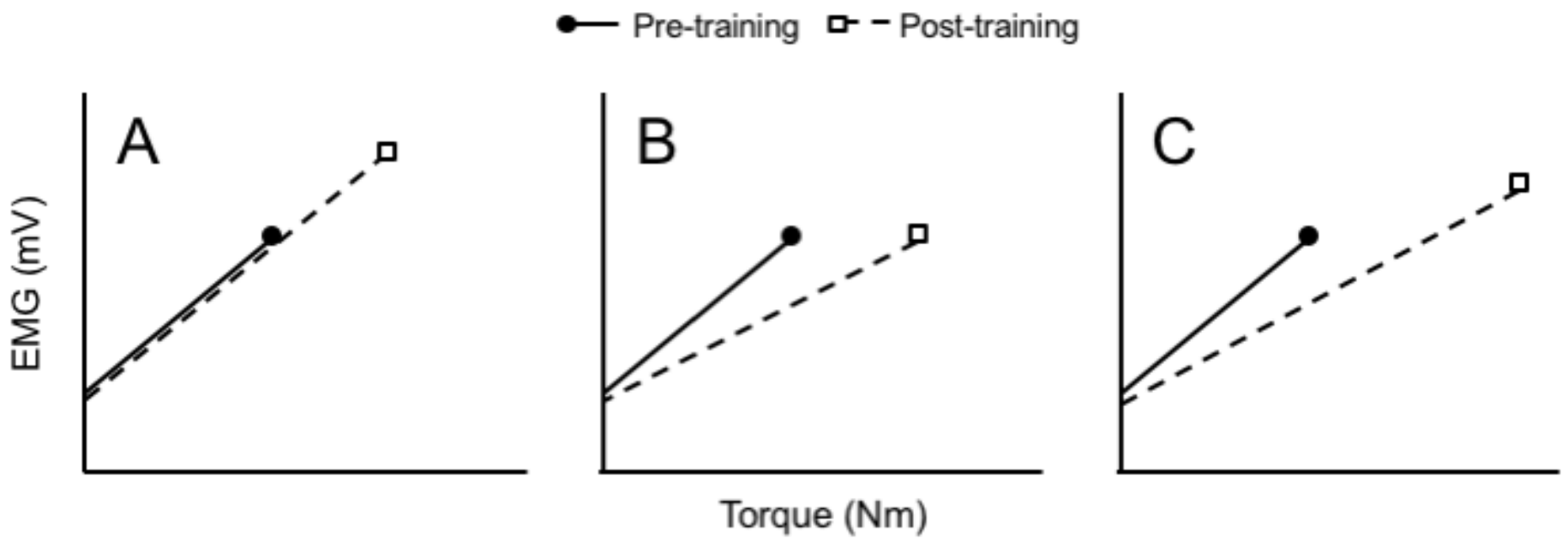
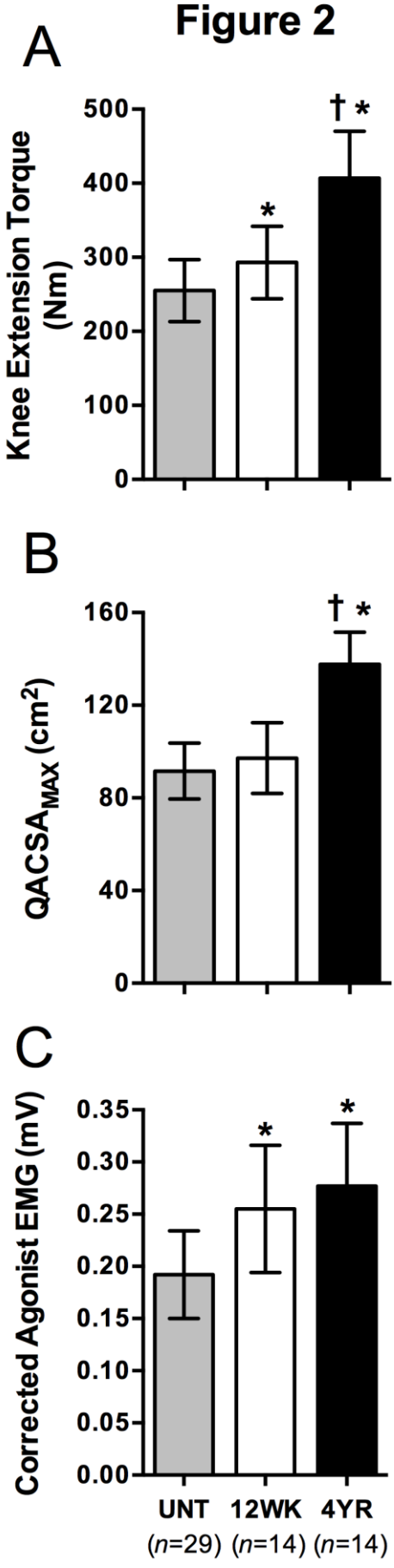


Figure 1

Figure 2


A
Knee Extension Torque (Nm)
500
400
300
200
100
0
† *
*
B
QACSA$_{MAX}$ (cm^2)
160
120
80
40
0
† *
C
Corrected Agonist EMG (mV)
0.35
0.30
0.25
0.20
0.15
0.10
0.05
0.00
*
*
UNT
12WK
4YR
(n=29)
(n=14)
(n=14)

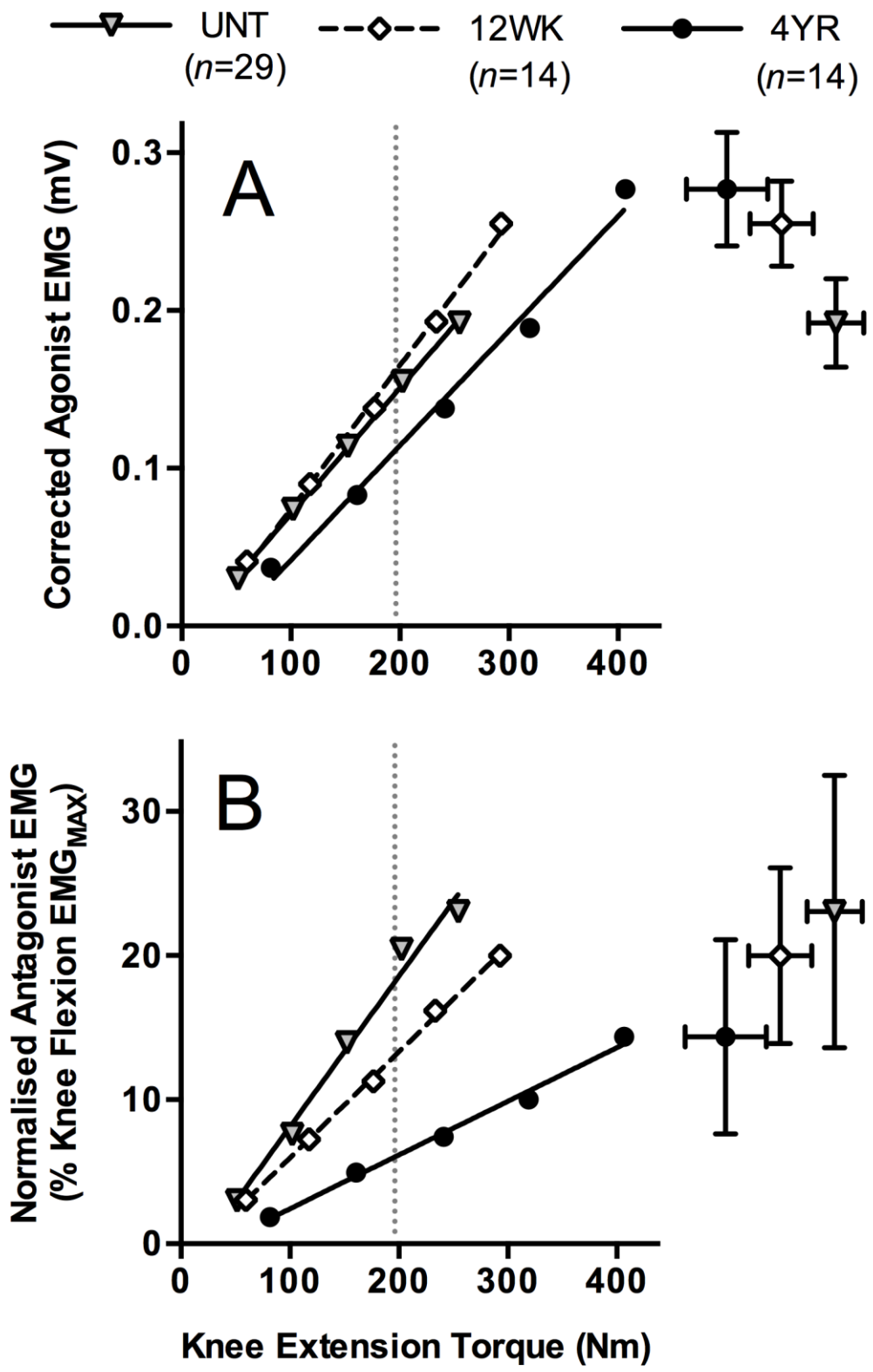

UNT
(n=29)
12WK
(n=14)
4YR
(n=14)
A
Corrected Agonist EMG (mV)
0.3
0.2
0.1
0.0
0
100
200
300
400
B
Normalised Antagonist EMG
(% Knee Flexion EMG$_{MAX}$)
30
20
10
0
0
100
200
300
400
Knee Extension Torque (Nm)

Figure 3

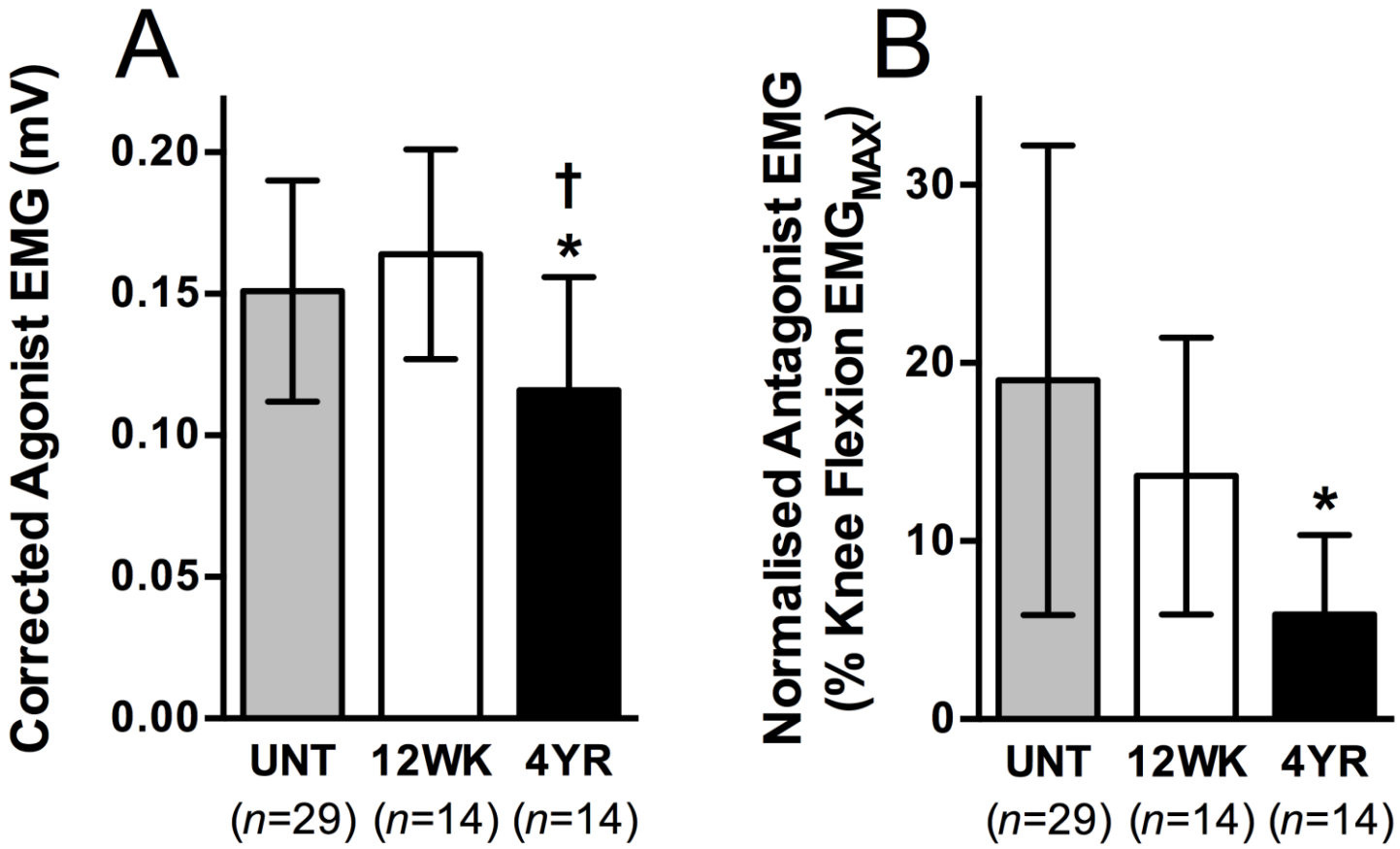

A
Corrected Agonist EMG (mV)
0.20
0.15
0.10
0.05
0.00
†
*
UNT
12WK
4YR
(n=29)
(n=14)
(n=14)
B
Normalised Antagonist EMG
(% Knee Flexion EMG$_{MAX}$)
30
20
10
0
*
UNT
12WK
4YR
(n=29)
(n=14)
(n=14)

Figure 4

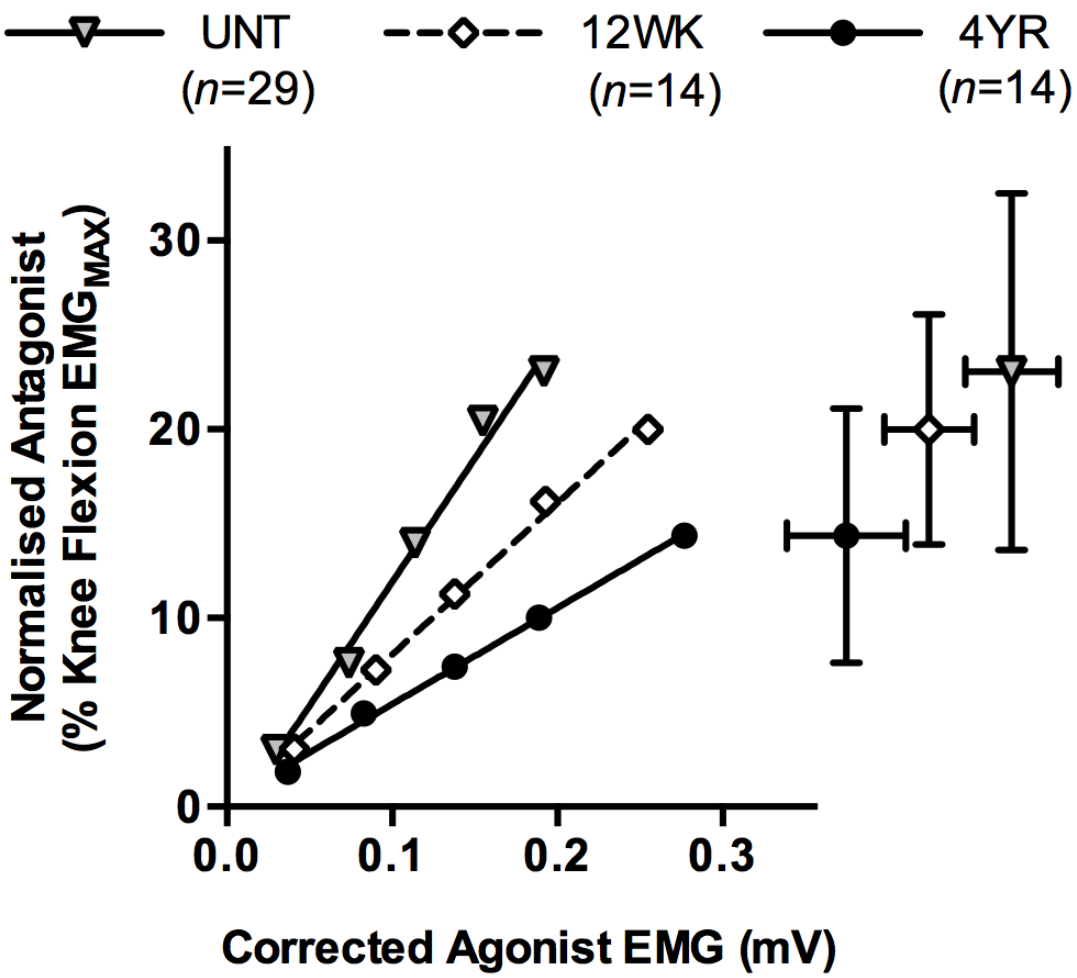

UNT
(n=29)
12WK
(n=14)
4YR
(n=14)
Normalised Antagonist
(% Knee Flexion EMG$_{MAX}$)
30
20
10
0
0.0
0.1
0.2
0.3
Corrected Agonist EMG (mV)

Figure 5