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Power Training–induced Increases in Muscle Activation during Gait in Old Adults

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ABSTRACT
BEIJERSBERGEN, C. M. I., U. GRANACHER, M. GÄBLER, P. DEVITA, and T. HORTOBÁGYI. Power Training–induced Increases in Muscle Activation during Gait in Old Adults. Med. Sci. Sports Exerc., Vol. 49, No. 11, pp. 2198–2205, 2017. Introduction/Purpose: Aging modifies neuromuscular activation of agonist and antagonist muscles during walking. Power training can evoke adaptations in neuromuscular activation that underlie gains in muscle strength and power but it is unknown if these adaptations transfer to dynamic tasks such as walking. We examined the effects of lower-extremity power training on neuromuscular activation during level gait in old adults. Methods: Twelve community-dwelling old adults (age ≥ 65 yr) completed a 10-wk lower-extremity power training program and 13 old adults completed a 10-wk control period. Before and after the interventions, we measured maximal isometric muscle strength and electromyographic (EMG) activation of the right knee flexor, knee extensor, and plantarflexor muscles on a dynamometer and we measured EMG amplitudes, activation onsets and offsets, and activation duration of the knee flexors, knee extensors, and plantarflexors during gait at habitual, fast, and standardized (1.25 ± 0.6 m s−1) speeds. Results: Power training-induced increases in EMG amplitude (−41%; 0.47 ≤ d ≤ 1.47; P ≤ 0.05) explained 33% (P = 0.049) of increases in isometric muscle strength (−43%; 0.34 ≤ d ≤ 0.80; P ≤ 0.05). Power training induced increases in plantarflexor activation during push-off (+11%; d = 0.38; P = 0.045) explained 57% (P = 0.004) of the gains in fast gait velocity (+4%; d = 0.31; P = 0.059). Furthermore, power training increased knee extensor activation (~18%; 0.26 ≤ d ≤ 0.29; P ≤ 0.05) and knee extensor coactivation during the main knee flexor burst (~41%; 0.26 ≤ d ≤ 0.44; P ≤ 0.05) at habitual and fast speed but these adaptations did not correlate with changes in gait velocity. Conclusions: Power training increased neuromuscular activation during isometric contractions and level gait in old adults. The power training–induced neuromuscular adaptations were associated with increases in isometric muscle strength and partly with increases in fast gait velocity. Key Words: WALKING, MUSCLE, EXERCISE, EMG

D isproportionally slow walking speed in old age predicts numerous medical, cognitive, and motor dysfunctions. Old adults older than 65 yr walking at a habitual speed ≥ 1.0 m s−1 tend to be healthier and have a higher functional status than those who walk slower (2). At any age, walking at or slower than 0.6 m s−1 substantially increases the risk for mobility disability, falls, and even for early mortality (2,36). As the number of old adults continues to rise, maintaining walking speed and delaying the onset of mobility disability are clinically important and have become universal health care priorities.

Slow walking speed, short steps, and a high cadence are visible hallmarks of age gait (8,21,23). Aging also affects the neuromuscular control of the lower-extremity muscles during gait. That is, old adults compared with young adults typically walk with greater levels of agonist activation (19) and particularly antagonist coactivation of the lower-extremity muscles (19,21,30,32). Disproportional coactivation can inhibit agonist muscle activation, reduce the net force generated (4,18), and increase the metabolic cost of transport (19,28,30). Nevertheless, elderly may use the increased coactivation to compensate for reductions in muscle strength and to stabilize joints (18).

Longitudinal studies showed that decline in maximal voluntary knee extensor activation contributes to muscle weakness before gait velocity starts to slow in old age (12) and high compared with low functioning old adults have
lower maximal voluntary plantarflexor activation (11). Power training incorporates exercises with moderately heavy weights and high movement velocities and can increase old adults’ muscle strength and power (3,5,10,31,34) as well as functional performance, including walking speed (10,20). Power training–induced increases in voluntary activation of the agonist muscle contribute to the gains in muscle strength and power (4,26). Such increases in voluntary activation of the knee extensors also correlated with increases in gait velocity in mobility-limited old adults (22). Whether or not power training improves muscle strength and power by reducing antagonist muscle activation is unclear because coactivation increased and also decreased after power training (4,10).

Much less is known about power training–induced adaptations in neuromuscular activation during gait. Twelve weeks of high-velocity heavy-resistance training (75%–80% of one-repetition maximum) increased stair ascent velocity but neuromuscular activation increased only in one of five recorded muscles, the rectus femoris, without changing coactivation (17). These data tentatively support the never tested idea that power training could induce changes in neuromuscular activation during level walking in old adults. We thus examined the effects of 10-wk of lower-extremity power training on gait velocity and neuromuscular activation of lower-extremity muscles during level walking in healthy old adults. We hypothesized that power training produces correlated improvements in gait velocity and increases in agonist activation or decreases in antagonist coactivation.

METHODS

Experimental design and participants. Data used in the present study are from participants enrolled in the Potsdam Gait Study and the study design, sample size calculation, and data collection methods have been detailed previously in the form of a study protocol (7). Participants were community-dwelling old adults age ≥ 65yr without mobility limitations (5,6). Twelve participants completed 10 wk of power training and subsequently 10 wk of detraining. Fourteen participants completed 10 wk of a control period, and three of these participants subsequently completed 10 wk of power training. Testing was performed at baseline, after 10 wk, and after 20 wk. We combined the three participants who conducted power training after they had completed the control period with the 12 participants who started with the power training, and we used this group of 15 participants to analyze power-training effects. All participants provided written informed consent before testing and the ethics committee of the University of Potsdam, Germany, approved the study protocol (reference number 40/2014) (7) that was conducted according to the ethical standards of the Helsinki Declaration.

Interventions. The lower-extremity power training program consisted of 30 sessions administered over 10 wk and was designed to improve lower-extremity power (7). Participants performed leg press, ankle press, knee extension, and knee flexion exercises. Participants exercised using bilateral movements and performed three sets of 6 to 10 repetitions at 40% to 60% of their biweekly measured three-repetition maximum. We instructed participants to lift the weights as rapidly and at high movement velocities during the concentric phase, as described in detail previously (5,6). Participants were instructed to return to or maintain their habitual levels of activity that was present before enrolling in the study for the control and detraining periods.

Data collection. According to guidelines of the International Society of Electrophysiology and Kinesiology (27), we recorded surface EMG activity in five muscles of the right leg, that is, vastus lateralis (VL), vastus medialis (VM), biceps femoris (BF), gastrocnemius medialis (GM), and soleus (SL). The skin was shaved, slightly abraded, degreased, and disinfected and electrodes affixed to the skin with the interelectrode resistance below 5 kΩ. Aligned parallel to the muscle fibers, we placed bipolar surface electrodes (Ambu®, type Blue Sensor P-00-S/S; Ag/AgCl, diameter: 13 mm, center-to-center distance: 25 mm, Ballerup, Denmark) on the muscle belly and a reference electrode was placed on the medial aspect of the tibia. We marked the electrode position with waterproof marker on the skin, and markers were regularly retraced during training sessions to enable precise electrode application in the post tests. The surface EMG signals were amplified, telemetrically transmitted (TeleMyo 2400G2; Noraxon, Scottsdale, AZ), converted to analog signals (TeleMyo 2400R G2; Noraxon) and synchronized with the ground reaction force by analog-to-digital conversion on the same A/D board and sampled at 1 kHz.

We recorded torque and EMG data during maximal voluntary contractions (MVC) of the knee flexors, knee extensors, and plantarflexors on an isokinetic dynamometer (Isomed 2000®, Hemau, Germany) (7). As a warm-up, participants performed a series of 10 submaximal isokinetic contractions, followed by three 3- to 5-s-long isometric MVC separated by 30 s of rest. During knee flexion/extension testing, participants were in a sitting position with the knee fixed at an angle of 45° in the dynamometer. During plantarflexion testing, participants were in a supine position with the ankle joint in neutral position and the knee extended. We selected the trial with the highest isometric torque value for further analysis.

We recorded surface EMG data while the participant walked on a 6.5 × 1.5-m level walkway and collected five gait trials at habitual, fast (“walk as fast and safely as you can, but do not run”), and standardized (1.25 ± 0.6 m s⁻¹) walking speed (7), 15 gait trials in total. The starting position was a taped line on the floor, and participants performed three practice trials to ensure participants stepped on the force platform with their right foot and without altering their gait pattern.

Data analysis. We performed two analyses on the EMG data, whereby one analysis focused on the timing of the agonist and antagonist EMG bursts and the second analysis determined the amplitude of the agonist and antagonist
muscle activation. For each gait trial, we time-normalized the EMG signals to stride durations and then performed a timing analysis. Using the Teager-Kaiser Energy Operator (25,33), we determined the relative onset and offset (% of stride) of the main EMG burst in each of the muscles. We averaged the timing of VL and VM to characterize the main knee extensor burst and averaged the timing of GM and SL to characterize the main plantarflexor burst. Next, we bandpass-filtered the raw-EMG (20–450 Hz) and applied a root mean square (RMS) envelope using a 40-ms smoothing window. We averaged the RMS-EMG of VL and VM to characterize knee extensor activity and averaged the RMS-EMG of GM and SL to characterize plantarflexor activity, a method used previously (16). The onset and offset points from the timing analysis were used as a window in which we determined the peak and mean RMS-EMG amplitudes of the agonist and antagonist muscles. We determined knee flexor coactivation during the main burst of the knee extensors and determined knee extensor coactivation during the main burst of the knee flexors (21). We also computed gait velocity from kinematic analysis, which we described in detail previously (5–7).

For the MVC trials, we also calculated the RMS-EMG using a 40-ms bin and determined peak RMS-EMG amplitude and mean RMS-EMG amplitude for a 1.0-s-long period centered on the peak RMS-EMG value. We used peak isometric torque as a measure of maximal muscle strength.

Statistical analysis. We report data as means and SD. For all gait variables, we used participants’ average of five trials per walking speed condition for the statistical analysis. EMGpeak and EMGmean values strongly correlated during the MVC and gait trials (r ≥ 0.8, P < 0.05) which is why we decided to present EMGmean values as a measure of EMG amplitude only. Main outcomes were muscle strength (N/m), gait velocity (m/s), muscle activation during strength and gait testing (mV), and timing of muscle activation during walking (% of stride). We used the Shapiro–Wilk test to confirm normality of data and analyzed all variables with a paired t test comparing pre–post values for power training and control. The Wilcoxon signed rank test was used when data was not normally distributed. We were unable to perform a repeated-measures ANOVA because three participants crossed over from the control to the power training intervention and (6). Within-group effect sizes (d) were calculated using z scores for Cohen d to ascertain if an effect was practically meaningful (13). According to Cohen, effect sizes can be classified as small (0.00 ≤ d ≤ 0.49), medium (0.50 ≤ d ≤ 0.79), and large (d ≥ 0.80) (13). We used simple linear regression analysis to predict changes in isometric torque from changes in agonist or antagonist EMG amplitudes. Additionally, we predicted changes in gait velocity from changes in agonist or antagonist EMG amplitudes. We quantified the associations between pairs of variables and between changes in variables as correlation coefficient (r value), level of significance (P value), and the amount of variance explained (r² value). Values of r = 0.10 indicate small, r = 0.30 medium, and r = 0.50 large size of correlation (13). We analyzed the data with SPSS 23.0 (SPSS Inc., Chicago, IL) and set the level of significance at P < 0.05.

RESULTS

Participants. We excluded three participants from the power-training group and one from the control group due to poor quality of the EMG signals. Thus, data from 12 participants were included for the analysis of the power training intervention (age, 72.1 ± 5.4 yr; BMI, 26.2 ± 4.1 kg/m²), and 13 for the control intervention (age, 69.7 ± 5.0 yr; BMI, 25.1 ± 3.2 kg/m²). An additional three participants dropped out from the detraining period leaving not enough participants remaining to statistically analyze detraining effects (n = 9) which is why we report results of the power training and control interventions only.

Isometric muscle strength and maximal EMG amplitude. Figure 1 shows the changes in maximal isometric strength and EMG amplitude of the agonist muscle. Power training increased isometric muscle strength of the knee flexors (15% ± 10%, d = 0.34, P = 0.002), knee extensors (22% ± 15%, d = 0.74, P ≤ 0.001), and plantarflexors (93% ± 101%, d = 0.80, P = 0.002) and also EMG amplitudes of the knee flexors (45% ± 39%, d = 1.47, P = 0.004), knee extensors (53% ± 61%, d = 0.50, P = 0.013), and plantarflexors (31% ± 56%, d = 0.47, P = 0.076). No changes occurred during the control period in isometric muscle strength (d ≤ 0.31, P ≥ 0.303) but EMG amplitude of the knee extensors increased by 61% ± 66% (d = 0.50, P = 0.021, Fig. 1). We observed no changes in knee flexor or knee extensor coactivation during MVC after power training or the control period (d ≤ 0.28, P ≥ 0.142, data not shown).

![EMG Amplitude and Isometric Strength](http://www.acsm-msse.org)
Gait velocity. Power training increased fast gait velocity by 3.5% ± 7.1% (d = 0.31, P = 0.059, Table 1) but habitual gait velocity was similar pre–post (+5.4% ± 11.9%, d = 0.42, P = 0.079). Habitual and fast gait velocity did not change after the control period (d ≤ 0.11, P ≥ 0.240) and standardized gait velocity was similar pre–post in both the power training and control group.

Muscle activation and coactivation during gait. The results of the EMG analysis were generally similar for walking at habitual and standardized speeds, and Table 1 shows only the changes in timing of agonist muscle activation at habitual and fast speeds. Power training delayed knee flexor offset at habitual (6.2% ± 8.4%, d = 0.66, P = 0.020) and fast speed (4.6% ± 5.6%, d = 0.58, P = 0.009), resulting in a longer duration of knee flexor activation at fast speed (21% ± 39%, d = 0.48, P = 0.016). Power training also delayed the offset of the knee extensors (3.7% ± 6.7%, d = 0.77, P = 0.040) and plantarflexors (0.7% ± 1.2%, d = 0.45, P = 0.040).

Figure 2 shows group average RMS envelopes of the EMG signals recorded at habitual and fast speeds for power training and control groups. Power training increased knee extensor (21% ± 26%, d = 0.26, P = 0.013) and plantarflexor (28% ± 45%, d = 0.65, P = 0.006) activation at habitual speed as well as knee extensor (16% ± 21%, d = 0.29, P = 0.022) and plantarflexor (11% ± 17%, d = 0.38, P = 0.045) activation at fast speed.

Figure 3 shows knee flexor and extensor coactivation during gait. Power training increased knee extensor coactivation during the main knee flexor burst at habitual (28% ± 33%, d = 0.44, P = 0.033) and fast (19% ± 22%, d = 0.26, P = 0.010) speed, whereas knee flexor coactivation during the main knee extensor was unchanged (d ≤ 0.39, P ≥ 0.271). Knee flexor or extensor coactivation during gait was unchanged after the control period (d ≤ 0.52, P ≥ 0.053, Fig. 3).

Correlation analyses. Power training–induced changes in isometric muscle strength correlated with overall changes in EMG amplitudes (r = 0.578, P = 0.049, n = 12, Fig. 4). We found no correlations between power training–induced changes in gait velocity and timing or magnitude of knee flexor or knee extensor activation or coactivation at habitual or fast speed. Measured at fast speed, power training–induced changes in gait velocity correlated with changes in plantarflexor offset (r = 0.662, P = 0.019, n = 12, Fig. 5A) and plantarflexor activation (r = 0.760, P = 0.004, n = 12, Fig. 5B).

DISCUSSION

We examined the effects of 10 wk of power training on lower-extremity neuromuscular activation during walking in old adults. In addition to gains in maximal EMG amplitudes and isometric strength, old adults showed elevated knee extensors activation and coactivation during early stance and elevated plantarflexor activation during push-off. The data suggest that the power training–induced increases in agonist muscle activation underlie the increases in isometric muscle strength and gait velocity. We discuss how power training modifies neuromuscular activation and increases old adults’ leg muscle strength and walking performance.
We previously reported that the present power-training program resulted in improvements in training loads (~51%, 0.56 ± 1.76) and isokinetic muscle power (~30%, 0.35 ± 0.71) (6). Despite that the power training involved dynamic exercise whereby elders moved moderately heavy weights rapidly during the concentric actions, power training also improved maximal isometric force (Fig. 1). These data confirm previous findings that power training interventions that progressively increase training intensity can improve healthy old adults’ leg muscle power and also muscle strength (6,10,14,24).

In the present study, the power training–induced gains in isometric muscle strength (~43%) and EMG amplitudes (~41%) were of similar magnitude (Fig. 1). Additionally, the gains in EMG amplitudes explained 33% of the variance of the gains in isometric strength (Fig. 4). These data agree with previous findings that short-term training-induced

![FIGURE 2—Group average RMS-EMG over a full gait cycle (0%–100%) recorded during walking at habitual and fast speeds in power training (left, n = 12) control (right, n = 13) groups. Knee flexor activation is represented by BF activation; knee extensor activation is represented by averaged VL and VM activation (VL + VM)/2; plantarflexor activation is represented by averaged GM and SL activation (GM + SL)/2. Dark gray areas indicate +1 or –1 standard deviation at pretest at habitual speed. Light gray areas indicate +1 or –1 standard deviation at pretest at fast speed. Vertical line at 40% gait cycle indicates heel strike. Muscle activation was calculated during the main agonist burst for knee flexors (~30%–55% gait cycle), knee extensors (~40%–70% gait cycle), and plantarflexors (~60%–100% gait cycle). *Significant change pre–post at habitual speed (P ≤ 0.05). †Significant change pre–post at fast speed (P ≤ 0.05).

![FIGURE 3—Antagonist coactivation during walking at habitual and fast speed for power training (n = 12) and control (n = 13) groups. Knee flexor coactivation was measured during the main knee extensor burst (~40%–65% of stride); Knee extensor coactivation was measured during the main knee flexor burst (~30%–55% of stride). Values are mean and standard deviations and expressed in millivolts (mV). *Significant change pre–post (P ≤ 0.05).]
increases in maximal isometric muscle strength can be largely accounted for by increased motor unit activation of the trained agonist muscle (4). Mechanism that underlie the increased neuromuscular activation can involve, but are not limited to, increased neural drive via corticospinal pathways, increased motor neuron and/or muscle fiber excitability, increased number of active motor units, and/or increased conduction velocity (1,26).

Muscle activation and coactivation during gait. Muscle activation and coactivation during gait are generally higher in old compared with young adults (19,21,30,32) and the increase in coactivation presumably contributes to joint stability (18). One study reported a strong association between power training–induced increases in maximal voluntary activation of the knee extensors during MVC and improvements in gait velocity in mobility limited old adults (22). However, the present study is the first to examine the effects of power training on muscle activation during walking and to relate the adaptations to changes in gait velocity. In general, the power training–induced adaptations in muscle activation around the knee were similar at habitual and fast speed (Table 1). After training but not the control period, knee flexor activation became longer and the percentage of stride during which knee flexors and extensors were simultaneously active increased. Despite the temporal changes in knee flexor activation, power training did not change the magnitude of knee flexor activation or coactivation. In contrast, after training knee extensor activation and coactivation increased (Figs. 2 and 3). In line with the concept that increased coactivation is important for joint stability, we interpreted the increased magnitudes of knee extensor activation (~18%) and coactivation (~24%) around heel strike as a mechanism to stiffen the knee joint during heel strike and loading response. Noticeable is that power training–induced adaptations in neuromuscular activation of the knee flexors or extensors (i.e., timing or magnitude) only played a minor role in improving gait velocity, because these adaptations did not correlate with gait velocity.

Power training increased plantarflexor activation during push-off (Fig. 2) and delayed activation-offset at fast gait speed. Indeed, the elders walked 5% faster, delayed their plantarflexor offset by 1%, increased their plantarflexor activation during push-off by 10%, and the gains in plantarflexor offset and activation explained 44% to 57% of the gains in fast walking speed (Fig. 5). These data only tentatively support the idea that improved plantarflexor muscle activation acted as an enabler mechanism for an improved fast walking speed after power training in old adults. Paradoxically, we previously showed that the present power training intervention resulted in an 8% decrease in plantarflexor velocity during push-off (6) and an 11% reduction in ankle peak power during push-off (5). These discrepant results suggest a disassociation between the different types of measures (i.e., kinematics, kinetics, EMG) and future research is needed to clarify the role of each type of measure and their interaction for quantifying training improvements.

FIGURE 4—Association between power training–induced changes in maximal isometric torque and changes in EMG amplitude. Each data point represents an old adult and the data points were created by averaging knee flexor, knee extensor, and plantarflexor isometric torque or EMG amplitude ((ΔKF + ΔKE + ΔPF)/3). The association is characterized by $y = 1.04x + 6.9$, $R^2 = 0.33$ ($P = 0.049$).

FIGURE 5—Associations between power training–induced changes in fast gait velocity and changes in plantarflexor offset or activation. Panel A: Association between changes in fast gait velocity and changes in plantarflexor offset. The association is characterized by $y = 0.11x + 0.3$, $R^2 = 0.44$ ($P = 0.019$). Panel B: Association between changes in fast gait velocity and changes in plantarflexor activation. The association is characterized by $y = 1.83x + 4.5$, $R^2 = 0.57$ ($P = 0.004$).
effects on gait performance in both clinical and research settings. Furthermore, research should explore the role of additional gait and/or dynamic balance training providing real-time feedback of the propulsive force so that old adults can learn to optimize the use of the newly acquired levels of plantarflexor activation (15). Other factors that may limit the ability to incorporate the improved plantarflexor activation capacity are the age-related architectural changes in the ankle muscle–tendon complex (29,35).

Relative muscle activation. The power training–induced gains in muscle activation differed between tasks (i.e., MVC vs. gait) and between muscle groups, which is why we presented our EMG data in absolute units. Nevertheless, expressing muscle activation during gait as a percent of maximal EMG amplitude during MVC (%MVC) provides information about the level of maximal available capacity that old adults use during gait. The gains in knee flexor activation were greater during MVC (45%) compared to gait (~1%) and consequently, the elderly walked at lower levels of their maximal knee flexor EMG capacity after training (42–27 %MVC at habitual speed and 70–46 %MVC at fast speed, both P ≤ 0.05). Power training did not change the level of knee extensor activation at habitual and fast testing speed (32–28 %MVC and 57–44 %MVC, both P ≤ 0.05). Additionally, the level of plantarflexor activation during gait was 108 %MVC at habitual speed and 180 %MVC at fast speed and further analysis showed that power training did not change these levels (P > 0.05). The above-maximal level is probably caused by the difference in joint positions and type of contraction between the MVC and gait tests. Nonetheless, these data suggest that old adults walk with relatively high levels of plantarflexor activation, a level that power training did not reduce. Overall, old adults exploited the power training–induced increases in maximal knee extensor and plantarflexor muscle activation.

Limitations and conclusion. One limitation of the current study is that the old adults were without mobility limitations, which may have reduced the effectiveness of the power training to improve gait velocity (9) and neuromuscular activation during gait. Second, although EMG is a useful tool for assessing neuromuscular control, a variety of non-neural factors can influence the signal, including the amount of subcutaneous adipose tissue and the consistency of electrode placement between tests. Finally, due to inconvenience and time constrains, we did not measure EMG activity of any hip extensors (i.e., gluteus maximus) or hip flexors (i.e., rectus femoris), and future studies should determine if power training increases hip muscle activation during gait.

In conclusion, we observed that a 10-wk power-training program produced substantial improvements in agonist muscle activation during isometric actions and during walking in old adults. The power training–induced increases in agonist muscle activation may in part mediate the increases in isometric strength and fast gait velocity. The present results support the use of power training for improving neuromuscular activation and functional performance in old adults.

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