Chapter 5

Hip mechanics underlie lower extremity power training-induced increase in old adults' fast gait velocity: The Potsdam Gait Study (POGS)

Chantal Beijersbergen
Urs Granacher
Martijn Gäbler
Paul DeVita
Tibor Hortobágyi

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Abstract

Background: Aging is associated with slowed gait and old compared with young adults generally walk with greater positive hip work (H1) and reduced positive ankle work (A2). The role of exercise interventions on old adults' gait mechanics that underlie training-induced improvements in gait velocity is unclear. We examined the effects of lower extremity power training and detraining on old adults' gait kinetics. Methods: As part of the Potsdam Gait Study (POGS), healthy old adults completed a no-intervention control period (69.1 ± 4.4 yrs, n = 14) or a power training program followed by detraining (72.9 ± 5.4 yrs, n = 15). We measured isokinetic knee extensor and plantarflexor power and measured hip, knee and ankle kinetics at habitual, fast and standardized walking speeds. Results: Power training significantly increased isokinetic knee extensor power (25%), plantarflexor power (43%), and fast gait velocity (5.9%). Gait mechanics underlying the improved fast gait velocity included increases in hip angular impulse (29%) and H1 work (37%) and no changes in positive knee (K2) and A2 work. Detraining further improved fast gait velocity (4.7%) with reductions in H1 (-35%), and increases in K2 (36%) and A2 (7%). Conclusion: Power training increased fast gait velocity in healthy old adults by increasing the reliance on hip muscle function and thus further strengthened the age-related distal-to-proximal shift in muscle function.

5.1. INTRODUCTION

Even healthy aging modifies locomotion: steps become shorter, cadence increases, and walking speed slows [1,2]. Delaying gait-slowing in old age is important because old adults who walk just 0.1 m/s slower for their age and gender have a greater risk for developing mobility disability and adverse health outcomes [3]. Of the numerous age-related changes in neuromuscular function [4], reductions in joint moments and powers at hip, knee, and ankle joints underlie gait-slowing in old age [5]. However, when walking at the same speed, these reductions are not uniform across joints: old adults increase hip extensor power and work in compensation for reductions in ankle plantarflexor power and work during push-off [5–7].

Lower extremity muscle power is a predictor of mobility, including gait velocity, in old age [8–10], and is a better predictor of functional performance than lower extremity muscle strength [9]. Power training seems as an ideal choice of intervention because it can increase both force and velocity of muscle contraction [11,12] and also gait velocity [13–15].

Much less is known about the biomechanical mechanisms that underlie the coupling between increases in muscle power and gait velocity. According to the never tested idea, power training would increase maximal muscle power, which old adults would use during gait by generating higher rotational forces, joint powers, joint work, and increase the center of mass velocity [16]. One element of this mechanism could be the adaptation in joint kinematics, as power training increases old adults' ankle ROM during gait by increasing maximal plantarflexor position in late stance [17,18]. However, there is no evidence that power training improves joint kinetics measured during gait and if such increases correlate with changes in gait velocity. Although detraining decreases lower extremity muscle power [13], it is also unknown if the withdrawal of the exercise stimulus would mechanistically and reciprocally re-modify joint kinetics measured during gait and would concomitantly reduce gait velocity to the levels measured in the untrained state before the intervention.

In the present study, we examined the effects of lower extremity power training and detraining on healthy old adults' gait kinetics. We hypothesized that the training-induced increases in lower extremity muscle power would favorably modify old adults' gait kinetics by increasing joint moments and powers ultimately leading to faster walking. We expect detraining to reverse these adaptations. We also examined the relationship between changes in gait velocity and changes in isokinetic power and joint work during gait to determine if adaptations occur in a correlated manner.

5.2. METHODS

5.2.1. Study design and participants

Data used in the present study are from participants enrolled in the Potsdam Gait Study (POGS) and characterized as community-dwelling old adults aged ≥ 65 without mobility

limitations [19,20]. Twelve participants completed 10 weeks of power training with subsequently 10 weeks of detraining. Fourteen participants completed 10 weeks of a control period and three of these participants subsequently completed 10 weeks of power training. Testing was performed at baseline, after 10, and after 20 weeks. All participants provided written consent before testing and the ethics committee of the University of Potsdam, Germany, approved the study protocol (reference number 40/2014) that was conducted according to the ethical standards of the Helsinki Declaration.

5.2.2. Interventions

The lower extremity power training program consisted of 30 sessions administered over 10 weeks and focused on improving lower extremity power [19]. Participants performed leg press, ankle press, knee extension, and knee flexion exercises at 40–60% of the three-repetition maximum and were instructed to move the weights as rapidly and explosively as possible during the concentric phase, as described in detail previously [19,20]. 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.

5.2.3. Data collection

We measured maximal isokinetic power of the right knee extensors and plantarflexors using an isokinetic dynamometer (Isomed 20001, Hemau, Germany) [19]. Knee extensor power was tested at 60, 120, and 180 °/s and plantarflexor power at 20, 40, and 60 °/s as detailed previously [19].

Participants walked on a 6.5 1.5-m level walkway and wore a pair of tight shorts, t-shirt, and their own athletic shoes. We collected five gait trials at a habitual, fast ("Walk as fast and safely as you can, but do not run"), and standardized (1.25 ± 0.6 m/s) walking speed [19], fifteen gait trials in total. The starting position was a taped line on the floor and participants performed three practices trials to ensure participants stepped on the force platform with their right foot and without altering their gait pattern.

We affixed 18 reflective markers on the right foot, shank, thigh, and pelvis (2nd and 5th metatarsal head, anterior foot, posterior calcaneus, lateral malleolus, lateral shank, lateral epicondyle of the femur, lateral thigh, anterior superior iliac spine, posterior superior iliac spine). Participants walked with their right leg over an AMTI force platform (Watertown, Massachusetts, USA) and we captured 3D marker kinematics at 100 Hz using nine infrared cameras (Vicon, Denver, USA) and ground reactions forces and moments of force (1 kHz, gain of 4000).

5.2.4. Data analysis

We used standardized methods to reduce the kinematic and kinetic data [19]. We filtered kinematic data with a 4th order low- pass Butterworth filter at 6 Hz and ground reaction

forces with a 2nd order low-pass Butterworth filter at 45 Hz. We modelled the right lower extremity as a rigid, linked-segment system and performed inverse-dynamics gait analysis using Visual 3D (CMotion Inc., Rockville, Maryland, USA). We computed sagittal plane joint moments and powers at the hip, knee, and ankle joints during one stride (right toe off to right toe off) and normalized kinetic data to body mass and height. We extracted peak joint moments and joint angular impulses during hip extensor moment in early stance, knee extensor moment in early stance, and plantarflexor moment in late stance. We extracted peak joint powers and joint work during positive hip extensor power in early stance (H1), negative knee extensor power in early stance (K1), positive knee extensor power in mid-stance (K2), and positive plantarflexor power in late stance (A2). Positive power refers to concentric contractions and energy generation, whereas negative power refers to eccentric contractions and energy absorption. We calculated the total positive work performed as the sum of H1, K2, and A2 positive work and expressed the relative work performed during each power phase as percentage of total work.

5.2.5. Statistical analysis

We report means ± SDs. For each participant, we used the average of five gait trials for each walking speed in the statistical analysis. Of the isokinetic trials, the trial that produced the highest muscle power for each testing speed at each joint was included in the statistical analysis. We used the Shapiro-Wilk test to confirm normality of the data and analyzed all variables with a paired t-test comparing pre-post values for power training, detraining, and control. The Wilcoxon Signed Rank Test was used when data was not normally distributed. Two of 30 kinetic variables showed a significant change in the mean values after the control period (Table C.1 in Appendix C), suggesting that any change in gait kinetics was due to the power training or detraining, unbiased by a repeated testing or learning effect. We used simple linear regression analysis to predict changes in joint powers from changes in isokinetic power and changes in gait velocity from changes in kinetic variables. We analyzed the data with SPSS 23.0 (SPSS Inc., Chicago, IL) and set the level of significance at $P < 0.05$.

5.3. RESULTS

At baseline, participants in the power training, detraining, and control groups were statistically and functionally similar in six of seven variables (Table C.2 and [20]).

5.3.1. Maximal isokinetic power

Fig. 5.1 summarizes the changes in maximal isokinetic power in the three groups. Power training increased knee extensor and plantarflexor isokinetic power across all contraction velocities ($P < 0.001$). Maximal isokinetic power remained unchanged after detraining and the control period ($P > 0.05$).

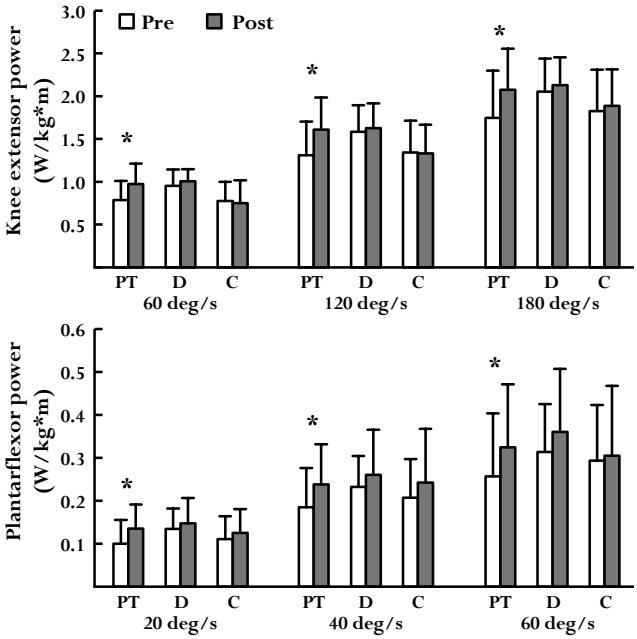

Figure 5.1. Maximal isokinetic power for the knee extensors (top) and plantarflexors (bottom) across three contraction velocities. Values are mean ± SD. PT; Power training (n = 15), D; Detraining (n = 12), C; Control (n = 14). *Significant change pre-post.

5.3.2. Gait velocity

There was no change in habitual or standardized gait velocity after power training, detraining, or control (Table C.3). Power training increased fast gait velocity by 5.9% (1.85 ± 0.28 to 1.95 ± 0.38 m/s, P = 0.026) and detraining additionally increased fast gait velocity by 4.7% (1.91 ± 0.22 to 2.00 ± 0.25 m/s, P = 0.052). We described in detail the changes in muscle power and gait kinematics previously [20].

5.3.3. Joint moments

We do not present the joint kinetics data at standardized speed because the changes were similar to those at habitual speed. Table 5.1 summarizes intervention-induced changes in joint moments at habitual and fast speed. Measured at habitual speed, power training increased hip extensor peak moment (18%, P = 0.072) and angular impulse (34%, P = 0.083), and significantly decreased knee extensor peak moment (15%) and angular impulse (25%). Measured at fast speed, power training increased hip extensor peak moment (17%, P = 0.019) and angular impulse (29%, P = 0.070), and significantly decreased knee extensor angular impulse (11%) and plantarflexor peak moment (4.8%) (Fig. 5.2). Measured at habitual and fast speed, detraining decreased hip extensor peak moment (20%, P = 0.002, and 8%, P = 0.065), and increased knee extensor angular impulse (62% and 48%, P < 0.05).

5.3.4. Joint powers

Table 5.2 summarizes intervention-induced changes in joint powers. Power training did not affect joint powers measured at habitual speed. Measured at fast speed, power training

increased H1 peak power (31%, P = 0.061) and work (37%, P = 0.088), decreased K1 peak power (11%, P = 0.088) and work (25%, P = 0.071), and decreased A2 peak power (11%, P = 0.002) (Fig. 5.2). Measured at habitual and fast speed, detraining significantly decreased H1 peak power (33% and 27%) and work (33%, and 35%). Measured at fast speed, power training significantly increased K1 peak power (50%) and work (50%), and significantly increased K2 and A2 work (36% and 6.7%, respectively) (Fig. 5.2).

At baseline, individual joints contributed to total work 25 (H1), 15 (K2), and 60% (A2) (Table 5.3). Measured at fast speed, power training increased the relative contribution of H1 by 4% (P = 0.034) and non-significantly decreased the relative contribution of K2 and A2 by 2 and 3%. Measured at habitual and fast speed, detraining decreased the relative contribution of H1 (~10%) and increased the relative contribution of K2 (~4%) and A2 (~6%) (all $P < 0.05$, Table 5.3).

5.3.5. Correlation analyses

At baseline and in the power training group, habitual gait velocity correlated with knee extensor ($R^2 > 0.29$, $P < 0.05$), but not with plantarflexor isokinetic power ($R^2 < 0.22$, $P > 0.05$), while fast gait velocity correlated with both knee extensor and plantarflexor isokinetic power ($R^2 > 0.40$, $P < 0.05$). Changes in isokinetic power did not correlate with changes in habitual ($R^2 < 0.13$, $P > 0.05$) or fast ($R^2 < 0.06$, $P > 0.05$) gait velocity (Table C.4). At baseline and in the power training group, habitual gait velocity correlated with H1, K2 and A2 work ($R^2 > 0.29$, $P < 0.05$) and fast gait velocity correlated with H1 and K2 work ($R^2 > 0.44$, $P < 0.05$), but not with A2 work ($R^2 = 0.19$, $P = 0.105$). Changes in habitual or fast gait velocity did not correlate with changes in H1, K2, or A2 work ($R^2 < 0.208$, $P > 0.05$, Table C.5).

5.4. DISCUSSION

Ten weeks of power training increased lower extremity muscle power and fast but not habitual gait velocity in community-dwelling old adults. We partially confirm the hypothesis that the mechanism underlying power training-induced increases in gait velocity involve increases in the mechanical output at the hip joint. Ten weeks of detraining did not reduce lower extremity muscle power but further increased fast gait velocity. The data support the hypothesis that detraining reversed the power training-induced adaptations in joint kinetics marked by decreased hip and increased knee and ankle mechanical outputs. These results are the first demonstrations of how an exercise stimulus and its removal can cause reciprocal adaptations in community-dwelling old adults' gait kinetics.

Lower extremity muscle power is a predictor of mobility, including gait velocity [8–10], and our results extend these findings, as knee extensor and plantarflexor muscle power explained 40–57% of the variance in fast gait velocity (Table C.4). One possibility why knee extensor but not plantarflexor power correlates with habitual gait velocity (R^2

Table 5.1. Peak joint moments and angular impulse variables normalized to body mass and height before and after power training (n = 15) and detraining (n = 12) at habitual and fast walking speeds.

	Power training						Detraining					
	Habitual			Fast			Habitual			Fast		
	Pre	Post	P	Pre	Post	P	Pre	Post	P	Pre	Post	P
Joint moment, Nm/kg*m												
HM	0.46(0.14)	0.51(0.13)	0.072	0.73(0.24)	0.82(0.25)	**0.019**	0.53(0.13)	0.42(0.11)	**0.002**	0.81(0.22)	0.72(0.20)	0.065
KM	0.39(0.12)	0.33(0.18)	**0.036**	0.60(0.21)	0.56(0.25)	0.185	0.29(0.13)	0.36(0.16)	0.081	0.50(0.21)	0.62(0.18)	**0.009**
AM	0.95(0.07)	0.97(0.06)	0.096	0.97(0.10)	0.93(0.12)	**0.025**	0.98(0.06)	0.97(0.07)	0.502	0.96(0.11)	0.93(0.12)	0.225
Angular impulse, Nms/kg*m												
HM	0.06(0.03)	0.07(0.03)	0.083	0.07(0.03)	0.08(0.03)	0.070	0.08(0.04)	0.05(0.03)	**0.011**	0.08(0.03)	0.06(0.03)	**0.014**
KM	0.07(0.03)	0.05(0.04)	**0.016**	0.08(0.04)	0.06(0.03)	**0.047**	0.04(0.03)	0.06(0.04)	**0.039**	0.05(0.03)	0.07(0.03)	**0.008**
AM	0.45(0.04)	0.45(0.04)	0.353	0.45(0.03)	0.44(0.04)	0.113	0.45(0.03)	0.45(0.02)	0.637	0.45(0.03)	0.44(0.04)	0.440

Values are mean (±SD). HM; hip extensor moment in early stance, KM; knee extensor moment in early stance, AM; plantarflexor moment in late stance (See also Fig. 5.2). We refer to Table C.1 in Appendix C for joint moment variables pre-post the control period. Significant P values are denoted in bold.

Table 5.2. Peak joint powers and work variables normalized to body mass and height before and after power training (n = 15) and detraining (n = 12) at habitual and fast walking speeds.

	Power training						Detraining					
	Habitual			Fast			Habitual			Fast		
	Pre	Post	P	Pre	Post	P	Pre	Post	P	Pre	Post	P
Joint power, W/kg*m												
H1	0.46(0.23)	0.52(0.29)	0.161	0.82(0.53)	1.02(0.73)	0.061	0.57(0.30)	0.34(0.22)	**0.003**	0.90(0.37)	0.65(0.39)	**0.019**
K1	-0.47(0.27)	-0.43(0.37)	0.230	-1.08(0.57)	-0.93(0.44)	0.088	-0.34(0.21)	-0.44(0.30)	0.197	-0.83(0.43)	-1.14(0.46)	**0.005**
K2	0.35(0.21)	0.32(0.19)	0.159	0.78(0.48)	0.79(0.60)	0.451	0.29(0.15)	0.32(0.19)	0.419	0.66(0.37)	0.80(0.36)	0.097
A2	2.02(0.44)	1.99(0.46)	0.410	2.57(0.73)	2.26(0.63)	**0.002**	2.06(0.46)	2.15(0.76)	0.458	2.34(0.66)	2.51(0.90)	0.107
Joint work, J/kg*m												
TW	0.25(0.07)	0.25(0.07)	0.363	0.34(0.11)	0.34(0.11)	0.412	0.25(0.07)	0.25(0.07)	0.363	0.33(0.10)	0.32(0.09)	0.170
H1	0.06(0.04)	0.07(0.05)	0.140	0.08(0.06)	0.10(0.06)	0.088	0.08(0.05)	0.04(0.04)	**0.014**	0.10(0.06)	0.06(0.06)	**0.008**
K1	-0.03(0.02)	-0.03(0.02)	0.340	-0.06(0.03)	-0.05(0.02)	0.071	-0.02(0.01)	-0.02(0.02)	0.388	-0.04(0.02)	-0.06(0.02)	**0.004**
K2	0.03(0.02)	0.03(0.02)	0.129	0.05(0.03)	0.05(0.04)	0.445	0.02(0.02)	0.03(0.02)	0.167	0.04(0.03)	0.06(0.03)	**0.049**
A2	0.15(0.04)	0.15(0.03)	0.342	0.20(0.07)	0.19(0.05)	0.139	0.15(0.04)	0.16(0.05)	0.462	0.19(0.06)	0.20(0.07)	**0.021**

Values are mean (±SD). H1; positive hip extensor power in early stance, K1; negative knee extensor power in early stance, K2; positive knee extensor power in mid-stance, A2; positive plantarflexor power in late stance (See also Fig. 5.2). TW; Total positive work. We refer to Table C.1 in Appendix C for joint power variables pre-post the control period. Significant P values are denoted in bold.

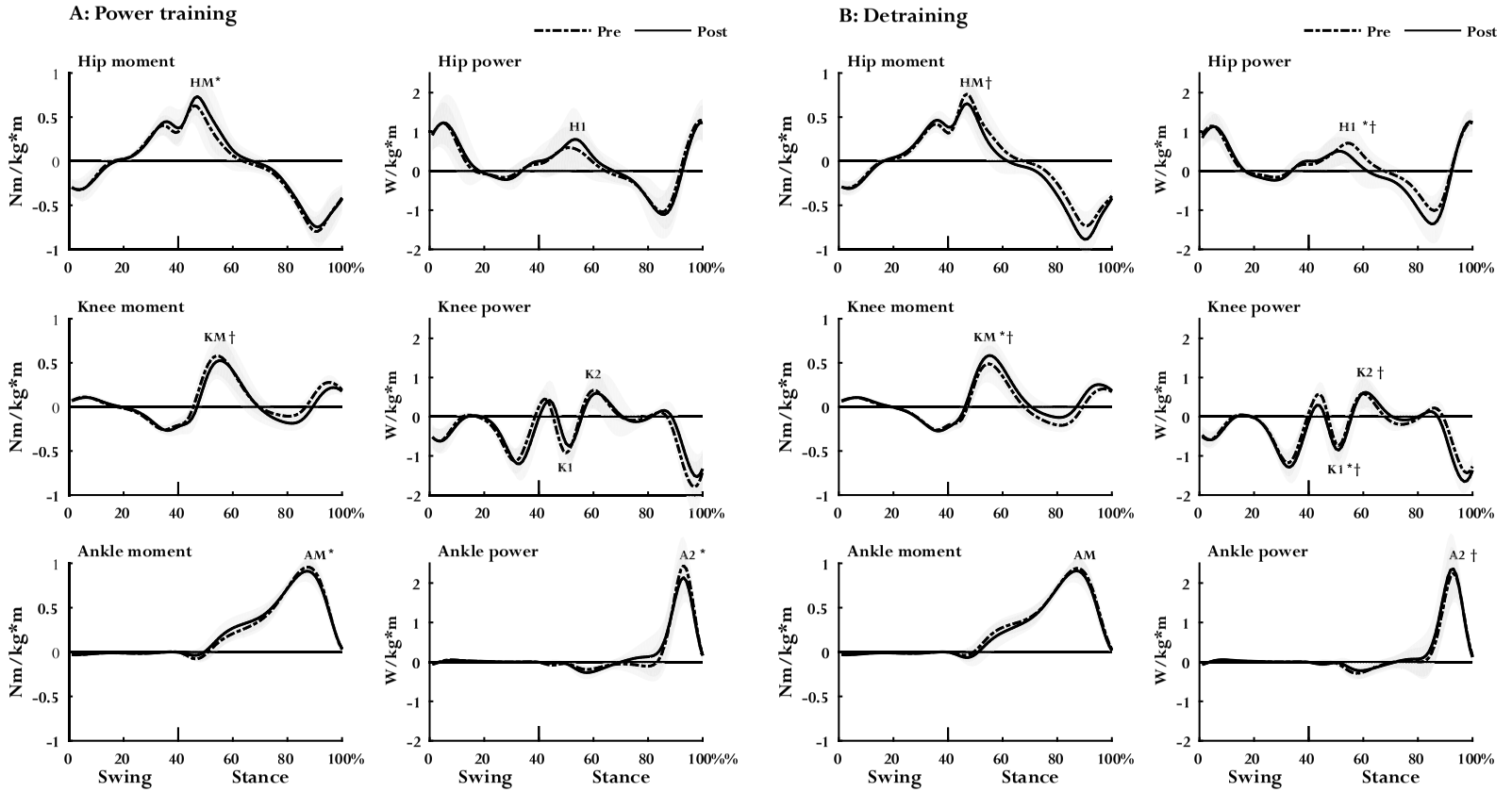

Figure 5.2. Mean joint moment and power curves averaged over a full gait cycle (0–100%) pre- and post-power training (Panel A, n = 15) and detraining (Panel B, n = 12) at fast speed. Vertical line at 40% gait cycle indicates heel strike. Gray areas indicate +1 and -1 standard deviation. HM; hip extensor moment in early stance, KM; knee extensor moment in early stance, AM; plantarflexor moment in late stance, H1; positive hip extensor power in early stance, K1; negative knee extensor power in early stance, K2; positive knee extensor power in mid-stance, A2; positive plantarflexor power in late stance. Positive values indicate extensor or plantarflexor moment and power generation. Negative values indicate flexor or dorsiflexor moment and power absorption. *Significant change in peak moment or power. †Significant change in angular impulse or work.

Table 5.3. Relative joint work before and after power training (n = 15) and detraining (n = 12) at habitual and fast walking speeds.

		Power training			Detraining		
		Pre	**Post**	**P**	**Pre**	**Post**	**P**
Habitual							
	H1	22.6 (11.1)	26.5 (14.5)	0.104	29.3 (14.8)	18.7 (14.4)	**0.011**
	K2	13.9 (5.9)	11.9 (8.3)	0.119	9.7 (6.6)	13.1 (7.6)	**0.036**
	A2	63.5 (8.0)	61.6 (9.7)	0.180	61.0 (10.5)	68.2 (10.9)	**0.033**
Fast							
	H1	23.3 (12.8)	27.5 (12.4)	**0.034**	28.6 (12.6)	19.6 (13.9)	**0.004**
	K2	16.4 (6.2)	14.9 (8.6)	0.164	13.4 (8.7)	17.3 (7.6)	**0.013**
	A2	60.3 (9.7)	57.6 (10.2)	0.103	57.9 (8.4)	63.1 (9.2)	**0.035**

Values are mean (±SD) and presented as % of total positive work. H1; positive hip extensor power in early stance, K2; positive knee extensor power in mid-stance, A2; positive plantarflexor power in late stance (See also Fig. 5.2). Significant P values are denoted in bold.

= ~0.30) is because knee extension velocity during gait (~80 °/s [20]) is within the range of isokinetic test velocities (60–180 °/s), while the plantarflexor velocity during gait (~270 °/s [20]) was outside the range of the isokinetic test velocities (20–60 °/s). The ~34% increase in muscle power in the present study are in the range of changes reported previously [14,15], confirming the responsiveness of healthy old adults' skeletal muscles to such an exercise stimulus across a range of contraction velocities. The lack of correlation between changes in muscle power and changes in habitual or fast gait velocity ($R^2 < 0.10$, $P > 0.05$) confirm our previously published hypothesis that old adults incorporate only a small portion of the training- induced muscle power gains into gait [16].

K2 work compared with H1 and A2 work was the strongest predictor of fast gait velocity and explained 50% of the variance in gait velocity. Increases in knee extensor strength after 12 weeks of strength training in healthy old adults explained 44% of the increases in stride length and the authors assumed that better knee extensor function improved gait velocity [18]. This assumption contrasts with our results showing that although power training decreased knee extensor output by 3.2% and negative K1 work by 8.2%, the positive K2 work that extends the knee in mid stance, did not change (Table 5.2, Fig. 5.2). Furthermore, we found no relationship between changes in gait velocity and changes in knee extensor muscle power (Table C.4) and K2 work (Table C.5). The relative contribution of the knee extensor muscles to the total positive work is small (~15%) and we show that despite power training inducing large improvements in maximal knee extensor power (Fig. 5.1), the relative contribution of the knee extensors (Table 5.3) or knee extensor function in general during gait did not change. All in all, these data counter the prevailing view that prescribes a putative role to knee extensor function in fending of mobility disability [18].

Although plantarflexor muscle power is a predictor of old adults' plantarflexor power during walking [21] and the 19% age-related reduction in A2 work [16] is a key intervention target [7], we observed paradoxical adaptations in ankle function. Power

training caused a redistribution of plantarflexor moment and power generation from push-off to mid-stance, whereby plantar- flexor moment and power in mid-stance increased and peak plantarflexor moment and power in late stance decreased (Fig. 5.2). The 11% reduction in A2 peak power is possibly the consequence of the reduction in both plantarflexor peak moment (-4.8%) and plantarflexor velocity during push-off (-7.9% [20]). Taken together, old adults only minimally capitalized on the ~43% increase in maximal plantarflexor muscle power, as this large increase did not lead to a more forceful and powerful push-off. These data suggest that in addition to improving plantarflexor muscle power, old adults may need an enabling or a learning mechanism that facilitates the use of the newly acquired abilities in gait. One possibility is to supplement power training with gait training by providing real-time feedback of the propulsive force [22]. Other factors that may limit the ability to incorporate the improved plantarflexor capacity are the age-related architectural changes in ankle muscle-tendon complex [23,24], neural activation and velocity impairments of these muscles [25], or agonist-antagonist coactivation [26].

Faster walking generally requires greater H1, K2, and A2 work [7,21] and positive work generated at the hip and ankle generally contributes to the forward propulsion of the body. Aging modifies the relative contribution of H1 and A2 work to the total positive work, whereby healthy old compared with young adults walk with relatively more H1 and less A2 work [6,7]. Instead of reshaping gait mechanics toward a young pattern and correcting the reduced A2 work, lower extremity power training increased the relative contribution of H1 work, an adaptation also observed in response to six weeks of lower extremity strength training in patients with knee OA [27]. It is not clear why interventions would further increase the reliance on hip extensor function. One possibility is that old adults chose the strategy of higher cadence more so than longer strides to improve gait velocity. Indeed, A2 compared with H1 power is a better predictor of step length in healthy old adults ($R^2 = 0.52$ vs. $R^2 = 0.04$) [2] and we previously reported that changes in cadence explained 75% ($P < 0.001$) of the variance in changes in fast gait velocity versus 24% ($P = 0.066$) of the variance explained by stride length [20].

Power training increased the hip extensor output generated in early stance by exerting a greater net hip extensor moment resulting in a greater H1 power and larger H1 work generation (Fig. 5.2). Old adults thus increased their hip function during early stance presumably resulting in greater hip stabilization during loading response and more forward propulsion of the body. The unchanged K2 and A2 work suggest that the increase in H1 work is the driving factor in the power training-induced increase in fast gait velocity. We indeed found that old adults rely more on their hip muscles during gait after power training (Table 5.3), however we inexplicably found no correlation between changes in gait velocity and changes in H1 work (Table C.5). Additionally, the unchanged total work together with increases in gait velocity reflects that perhaps mechanisms other

than the ones we examined here contributed to the training-induced increases in fast gait velocity such as increased medio-lateral stability, optimized energy consumption, or reduced antagonist muscle coactivation. Nevertheless, our results clearly show that old adults performed more work in early and mid-stance, reflected by increased H1 work and more gradually distributed A2 work.

Withdrawal of the exercise stimulus did not decrease muscle power (Fig. 5.1) and the further increase in fast gait velocity is remarkable and suggests that old adults use factors other than improved muscle power to increase gait velocity [20]. Additionally, the increase in fast gait velocity after detraining points to the involvement of a different mechanism that acted during training because gait velocity increased after detraining while we observed reciprocal adaptations in joint kinetics after power training (Fig. 5.2, Table 5.3).

One limitation of the present study is that the community-dwelling old adults were healthy without mobility limitations and with a relatively good muscle function at baseline, a factor that limited the effectiveness of the power training to increase habitual gait velocity. Future studies should confirm the present results and extend the data to frail elderly. Second, we were unable to reliably measure isokinetic hip power on the dynamometer and future studies will determine if and how much of the increase in maximal hip power in old adults can be incorporated into hip joint power during gait. Third, we found small effect sizes for kinetic variables (not reported) and future studies should involve larger sample sizes to enhance statistical power. Finally, the biomechanical analysis does not take into consideration the actual muscle forces, but only the net joint moments caused by muscles acting to control the observed movements.

In conclusion, lower extremity power training improved fast gait velocity by increasing hip mechanical output and detraining caused reciprocal adaptations in joint kinetics albeit further increases in fast gait velocity. Muscle power is a good predictor of gait velocity but not a limiting factor for improving healthy old adults' gait velocity. Interventions should not be limited to plantarflexor training but should include exercises designed to increase peak power of the lower extremity.

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