Achilles Tendinopathy Modulates Force Frequency Characteristics of Eccentric Exercise

NICOLE L. GRIGG[1], SCOTT C. WEARING[2,3], JOHN M. O'TOOLE[4], and JAMES E. SMEATHERS[1]

[1]Institute of Health and Biomedical Innovation, Queensland University of Technology, Kelvin Grove, Queensland, AUSTRALIA; [2]Faculty of Health Sciences and Medicine, Bond University, Gold Coast, Queensland, AUSTRALIA, [3]Center of Excellence for Applied Sport Science Research, Queensland Academy of Sport, Sunny Bank, Queensland, AUSTRALIA; and [4]DeustoTech, University of Deusto, Bilbao, SPAIN

ABSTRACT

GRIGG, N. L., S. C. WEARING, J. M. O'TOOLE, and J. E. SMEATHERS. Achilles Tendinopathy Modulates Force Frequency Characteristics of Eccentric Exercise. *Med. Sci. Sports Exerc.*, Vol. 45, No. 3, pp. 520–526, 2013. **Introduction**: Previous research has demonstrated that ground reaction force (GRF) recorded during eccentric ankle exercise is characterized by greater power in the 8- to 12-Hz bandwidth when compared with that recorded during concentric ankle exercise. Subsequently, it was suggested that vibrations in this bandwidth may underpin the beneficial effect of eccentric loading in tendon repair. However, this observation has been made only in individuals without Achilles tendinopathy. This research compared the force frequency characteristics of eccentric and concentric exercises in individuals with and without Achilles tendinopathy. **Methods**: Eleven male adults with unilateral midportion Achilles tendinopathy and nine control male adults without tendinopathy participated in the research. Kinematics and GRF were recorded while the participants performed a common eccentric rehabilitation exercise protocol and a concentric equivalent. Ankle joint kinematics and the frequency power spectrum of the resultant GRF were calculated. **Results**: Eccentric exercise was characterized by a significantly greater proportion of spectral power between 4.5 and 11.5 Hz when compared with concentric exercise. There were no significant differences between limbs in the force frequency characteristics of concentric exercise. Eccentric exercise, in contrast, was defined by a shift in the power spectrum of the symptomatic limb, resulting in a second spectral peak at 9 Hz, rather than 10 Hz in the control limb. **Conclusions**: Compared with healthy tendon, Achilles tendinopathy was characterized by lower frequency vibrations during eccentric rehabilitation exercises. This finding may be associated with changes in neuromuscular activation and tendon stiffness that have been shown to occur with tendinopathy and provides a possible rationale for the previous observation of a different biochemical response to eccentric exercise in healthy and injured Achilles tendons. **Key Words:** PHYSIOLOGICAL TREMOR, REHABILITATION, TENDON, MECHANICAL PROPERTIES

Eccentric exercise is a popular and effective treatment for Achilles tendinopathy (15,24,31,32). However, the mechanisms underlying the beneficial effect of eccentric exercise are yet to be established. Previous research has examined the biomechanical characteristics of eccentric exercise in detail and noted that ground reaction force (GRF) recorded during eccentric ankle exercise was characterized by greater signal power in the 8- to 12-Hz bandwidth when compared with that recorded during concentric ankle exercise (11). This observation indicates that the magnitude of 8- to 12-Hz vibrations, to which the tendon is exposed, is greater during eccentric than concentric ankle exercise (11) and has led these authors to suggest that vibrations in the frequency range of 8 to 12 Hz may be associated with the beneficial effect of eccentric but not concentric rehabilitation exercises (11). To date, however, the frequency characteristics of motor output force recorded during eccentric and concentric ankle exercises have only been examined in healthy participants without Achilles tendinopathy.

It is particularly important to investigate the spectral features of the motor output force in cases of clinical tendinopathy because pain and changes in tendon stiffness, which are inherent to the condition (4,26), have the potential to modify the frequency characteristics of the force (12,19). Based on the hypothesis that vibrations in the 8- to 12-Hz bandwidth play a role in tendon adaptation (11), a potential change in the force frequency characteristics due to tendinopathy may, in part, explain a previous observation of a different biochemical response to eccentric loading in healthy and injured tendons (16). The purpose of the current research, therefore, was to compare the force frequency characteristics of eccentric rehabilitation exercises

Address for correspondence: Nicole L. Grigg, Ph.D. Center for Musculoskeletal Research, Griffith University Gold Coast, Queensland 4222, Australia; E-mail: n.grigg@griffith.edu.au.
Submitted for publication April 2012.
Accepted for publication October 2012.
Supplemental digital content is available for this article. Direct URL citations appear in the printed text and are provided in the HTML and PDF versions of this article on the journal's Web site (www.acsm-msse.org).

0195-9131/13/4503-0520/0
MEDICINE & SCIENCE IN SPORTS & EXERCISE®

DOI: 10.1249/MSS.0b013e31827795a7

to concentric exercises in individuals with and without Achilles tendinopathy.

METHODS

Participants. Eleven male adults with unilateral Achilles tendinopathy and nine control male adults participated in the research. Inclusion criteria for Achilles tendinopathy participants comprised the following: pain and a sensation of stiffness particularly at the onset of loading, palpable focal thickening of the Achilles tendon 20–60 mm proximal to the calcaneal insertion, unilateral symptoms of greater than 6-wk duration (9), and sonographic evidence of focal thickening and hypoechogenicity (18,27). Volunteers presenting with insertional tendinopathy, bilateral symptoms, or a history of Achilles tendon surgery, rupture, partial tear, or calf muscle injury were excluded. Participants with Achilles tendinopathy had a mean score of 62 ± 4 on the Victorian Institute of Sport Assessment Achilles, where a score of 0 represents the worst symptoms possible and a score of 100 represents no symptoms (26). The median symptom duration was 10 months (range, 2–30 months). Nine of the 11 participants with tendinopathy were individually matched to nine control participants on age, height, and mass. Volunteers were excluded from the control group if they reported a medical history of Achilles tendon pain or calf muscle injury. The study received University Human Research Ethics approval, and all participants provided written informed consent.

Procedure. Lower limb kinematics and GRF were recorded while participants performed the exercise protocol. The exercise protocol was based on a widely implemented therapeutic program for Achilles tendinopathy (2) and involved isolated eccentric loading of the triceps surae muscle tendon unit of a single limb, whereas the contralateral muscle tendon unit experienced isolated concentric loading. To begin the exercise, participants stood with the forefoot of a single limb positioned on the edge of a 90-mm-high step with the ankle maximally plantarflexed. Eccentric loading occurred as the participant lowered the heel below the level of the forefoot to a position of maximal dorsiflexion. The forefoot of the previously non–weight-bearing contralateral limb was then placed on the step in a maximally dorsiflexed position. All weight was transferred to the contralateral limb, and the limb that performed the eccentric muscle action was removed from the step. The participant then performed a concentric muscle action, moving from a position of maximal ankle dorsiflexion to maximal ankle plantarflexion. This concentric muscle action returned the body to the start position. At this time, the limb that performed the eccentric muscle action was again positioned on the step in maximal plantarflexion, all weight transferred to this limb, allowing the loading process to be repeated. The process, which consisted of one eccentric muscle action and one concentric muscle action, was repeated 15 times per exercise set. A total of six exercise sets (90 repetitions) were performed, and the exercise was performed without footwear.

Exercise sessions were conducted at a standard time of day, approximately 0600 h, and participants were required to refrain from physical activity, beyond that required for activities of daily living, for 24 h before exercise. This protocol was adopted to minimize the potential confounding effects of tendon conditioning arising from incidental walking activity and diurnal variations in muscle activation (10,23,28). Furthermore, participants with tendinopathy were requested to refrain from seeking treatment or performing prescribed rehabilitation exercises in the week before testing and during the study period. Each participant with Achilles tendinopathy performed the testing protocol twice, such that force and kinematic data were recorded for the symptomatic limb during both isolated eccentric and concentric exercise. The two exercise sessions were separated by a "washout" period of 4–7 d to ensure full recovery of muscular performance between exercise bouts (6). The order of eccentric and concentric exercise was counterbalanced between participants with tendinopathy. Control participants performed the testing process once, with the allocation of the left or right limb to either eccentric or concentric exercise counterbalanced.

Kinematic and kinetic analysis. The orientations of lower limb segments were recorded by an 11-camera motion analysis system sampling at 200 Hz (Vicon; Oxford Metrics Group, Oxford, England). The Plug-In-Gait (SCAR) model within Vicon Nexus (version 1.4.116, Vicon, Oxford Metrics Group) was used to model the lower body as seven rigid segments (pelvis, right and left upper leg, lower leg, and foot segments) (30). The model required the attachment of 15 passive markers (Ø 14 mm) to the participant. Markers were attached bilaterally to the dorsum of the second metatarsal head, lateral malleolus, posterior superior calcaneus, midtibia, lateral femoral condyle, midthigh, and anterior superior iliac spine. A marker was also positioned on the midpoint of the sacrum (Fig. 1).

A force plate (OR6-6200 Advance Mechanical Technology Inc., Watertown, MA) sampling at 1000 Hz was used to record GRF during the exercise. To allow maximal ankle joint dorsiflexion, a solid wooden step (90 mm high) was mounted to the middle of the force plate (Fig. 1). The step was confined entirely within the borders of the force plate and ensured that the body was centered over the plate. It was assumed that no deformation of the wooden step occurred during exercise performance. Kinematic data were synchronized with GRF via Vicon Nexus.

Data analysis. All marker displacements and joint kinematics were calculated by Vicon Nexus. Kinematic and GRF data were segmented using MATLAB software (version R2008a; MathWorks Inc., Natick, MA). The vertical displacements of the second metatarsal head and calcaneus were used to isolate the 15 eccentric and 15 concentric repetitions of each exercise set. Maximum and minimum sagittal ankle joint angles, ankle joint range of motion (ROM), and the average angular velocity of the ankle joint were determined for each exercise repetition.

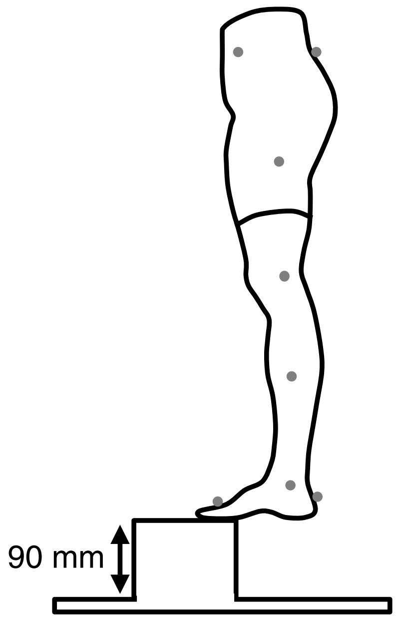

FIGURE 1—Kinematic marker system and step configuration.

The resultant GRF (GRFres) was calculated by taking the Euclidean norm of the three dimensional force vectors, i.e., GRFres $= \sqrt{F_x^2 + F_y^2 + F_z^2}$, where F_x represents the GRF in the x direction and likewise for F_y and F_z. After removal of the mean offset (bodyweight), the GRFres was high-pass filtered, with a 0.5-Hz cut-off frequency, to remove the trend associated with whole body movement. For all exercise repetitions, exercise conditions, and limbs, 99% of the spectral energy was below 15.5 Hz. Consequently, the residual content above 20 Hz was removed by low-pass filtering. Low- and high-pass filtering was conducted using linear-phase finite impulse response filters with even-order, symmetric filter coefficients (Type I). Filtered data were then down sampled from 1000 to 100 Hz, zero padded to 4096 sample points, and then Fourier transformed to calculate the magnitude of the power spectrum. Given significant differences in total power of the magnitude spectra between control and symptomatic limbs (3420 ± 375 vs 1638 ± 320 N^2, $P < 0.05$), each magnitude spectrum was normalized to total power. Normalization ensured that all spectra had a total energy value of one and allowed quantification and comparison of the relative power contained within specified bandwidths of each magnitude spectrum. The relative spectral power was then summed over nonoverlapping 1-Hz windows within the range 0.5 to 15.5 Hz. The average values of within-limb SE of measurement for relative power in the frequency windows of particular interest, 7, 8, 9, 10, and 11 Hz, were 0.008, 0.009, 0.008, 0.007, and 0.004, respectively.

The Statistical Package for the Social Sciences (version 17; SPSS Inc, Chicago, IL) was used for all statistical procedures. The age, height, and mass characteristics of the Achilles tendinopathy group were contrasted with those of the control group using one-way ANOVA. Because of the matching process, the control group was not independent of the tendinopathy group. Consequently, a variable "*pair*" was created to pair the data from Achilles tendinopathy participants with that of their matched controls. Linear mixed models were used to evaluate the effects of exercise condition (eccentric or concentric) and limb (control or symptomatic) on kinematic variables and relative power in each frequency window. Within each model, exercise condition and limb were treated as fixed effects, whereas *pair* and *exercise repetition* were modeled as random effects. For each linear mixed model, the underlying assumption of normality of the residual variance was met. *Post hoc* analyses of significant main effects were conducted using custom hypothesis tests within the models, whereas pairwise comparisons and 95% confidence intervals were used for *post hoc* analyses of significant interactions. In accordance with Cohen (7), the effect size was calculated for significant effects to determine the degree to which the null hypothesis was false, wherein large, medium, and small effect sizes were denoted by values greater than 0.8, 0.5, and 0.2, respectively.

RESULTS

Demographic details of the two participant groups are presented in Table 1. There were no statistically significant differences in the mean age, height, or mass of the control and Achilles tendinopathy groups.

Table 2 demonstrates the kinematic characteristics of the limbs during eccentric and concentric exercise. For both eccentric and concentric exercises, the symptomatic limb was characterized by significantly less dorsiflexion ($P < 0.05$) and significantly more plantarflexion ($P < 0.05$) compared with the control limb. Despite the differences in peak joint angles, ankle joint ROM was not significantly different between exercise conditions or limbs. However, eccentric exercise was performed at a significantly slower velocity than concentric exercise ($P < 0.05$). Moreover, the symptomatic limb performed both eccentric and concentric exercises at a significantly slower velocity compared with the control limb ($P < 0.05$).

The GRFres spectral data for each individual participant and exercise condition are accessible as a supplemental digital content (SDC) (see SDC 1, Supplemental Table 1, http://links.lww.com/MSS/A208: Mean (95% confidence interval) normalized power for each control and symptomatic limb during eccentric exercise and Supplemental Table 2: Mean (95% confidence interval) normalized power for each control and symptomatic limb during concentric exercise). Figure 2 demonstrates the mean relative power in the GRFres signal during eccentric and concentric exercise in symptomatic and control limbs, that is, the two-way interaction

TABLE 1. Mean (95% confidence interval) age, height, and mass of control and tendinopathy participant groups.

	Control	Tendinopathy
Age (yr)	49.0 (10.3)	48.2 (8.5)
Height (cm)	180.6 (4.4)	181.6 (5.1)
Mass (kg)	92.6 (12.9)	97.3 (15.3)

TABLE 2. Mean (95% confidence interval) kinematic characteristics of eccentric and concentric exercises in control and symptomatic limbs.

	Control	Symptomatic
Eccentric		
Peak dorsiflexion (°)	22.1 (2.5)	12.9 (2.1)[a]
Peak plantarflexion (°)	−10.1 (2.4)	−17.4 (2.0)[a]
Ankle ROM (°)	32.2 (1.3)	30.3 (1.1)
Ankle angular velocity ($°\cdot s^{-1}$)	37.5 (3.0)	29.9 (2.6)[a]
Concentric		
Peak dorsiflexion (°)	19.0 (2.5)	10.3 (2.3)[a]
Peak plantarflexion (°)	−13.2 (2.4)	−18.4 (2.3)[a]
Ankle ROM (°)	32.3 (1.3)	28.7 (1.3)
Ankle angular velocity ($°\cdot s^{-1}$)	46.8 (3.0)[b]	39.8 (2.9)[a,b]

[a]Significantly different from control limb ($P < 0.05$).
[b]Significantly different from eccentric exercise ($P < 0.05$).

between limb and exercise condition for each frequency window. For both limbs, the relative power in the GRFres signal was significantly different between eccentric and concentric exercises for the 1-Hz spectral power windows centered at 1, 2, 5, 6, 7, 8, 9, 10, and 11 Hz ($P < 0.05$). Within the 1- and 2-Hz windows, relative power was significantly greater for concentric exercise compared with eccentric exercise ($P < 0.05$), although the effect sizes were low (Table 3). At the higher frequencies (5, 6, 7, 8, 9, 10, and 11 Hz), this trend was reversed with significantly greater relative power observed during eccentric exercise ($P < 0.05$). Although each of these windows demonstrated significantly greater power during eccentric compared with concentric exercise, medium or large effect sizes were only observed for the 9- and 10-Hz windows for the control limb and the 8-, 9-, 10-, and 11-Hz windows for the symptomatic limb (Table 3). *Post hoc* analyses revealed that during concentric exercise, there were no significant differences in the relative signal power between control and symptomatic limbs in any of the frequency windows. However, during eccentric exercise, compared with the control limb, the symptomatic limb demonstrated significantly greater relative power in the 7- and 8-Hz windows with small effect sizes observed and significantly lower relative power in the 10- and 11-Hz windows with medium effect sizes (Table 4). These differences in the magnitude of relative power reflected a shift in the power spectrum to a lower frequency in individuals with Achilles tendinopathy (Fig. 2).

DISCUSSION

The purpose of this research was to evaluate the force frequency characteristics of eccentric and concentric ankle exercises in individuals with and without Achilles tendinopathy. Consistent with previous research (11), concentric exercise was characterized by greater relative power in the lower frequency bandwidth (0.5–2.5 Hz), whereas eccentric exercise was characterized by greater relative power in the higher frequency bandwidth (4.5–11.5 Hz). An earlier study investigating the same exercise protocol as the current research has shown that the amplitude of the surface electromyography signals recorded from the triceps surae muscle group was significantly lower during eccentric compared with concentric exercise (11). A lower level of activation to produce a given force for eccentric compared with concentric muscle actions is a commonly observed phenomenon and has been associated with a reduction in the number of active motor units and the rate at which the motor units discharge (1,5,8). Vibrations in the 8- to 12-Hz bandwidth are known to be a product of some factors including the rate

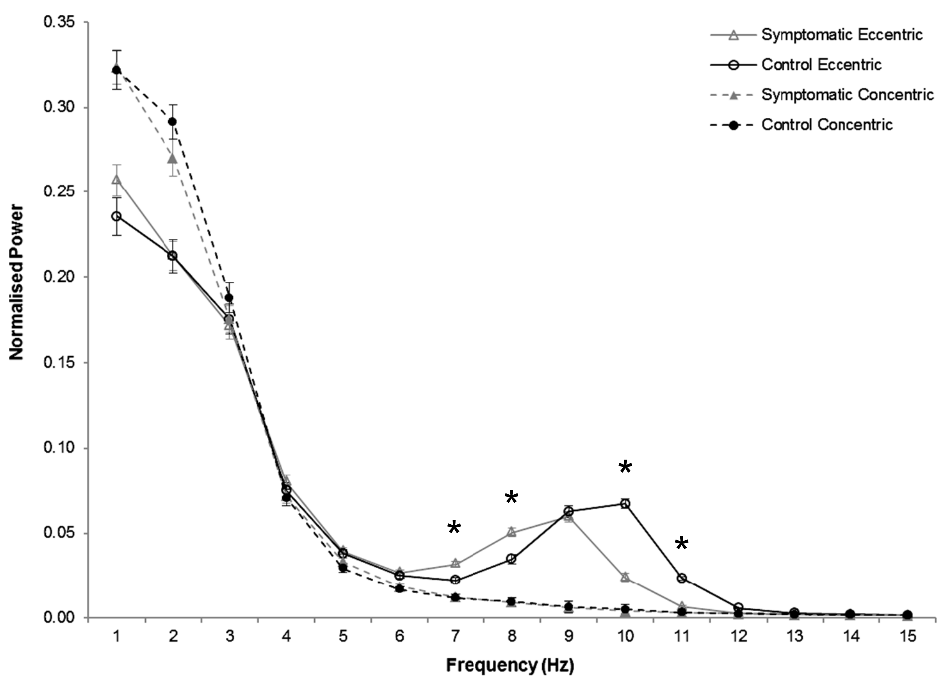

FIGURE 2—Mean normalized power within each 1-Hz window for control and symptomatic limbs during eccentric (*no fill*) and concentric (*filled*) exercise. *Error bars* represent 95% confidence intervals. *Significantly different from control limb ($P < 0.05$).

APPLIED SCIENCES

TABLE 3. Mean (95% confidence interval) normalized power and effect size for exercise condition in control and symptomatic limbs.

	Eccentric	Concentric	Effect Size
Control			
1 Hz	0.236 (0.011)	0.322 (0.011)[a]	−0.22
2 Hz	0.212 (0.010)	0.292 (0.010)[a]	−0.30
5 Hz	0.038 (0.002)	0.029 (0.002)[a]	0.13
6 Hz	0.025 (0.002)	0.017 (0.002)[a]	0.19
7 Hz	0.022 (0.002)	0.012 (0.002)[a]	0.21
8 Hz	0.035 (0.003)	0.009 (0.003)[a]	0.35
9 Hz	0.063 (0.004)	0.007 (0.004)[a]	0.57
10 Hz	0.067 (0.003)	0.005 (0.003)[a]	0.28
11 Hz	0.024 (0.001)	0.003 (0.001)[a]	0.78
Symptomatic			
1 Hz	0.257 (0.010)	0.324 (0.010)[a]	−0.29
2 Hz	0.212 (0.009)	0.271 (0.009)[a]	−0.22
5 Hz	0.039 (0.002)	0.033 (0.002)[a]	0.09
6 Hz	0.027 (0.001)	0.019 (0.001)[a]	0.19
7 Hz	0.032 (0.002)	0.012 (0.002)[a]	0.40
8 Hz	0.050 (0.002)	0.009 (0.003)[a]	0.57
9 Hz	0.060 (0.003)	0.006 (0.003)[a]	0.60
10 Hz	0.024 (0.002)	0.004 (0.002)[a]	0.87
11 Hz	0.006 (0.001)	0.003 (0.001)[a]	0.13

[a]Significantly different from eccentric exercise ($P < 0.05$).

at which the motor units discharge (19). From this evidence, it may be hypothesized that the observed difference in the distribution of relative spectral power between eccentric and concentric exercise may possibly be associated with differences in the neuromuscular activation of the two muscle actions (11). The higher magnitude of 8- to 12-Hz vibrations generated during eccentric compared with concentric exercise has been put forward as a potential mechanism for the clinical efficacy of eccentric but not concentric exercise in the treatment of Achilles tendinopathy (11). However, there is currently no evidence directly linking the magnitude of 8- to 12-Hz vibrations with a favorable biochemical response leading to the resolution of tendinopathy symptoms. The current study has, for the first time, shown that eccentric exercise is characterized by greater relative signal power in the 8- to 12-Hz bandwidth compared with concentric exercise when performed by participants with symptomatic Achilles tendinopathy. As such, the higher magnitude of vibrations in the 8- to 12-Hz bandwidth endures as a plausible although yet to be verified mechanism for the effectiveness of eccentric exercise in the treatment of tendinopathy.

For the control limb, the bimodal power spectrum of eccentric exercise was characterized by the second spectral peak occurring at 10 Hz. Although this observation is consistent with previous research (12), the symptomatic limb was defined by a shift in the power spectrum, resulting in the second spectral peak occurring at 9 Hz. Vibrations in the 8- to 12-Hz frequency range, commonly called physiological tremor, are the product of several factors including the firing frequency of motor units, the synchronization of motor unit firing, the mechanical properties of the bone, muscle and soft tissue, peripheral stretch reflex, and CNS oscillations (19). Although there are some factors that contribute to the spectral features of physiological tremor, factors most relevant to the observed shift in the second spectral peak with Achilles tendinopathy include differences in the kinematics of the movement, activation of the triceps surae muscle group, and the compliance of the Achilles tendon.

In the symptomatic limb, eccentric exercise was characterized by a relatively plantarflexed initial position, a less dorsiflexed final position, and a slower angular velocity compared with the control limb. It is possible, therefore, that the movement strategy used by the Achilles tendinopathy group during eccentric exercise resulted in a shift of the relative power spectrum to a lower frequency. However, previous research has demonstrated that the movement velocity does not influence the frequency of peak power in the 8- to 12-Hz bandwidth (14), suggesting that the slower ankle movement adopted by participants with Achilles tendinopathy is unlikely to account for the shift in the relative power spectrum. It is possible that the relatively plantarflexed movement strategy adopted in the Achilles tendinopathy group resulted in a relative shortening of the muscle fascicles of the triceps surae (20). However, it is conceivable that contracture of the series elastic elements may occur with tendinopathy, and the more plantarflexed position represented an equivalent muscle fascicle working length across control and symptomatic limbs (22). Furthermore, previous research has shown that during isometric plantarflexion, modification of the knee joint angle and subsequent changes in the length of the medial and lateral gastrocnemius did not result in a shift of the second spectral peak, which occurred at 8.5 Hz in both flexed and extended knee positions (34). As such, it would appear unlikely that the relatively plantarflexed movement strategy accounted for the shift in the second spectral peak to a lower frequency in Achilles tendinopathy.

The observation of a shift in the GRFres power spectrum of eccentric exercise to a lower frequency for the symptomatic limb, with no appreciable difference in the magnitude of relative power between control and symptomatic limbs, is in contrast to previous observations made when pain was artificially induced in the Achilles tendon by intratendinous injection of hypertonic saline (12). In that case, power at 10 Hz reportedly increased during eccentric ankle exercise without a shift in the frequency of the second spectral peak (12). Given that tendinopathy is associated with pain (26), it was anticipated that, compared with the control group, the Achilles tendinopathy group would have possessed greater power at 10 Hz during eccentric loading, which was not the case. Although Achilles tendinopathy has been associated with higher activation levels of the triceps surae compared with pain free tendons (25), artificially

TABLE 4. Mean (95% confidence interval) normalized power and effect size for limb during eccentric exercise.

Frequency Window (Hz)	Control	Symptomatic	Effect Size
7	0.022 (0.002)	0.032 (0.002)[a]	0.19
8	0.035 (0.003)	0.050 (0.002)[a]	0.21
10	0.067 (0.003)	0.024 (0.002)[a]	−0.59
11	0.024 (0.001)	0.006 (0.001)[a]	−0.65

[a]Significantly different from control limb ($P < 0.05$).

APPLIED SCIENCES

induced Achilles tendon pain was associated with lower activation levels of these muscles (12). Thus, neuromuscular activation in artificially induced pain may not necessarily reflect that in tendinopathy and is potentially responsible for the disparity in force frequency characteristics of the two conditions. Interestingly, a study of muscle fatigue has demonstrated that complete muscular fatigue produced a shift in the second spectral peak from 8.7 to 10.5 Hz (13). This shift in the second spectral peak to a higher frequency was hypothesized to result from the cerebral oscillatory network operating at a faster rate to maintain the required force by recruiting additional motor units (13). Speculatively, the 1-Hz shift to a lower frequency observed in the current study may be associated with a change in the central control of muscle activation with tendinopathy. Although the current study does not provide evidence for the observed 1-Hz shift to be associated with changes in the central control of muscle activation with tendinopathy, this hypothesis is consistent with animal models of tendinopathy, in which central neural mechanisms have been implicated in the observed bilateral development of tendinopathy after a unilateral loading regimen (3).

An alternative explanation for the observed shift in the frequency power spectrum of the symptomatic limb during eccentric exercise relates to the stiffness of the tendon. As the Achilles tendon transmits forces generated by the triceps surae to the calcaneus (21), the frequency of force vibrations that are dampened is dependent on the stiffness of the tendon, with a more compliant tendon attenuating higher frequency vibrations (19,29). Degenerative change associated with tendinopathy has been shown to lower the stiffness of the Achilles tendon by approximately 20% (4), which in theory would result in a 1-Hz drop in the frequency attenuated by the tendon (29). Although this study cannot provide insight into the mechanisms underlying the observed 1-Hz shift in the second spectral peak of the motor output force in tendinopathy, a 1-Hz shift is sizable when considered in light of the physiological tremor bandwidth (8–12 Hz). Moreover, studies of muscular fatigue have shown that an increase of only 1.8 Hz occurs with complete muscular fatigue resulting from prolonged loading (13). Because *in vitro* studies have shown that the expression of a collagenase and the mechanical properties of cultured tendon fascicles are sensitive to the frequency of loading (17,33), the potential exists for the observed shift in the second spectral peak, from 10 Hz in healthy tendon to 9 Hz in tendinopathy, to result in different biochemical responses to eccentric loading for healthy and injured tendons. Although this hypothesis is yet to be investigated, the findings of the current research may partially explain the different biochemical response of tendons with and without tendinopathy to eccentric exercise, with eccentric training of the triceps surae shown to result in an increase in the rate of collagen I synthesis in Achilles tendinopathy but not healthy Achilles tendons (16).

The current research is limited by some factors, which should be considered when interpreting the results. The investigation used an eccentric loading protocol that is commonly used in clinical settings, and as such, no attempt was made to standardize ankle joint movement across participants. Future research may benefit from controlling movement kinematics by using prescribed ankle joint angles and movement velocities, although this would be at the expense of clinical relevance. Similarly, this research did not measure the neuromuscular activation of the triceps surae or Achilles tendon stiffness. Consequently, it is unknown whether the observed shift in the GRFres power spectrum may be associated with a change in neuromuscular activation and or the mechanical properties of the tendon with Achilles tendinopathy. Nonetheless, the findings of the current study demonstrate that common eccentric rehabilitation exercises are associated with lower frequency vibrations when performed by individuals with Achilles tendinopathy compared with individuals without tendinopathy. Importantly, this new finding has highlighted three key areas for future research: 1) the influence of tendon stiffness on the frequency characteristics of motor output force, 2) the role of neuromuscular activation in the development of tendinopathy, and 3) the relationship between force frequency characteristics and the tendon's biochemical response to loading.

CONCLUSIONS

This is the first study to show that the spectral features of the motor output force recorded during eccentric exercise differ between healthy control individuals and individuals with Achilles tendinopathy. The performance of eccentric exercise by individuals with Achilles tendinopathy was characterized by a shift in the frequency of the second peak of the typically bimodal GRFres power spectrum, resulting in the second spectral peak occurring at 9 Hz compared with 10 Hz in individuals without tendinopathy. This shift in the second spectral peak may possibly be related to changes in neuromuscular activation and or tendon stiffness, which has been shown to occur with tendinopathy. Because both control and tendinopathy groups demonstrated significantly higher relative power in the 8- to 12-Hz bandwidth during eccentric compared with concentric exercise, vibrations in this bandwidth remain a potential although yet to be verified mechanism for the beneficial effect of eccentric rehabilitation exercises. Differences in the frequency of peak power within this bandwidth, however, have the potential to provide a rationale for the previous observation of a different biochemical response to eccentric exercise in healthy and injured Achilles tendons.

Dr. Wearing is funded through a Smart Futures Fellowship, Department of Employment, Economic Development and Innovation, Queensland Government.

The authors have no conflicting interests that are directly related to the content of the manuscript, and no financial support was received for the preparation of this manuscript. The results of the present study do not constitute endorsement by the American College of Sports Medicine.

REFERENCES

1. Aagaard P, Simonsen EB, Andersen JL, Magnusson SP, Halkjaer-Kristensen J, Dyhre-Poulsen P. Neural inhibition during maximal eccentric and concentric quadriceps contraction: effects of resistance training. *J Appl Physiol*. 2000;89(6):2249–57.
2. Alfredson H, Pietila T, Jonsson P, Lorentzon P. Heavy-load eccentric calf muscle training for the treatment of chronic Achilles tendinosis. *Am J Sports Med*. 1998;26(3):360–6.
3. Andersson G, Forsgren S, Scott A, et al. Tenocyte hypercellularity and vascular proliferation in a rabbit model of tendinopathy: contralateral effects suggest the involvement of central neuronal mechanisms. *Br J Sports Med*. 2010;45(5):399–406.
4. Arya S, Kulig K. Tendinopathy alters mechanical and material properties of the Achilles tendon. *J Appl Physiol*. 2010;108(3):670–5.
5. Beltman JGM, Sargeant AJ, van Mechelen W, de Haan A. Voluntary activation level and muscle fiber recruitment of human quadriceps during lengthening contractions. *J Appl Physiol*. 2004;97(2):619–26.
6. Chatzinikolaou A, Fatouros IG, Gourgoulis V, et al. Time course of changes in performance and inflammatory responses after acute plyometric exercise. *J Strength Cond Res*. 2010;24(5):1389–98.
7. Cohen J. *Statistical Power Analysis for the Behavioral Sciences*. 2nd ed. Hillsdale (NJ): Lawrence Erlbaum Associates Inc; 1998. pp. 8–14.
8. Duchateau J, Enoka RM. Neural control of shortening and lengthening contractions: influence of task constraints. *J Physiol*. 2008;586(24):5853–64.
9. Fredberg U, Stengaard-Pedersen K. Chronic tendinopathy tissue pathology, pain mechanisms, and etiology with a special focus on inflammation. *Scand J Med Sci Sports*. 2008;18(1):3–15.
10. Grigg NL, Stevenson NJ, Wearing SC, Smeathers JE. Incidental walking activity is sufficient to induce time-dependent conditioning of the Achilles tendon. *Gait Posture*. 2010;31(1):64–7.
11. Henriksen M, Aaboe J, Bliddal H, Langberg H. Biomechanical characteristics of the eccentric Achilles tendon exercise. *J Biomech*. 2009;42(16):2702–7.
12. Henriksen M, Aaboe J, Graven-Nielsen T, Bliddal H, Langberg H. Motor responses to experimental Achilles tendon pain. *Br J Sports Med*. 2011;45(5):393–8.
13. Huang C-T, Hwang I-S, Huang C-C, Young M-S. Exertion dependent alternations in force fluctuation and limb acceleration during sustained fatiguing contraction. *Eur J Appl Physiol*. 2006;97(3):362–71.
14. Hwang IS, Wu PS. The reorganization of tremulous movements in the upper limb due to finger tracking maneuvers. *Eur J Appl Physiol*. 2006;98(2):191–201.
15. Kingma JJ, de Knikker R, Wittink HM, Takken T. Eccentric overload training in patients with chronic Achilles tendinopathy: a systematic review. *Br J Sports Med*. 2007;41(3):e3.
16. Langberg H, Ellingsgaard H, Madsen T, et al. Eccentric rehabilitation exercise increases peritendinous type I collagen synthesis in humans with Achilles tendinosis. *Scand J Med Sci Sports*. 2007;17(1):61–6.
17. Lavagnino M, Arnoczky SP, Tian T, Vaupel Z. Effect of amplitude and frequency of cyclic tensile strain on the inhibition of MMP-1 mRNA expression in tendon cells: an in vitro study. *Connect Tissue Res*. 2003;44(3–4):181–7.
18. Leung JL, Griffth JF. Sonography of chronic Achilles tendinopathy: a case-control study. *J Clin Ultrasound*. 2008;36(1):27–32.
19. McAuley JH, Marsden CD. Physiological and pathological tremors and rhythmic central motor control. *Brain*. 2000;123:1545–67.
20. Narici MV, Binzoni T, Hiltbrand E, Fasel J, Terrier F, Cerretelli P. In vivo human gastrocnemius architecture with changing joint angle at rest and during graded isometric contraction. *J Physiol*. 1996;496(1):287–97.
21. O'Brien M. The anatomy of the Achilles tendon. In: Muffulli N, ed. *Foot and Ankle Clinics: Achilles Tendon*. Philadelphia: Elsevier Saunders; 2005. pp. 225–338.
22. Padhiar N, Al-Sayeagh H, Chan O, King J, Maffulli N. Pennation angle of the soleus in patients with unilateral Achilles tendinopathy. *Disabil Rehabil*. 2008;30(20–22):1640–5.
23. Pearson SJ, Onambele GNL. Influence of time of day on tendon compliance and estimations of voluntary activation levels. *Muscle Nerve*. 2006;33(6):792–800.
24. Rees JD, Maffulli N, Cook J. Management of tendinopathy. *Am J Sports Med*. 2009;37(9):1855–67.
25. Reid D, McNair PJ, Johnson S, Potts G, Witvrouw E, Mahieu N. Electromyographic analysis of an eccentric calf muscle exercise in persons with and without Achilles tendinopathy. *Phys Ther Sport*. 2012;13(3):150–5.
26. Robinson JM, Cook JL, Purdam C, et al. The VISA-A questionnaire: a valid and reliable index of the clinical severity of Achilles tendinopathy. *Br J Sports Med*. 2001;35(5):335–41.
27. Robinson P. Sonography of common tendon injuries. *Am J Roentgenol*. 2009;193(3):607–18.
28. Sedliak M, Finni T, Cheng S, Haikarainen T, Hakkinen K. Diurnal variation in maximal and submaximal strength, power and neural activation of leg extensors in men: Multiple sampling across two consecutive days. *Int J Sports Med*. 2008;29(3):217–24.
29. Thomson WT. *Theory of Vibration with Application*. 5th ed. Upper Saddle River (NJ): Prentice Hall; 1998. p. 18.
30. Vicon. *Plug-in Gait Product guide—Foundation Notes 2.0*. Oxford (England): Vicon Motion Systems Limited; 2010. pp. 6–12.
31. Wasielewski NJ, Kotsko KM. Does eccentric exercise reduce pain and improve strength in physically active adults with symptomatic lower extremity tendinosis? A systematic review. *J Athl Train*. 2007;42(3):409–21.
32. Woodley BL, Newsham-West RJ, Baxter GD. Chronic tendinopathy: effectiveness of eccentric exercise. *Br J Sports Med*. 2007;41(4):188–98.
33. Yamamoto E, Kogawa D, Tokura S, Hayashi K. Effects of the frequency and duration of cyclic stress on the mechanical properties of cultured collagen fascicles from the rabbit patellar tendon. *J Biomech Eng*. 2005;127(7):1168–75.
34. Yoshitake Y, Kouzaki M, Fukuoka H, Fukunaga T, Shinohara M. Modulation of muscle activity and force fluctuations in the plantarflexors after bedrest depends on knee position. *Muscle Nerve*. 2007;35(6):745–55.