

Muscle activity damps the soft tissue resonance that occurs in response to pulsed and continuous vibrations

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Abstract

This study tested the hypotheses that when the excitation frequency of mechanical stimuli to the foot was close to the natural frequency of the soft tissues of the lower extremity, the muscle activity increases 1) the natural frequency and 2) the damping to minimize resonance. Soft tissue vibrations were measured with triaxial accelerometers, and muscle activity was measured by using surface myography from the quadriceps, hamstrings, tibialis anterior, and triceps surae groups from 20 subjects. Subjects were presented vibrations while standing on a vibrating platform. Both continuous vibrations and pulsed bursts of vibrations were presented, across the frequency range of 10–65 Hz. Elevated muscle activity and increased damping of vibration power occurred when the frequency of the input was close to the natural frequency of each soft tissue. However, the natural frequency of the soft tissues did not change in a manner that correlated with the frequency of the input. It is suggested that soft tissue damping may be the mechanism by which resonance is minimized at heel strike during running.

[damping](#) [frequency](#) [heel strike](#) [impact](#) [muscle](#) [running](#)

SOFT TISSUES ACT AS WOBBLING masses that vibrate in a damped manner in response to direct mechanical excitation (31). Muscle activity alters the vibration characteristics of the soft tissues with increases in muscle force correlating to increases in their frequency and damping coefficients (31). The natural frequencies of the triceps surae, quadriceps, and tibialis anterior range from ~10 Hz for the relaxed condition to 50 Hz for a fully active state (31). If the excitation frequency of a mechanical stimulus is close to the natural frequency of the soft tissues, then those tissues should be expected to resonate.

During walking and running, the impact shock that occurs at heel strike is indirectly transmitted to the soft tissues via the skeleton. Peak impact forces at heel strike are reached within 30 ms (7,23) and so have an excitation frequency in the range of 10–20 Hz. Soft tissue resonance should thus be expected to occur at heel strike; however, observations show that such vibrations are strongly damped with typically no more than a couple of cycles. It has been shown that long-term exposure to vibrations can have detrimental effects on the soft tissues including reductions in motor unit firing rates and muscle contraction force (2) and decreases in nerve conduction velocity and attenuated sensory perception (10,13). Therefore it has been proposed that during walking and running the soft tissues of the lower extremity have a strategy of minimizing the soft tissue vibrations (24).

Muscle activity in the lower extremities responds to the excitation frequency of the impact shock at heel strike (36), in a processes referred to as muscle tuning. However, the mechanisms of muscle tuning have not yet been identified. Increasing the natural frequency of the soft tissues away from the excitation frequency and increasing the damping coefficient of the tissue are two possible strategies by which the soft tissues could reduce tissue resonance. Both these strategies are possible using muscle activity (31) and may be the mechanisms behind muscle tuning during walking and running.

The purpose of this study was to identify how the lower extremity muscles minimize the soft tissue resonance that occurs in response to pulsed and continuous mechanical vibration. In particular, two hypotheses were tested: 1) muscle activity increases the natural frequency to minimize resonance when the excitation frequency is close to the natural frequency of the soft tissues, and 2) muscle activity increases the damping to minimize resonance when the excitation frequency is close to the natural frequency of the soft tissues. These hypotheses were tested in an abstracted context of walking and running, whereby mechanical vibration was delivered to the soft tissues indirectly through skeleton via the plantar surfaces of the foot. Furthermore, the body geometry was chosen to mimic the positioning and muscle activity that occurs at heel strike during running.

METHODS

Approach to the problem.

To test hypotheses about the vibration response to impact forces that typically occur during running, the impact related vibration task was presented to subjects while they stood on a vibration platform. For one test, a series of pulsed 5-mm displacements, with excitation frequency of 13.1 Hz, were presented at 0.5-s intervals to mimic compression of a running shoe at heel strike. This test was repeated for excitation frequencies ranging from 10 to 65 Hz to span the expected range of natural frequencies in the soft tissues of the lower extremity. The amplitude of the displacements for these tests was scaled so that the vibration power delivered to the body was constant. The experiment was repeated with bursts of continuous vibrations that matched the amplitude and excitation frequencies of the pulsed set. Subjects were required to stand with a limb posture that satisfied a compromise between achieving a similar geometry and muscle activity pattern within the lower extremity to that observed at heel strike during running. Myoelectric activity and soft tissue vibrations were measured from the quadriceps, hamstrings, tibialis anterior, and

triceps surae muscle groups. The intensity of muscle activity and the dissipated vibration power were compared for the different excitation regimes presented to the subjects.

Subjects.

Ten male (age 25.6 ± 1.2 yr, mass 75.6 ± 2.8 kg; means \pm SE) and 10 female (age 23.1 ± 0.5 yr, mass 61.1 ± 2.0 kg) subjects were tested. The subjects were athletic and were students at the Sporthochschule, Köln. Subjects gave their informed, written consent to participate in accordance with the University of Calgary's Conjoint Health Research Ethics Board policy on research using human subjects.

Muscle and soft tissue masses within the lower extremity were estimated from a series of length, breadth, girth, and skinfold measurements. An Executive Diameter steel tape was used to measure the proximal and distal thigh and the proximal and distal calf lengths and the subgluteal, midthigh, knee girth, ankle, and maximum calf girths while the subjects stood. The midthigh position was located at half the vertical distance between the greater trochanter (trochanterion) and the superior margin of the lateral tibial condyle (tibiale laterale). The midcalf position was located at the point of maximum girth of the calf between the tibiale laterale and the inferior aspect of the lateral malleolus (sphyrion fibulare). The proximal and distal thigh were the thigh segments above and below the midthigh, respectively. The calf measurements were similarly divided into proximal and distal sections, above and below the midcalf. The width of the biepicondylar femur and bimalleolare was measured using a GPM anthropometer (SibnerHegner, Zurich, Switzerland). The patellar, midthigh, proximal calf, midcalf, and medial calf skinfolds were measured with a Harpenden caliper with a constant pressure of 10 g/mm^2 . These anthropologic measurements were used to estimate the segmental volume and mass (21, 29). Empirical, predictive regression equations were used to estimate the segmental mass without the bone (9) and finally the masses of the quadriceps, hamstrings, tibialis anterior, and triceps surae soft tissues (8).

Protocol.

Vibrations were applied to the subjects' feet via a hydraulically driven platform (Schenck, Darmstadt) that was made available at the University of Bochum (Fig. 1). The platform was driven by a displacement-control system and was capable of delivering 12.5-kN force. The hydraulic actuator was isolated from the building by being mounted on an 8-ton mass linked to the floor via a spring-damper system. The table displacement was controlled by a personal computer using a National Instruments DAQCard-6062E 12-bit data acquisition card, updating at 1,000 Hz.

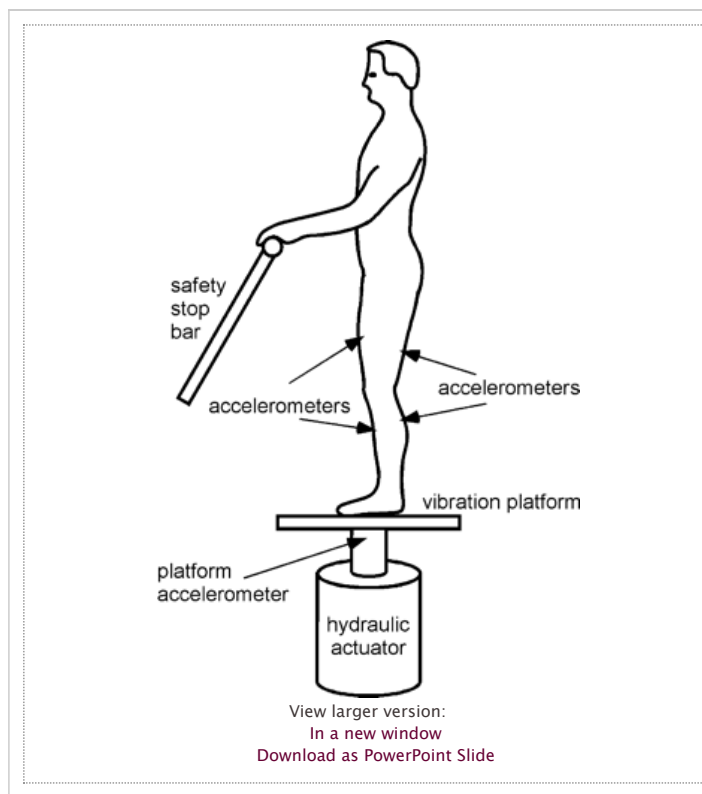


Fig. 1.

Subjects stood on a vibrating platform driven by a hydraulic actuator. During each trial, the subject would rest his or her hands on a safety bar that could stop the vibration if pressed. The position of the accelerometers is illustrated by the arrows.

Measurements on the lower leg occurred while the subjects stood with a knee-flexion angle of 37° . During measurements on the thigh, the subjects stood with a knee-flexion angle of 23° . A slightly flexed knee was used to mimic the knee posture at heel strike during running. Pilot testing of five subjects showed that these knee-flexion angles resulted in muscle activities being 20–193% of those found at heel strike during running. Standing with the heel in contact with the vibration platform resulted in uncomfortable and unsustainable vibrations traveling through the body. However, the long vibration loads during the experiment could be sustained by subjects standing on their forefeet with the heels raised 1 cm above the ground.

The test consisted of a cycle of 3 s of vibration followed by 3 s of no platform movement. During each vibration period, the frequency and amplitude of the platform vibration were kept constant. Frequencies of 10.0, 13.1, 17.1, 22.3, 29.1, 38.1, 49.7, and 65.0 Hz were tested and covered the range of natural frequencies previously described for the lower extremity muscles at different activation levels (31). These eight frequencies were presented in a randomized block. The randomized block was then repeated five times so that the subject experienced a total of 40 periods of vibration in each test. One such test was performed while measurements were taken from the lower leg, and then the test was

repeated while measurements were taken from the thigh. Tests were made when each period of vibration contained continuous sinusoidal oscillations of the platform. The peak-to-peak amplitude of the platform displacement was 5 mm at 13.1 Hz to mimic expected shoe compression during running. The displacement amplitude was scaled at the other frequencies so that the power delivered by the platform was constant across all frequencies. A second set of tests was performed in which each period of table movement consisted of pulses of seven bursts of isolated table displacements separated by 0.5-s intervals. The amplitude and duration of each displacement were changed to result in effective amplitudes and cycle frequencies that matched the continuous vibration experiments. Further experiments were made to determine the natural vibration frequencies of the soft tissues. In these experiments, 120-s periods of white noise (band-pass filtered between 5 and 100 Hz) were used to drive the platform displacements.

The response variables determined for the lower extremities were the myoelectric activity in the rectus femoris, biceps femoris (long head), tibialis anterior, and medial gastrocnemius muscles. Myoelectric activity was measured from the muscle bellies by using round bipolar surface electrodes (Ag/AgCl) after removal of the hair and cleaning of the skin with isopropyl wipes. Each electrode was 10 mm in diameter and had an interelectrode spacing of 22 mm. A ground electrode was placed on the lateral condyle of the knee. The electromyograms (EMGs) were preamplified at source (Biovision, Wehrheim, Germany). Tissue vibrations were also measured from the soft tissues of the quadriceps, hamstrings, tibialis anterior, and triceps surae muscle groups. Soft tissue vibrations were measured from the muscle bellies of the vastus lateralis, biceps femoris (long head), tibialis anterior, and lateral gastrocnemius by using skin-mounted triaxial accelerometers (EGAX accelerometer, nominal frequency response 0–600 Hz; Entran Devices). Accelerometers (<5 g) were attached to the skin surface by using Hollister medical adhesive glue, and a stretch adhesive bandage preloaded the accelerometer to improve the congruence of motion with the soft tissues (31, 32). One uniaxial accelerometer was mounted directly to the bottom surface of the platform to measure the platform movement. Myoelectric and acceleration signals were recorded at 2,400 Hz on the DAQCard-6062E 12-bit data acquisition card.

Vibration analysis.

Frequency spectra were generated for the acceleration signals in each direction by using Fourier transforms to yield the amplitude of the acceleration as a function of frequency $a(f)$. The soft tissue mass is considered a rigid mass for the analysis. For each given frequency f , the mean inertial power P required to oscillate the soft tissue mass m is given by

$$P = \frac{ma^2}{4\sqrt{2}\pi f}$$

Therefore the total inertial power P'_t required for the oscillation in the given direction is given by

$$P'_t = \frac{m}{4\sqrt{2}\pi} \int \frac{a^2}{f} df$$

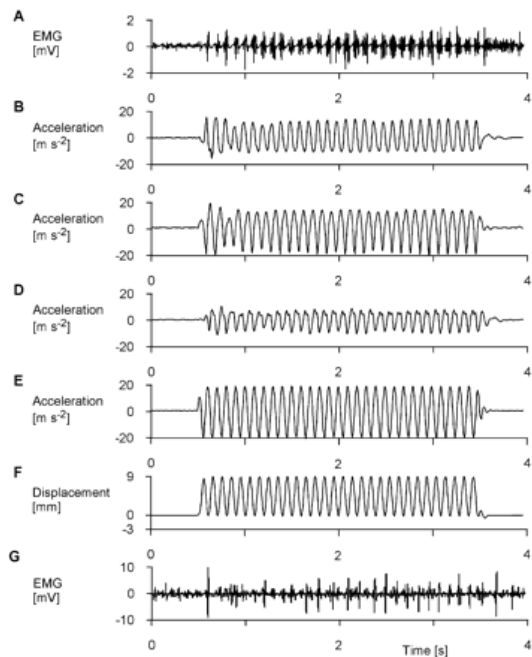
Soft tissue oscillations occur in all three orthogonal directions, and the total inertial power P_t required for the whole soft tissues vibration is given by the resultant power from the three directions (x , y , z)

$$P_t = \sqrt{P_{t,x}^2 + P_{t,y}^2 + P_{t,z}^2}$$

At the beginning of each period of vibration, the body could not anticipate its response. However, during the periods of continuous vibration, the soft tissue responses were apparent after 150 ms and resulted in a decrease in the amplitude of the acceleration (Fig.2). A steady P_t was calculated for a 1-s steady period that began 1 s after the onset of the vibration. The difference in inertial power P_t between the table and the soft tissue represents the power dissipated by the soft tissue during its steady response to the continuous vibration. Similarly, when exposed to bursts of pulsed vibrations, the body could not anticipate its response to the initial burst. However, soft tissue responses were typically apparent for all subsequent pulses (*pulses 2–7*) and resulted in a decrease in the amplitude of the acceleration (Fig.3). A mean P_t was calculated from the maximum peak-to-peak accelerations from each of *pulses 2–7*. Again, the difference in inertial power P_t between the table and the soft tissue represents the power dissipated by the soft tissue during its steady response to the pulses of vibration. The power dissipation was considered as showing a response to the experimental interventions if a maximum in the power dissipation occurred in the range of frequencies tested.

Fig. 2.

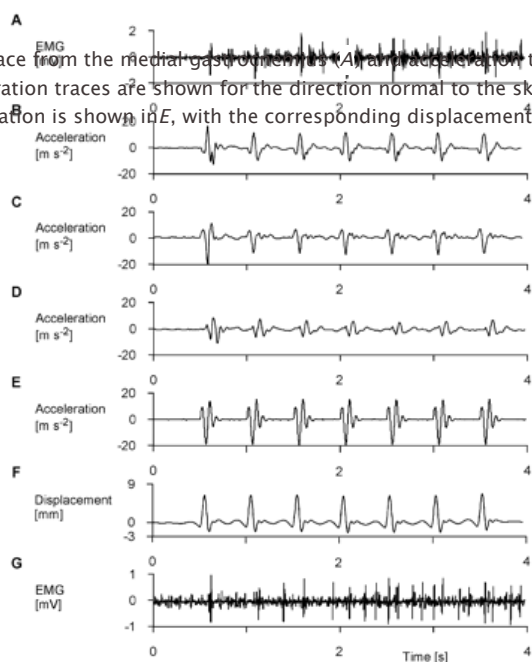
Electromyogram (EMG) trace from the medial gastrocnemius (A) and acceleration traces from the triceps surae (B–D) during 1 burst of continuous vibrations. Acceleration traces are shown for the direction normal to the skin surface (B), parallel to the tibia (C), and mediolateral (D). Platform acceleration is shown in E, with the corresponding displacement in F. The excitation frequency was 10 Hz, and the natural frequency for the triceps surae for this subject was 15 Hz. The equivalent EMG trace for the rectus femoris is shown in G.



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Fig. 3.

EMG trace from the medial gastrocnemius (A) and acceleration traces from the triceps surae (B–D) during 1 set of pulsed vibrations. Acceleration traces are shown for the direction normal to the skin surface (B), parallel to the tibia (C), and mediolateral (D). Platform acceleration is shown in E, with the corresponding displacement in F. The excitation frequency of each pulse was 10 Hz, and the natural frequency for the triceps surae for this subject was 15 Hz. The equivalent EMG trace for the rectus femoris is shown in G.



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The natural free vibration frequency was calculated from the soft tissue accelerations that followed each pulse of vibration using methods described by Wakeling and Nigg (32). Acceleration data were taken from the time window starting 100 ms after the onset of each pulse and lasting for 230 ms. An exponentially damped sinusoidal model was fitted to the data with a least squares fit. When the Rank–Pearson correlation coefficient between the measured and modeled vibration had a value >0.85 , the natural frequency was taken to be equal to the sinusoidal frequency in the model. Natural frequencies were calculated for the direction parallel to the major axis of the limb segment and for *impacts 2–7* from each burst.

EMG analysis.

The EMG signals were resolved into the myoelectric intensity in time–frequency space by use of wavelet techniques (30). A set of 11 wavelets was used with center frequencies ranging from 7 Hz (*wavelet 0*) to 395 Hz (*wavelet 10*). The intensity of the myoelectric signal is the power of the

signal contained within a given frequency band and is equivalent to twice the square of the root mean square activity (36). The total intensity was calculated across *wavelets 1-10*, which is equivalent to band-pass filtering the signal between 11 and 432 Hz. EMG signal was interpolated across the frequency band at which movement artifacts may have occurred. For each response, the interpolation was taken from 2 Hz above to 2 Hz below the peak soft tissue vibration frequency. This process did not systematically alter the calculated EMG intensity; however, it did reliably remove the movement artifact at these frequencies.

Previous studies have shown that, when this recording apparatus is used, distinct patterns of myoelectric activity can be identified at the frequency bands 25–75 Hz and 150–300 Hz (35), and these are likely the signals from different muscle fiber types (33, 35). The myoelectric signals were thus also resolved into their intensities in these high- and low-frequency bands.

During the continuous vibration experiments, the mean intensity of the EMG was determined for a 250-ms quiet period that ended 250 ms before each vibration began and then for a 1-s steady period that began 1 s after the onset of the vibration. A relative measure of the EMG intensity was taken as the ratio of the mean EMG intensity during the 1-s steady-state vibration period to the mean EMG intensity during the 250-ms quiet period before each vibration. The EMG intensity was considered as showing a response to the experimental interventions if it showed a maximum in the range of frequencies tested. For the pulsed bursts of vibrations, the mean EMG intensity was calculated for the 50-ms preactivation window that immediately preceded each pulse. The intensity was calculated for the preactivation before the initial pulse, and a mean intensity was calculated for the preactivations to *pulses 2-7*.

RESULTS

Soft tissue resonance frequencies.

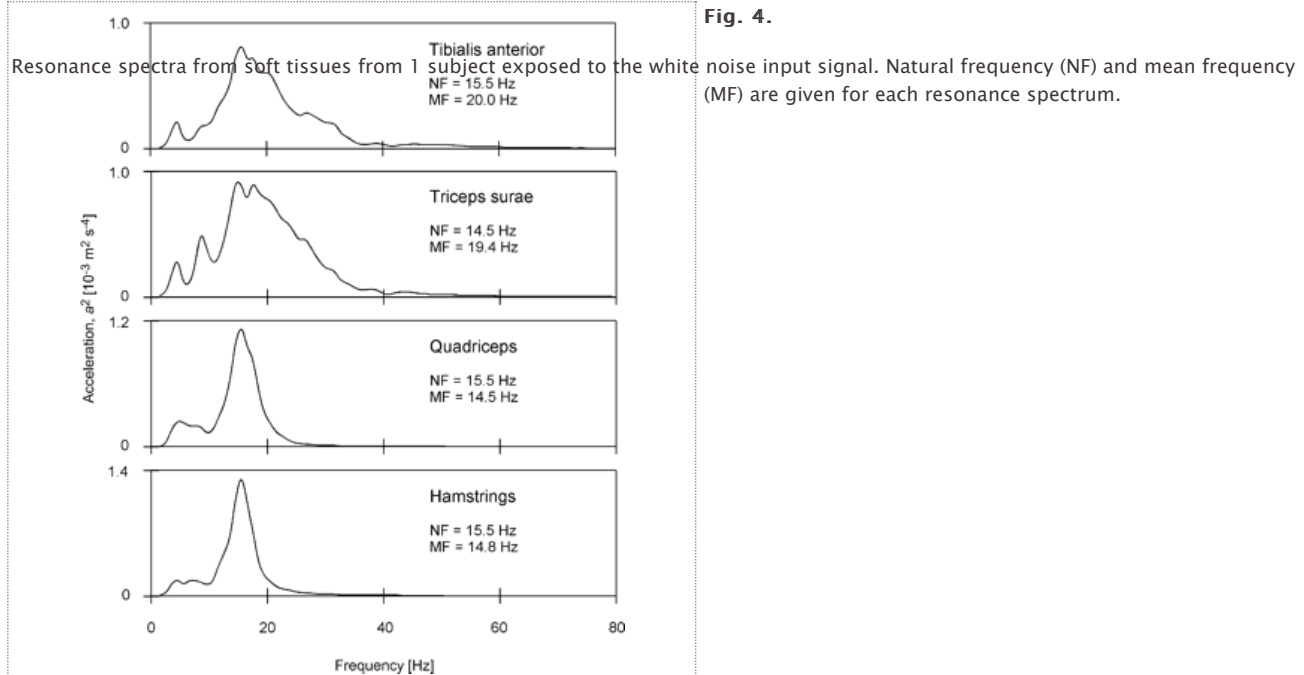
The male subjects had greater soft tissue masses than the female subjects (Table 1), and this difference was significant ($P < 0.05$, ANOVA). Analysis of covariance using mass as a covariate and muscle and gender as factors showed that there was no significant effect of the gender on either the natural or mean resonance frequencies. Therefore, the results for both genders were pooled for further analysis.

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Table 1.

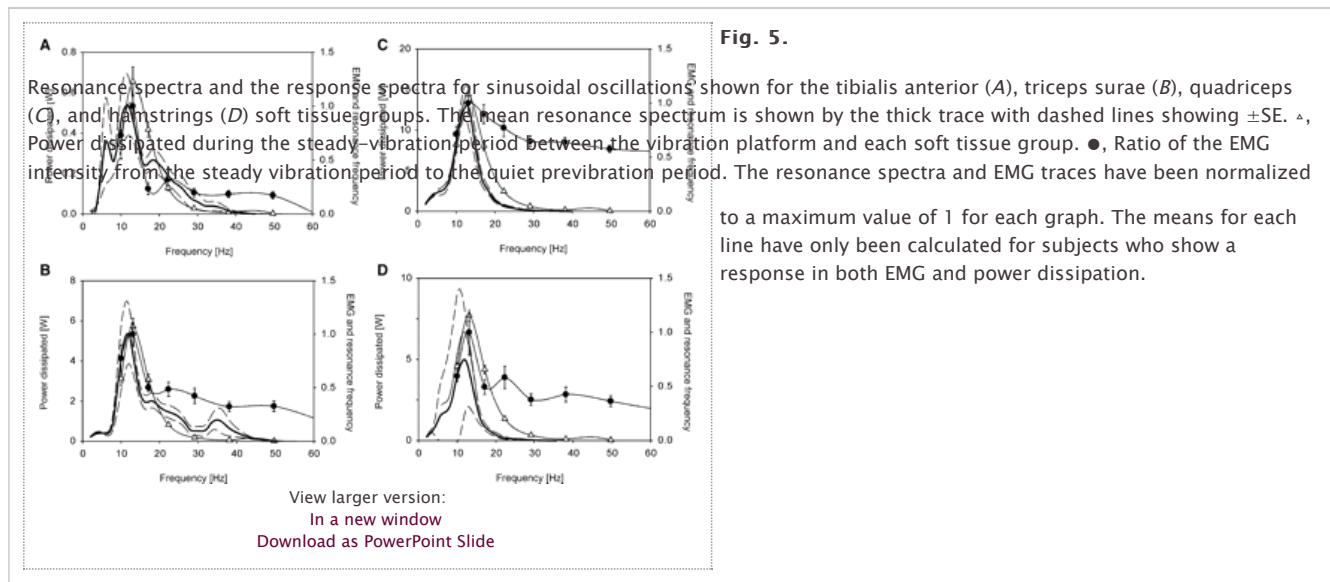
Masses of the soft tissues in the lower extremity

Sample resonance spectra from when the subjects stood on white-noise platform displacements can be seen in Fig.4. The peak of such spectra give the natural frequency, and the mean \pm SE natural frequencies pooled across all 20 subjects were 14.4 ± 0.6 , 15.9 ± 1.4 , 14.7 ± 0.3 , and 15.2 ± 0.3 Hz for the tibialis anterior, triceps surae, quadriceps, and hamstrings, respectively. The resonance spectra for the tibialis anterior and triceps surae were more skewed toward the higher frequencies than the resonance spectra for the quadriceps and hamstring soft tissues. This skewness resulted in significantly higher mean frequencies for the tibialis anterior and triceps surae (22.1 ± 0.8 and 20.1 ± 0.7 Hz, respectively) than for the quadriceps and hamstrings (14.4 ± 0.3 and 14.7 ± 0.3 Hz, respectively), as shown by a Tukey's honestly significant difference pairwise comparison test following ANOVA ($\alpha = 0.05$).



Soft tissue and muscle response to continuous vibrations.

Response spectra for both the EMG intensity and the power dissipation during the continuous-oscillation experiments are shown in Fig. 5. There were a total of 75 such responses for power dissipation and 31 responses for the EMG intensity out of a possible 80 subject-muscle combinations. The majority of cases in which responses did not occur resulted in the greatest EMG intensity and power dissipation occurring at the 10-Hz input signal. Both response spectra showed peaks close to the natural frequencies of the soft tissue masses. The shapes of the response spectra were similar to those of the resonance spectra for each soft tissue. Thus, when the input frequency was close to the natural frequency of each soft tissue, there was an increase in the muscle activity in that tissue, which resulted in an increase in the vibration damping and power dissipation. When the data were calculated from all the response cases observed, and not just those in which responses occurred in both EMG and power dissipation, the resulting response spectra showed all the same features as those in Fig. 5. The results from the continuous-vibration trials thus support the hypothesis that when the frequency of the input force is the same as the natural frequency of the soft tissue packages there is an increase in both the muscle activity and the vibration power dissipated by that tissue.



The EMG intensity during the 1-s steady-vibration periods was greater than the EMG intensity during the no-vibration quiet periods across all frequencies tested. These increases in EMG intensity occurred in the total intensity as well as in the low- and high-myoelectric-frequency bands (Table 2). ANOVA for each muscle showed that there was no significant difference between the relative increases for the total intensity and at the high- and low-frequency bands. The total EMG intensities when the subjects were tested at a 13.1-Hz input frequency ($n = 20$) were 6.3 ± 1.5 , 3.3 ± 0.8 , 1.8 ± 0.2 , and 4.2 ± 1.3 times the intensity of quiet EMG for the tibialis anterior, medial gastrocnemius, rectus femoris, and biceps femoris, respectively.

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Table 2.

The relative EMG intensity during the 1-s steady continuous-vibration period compared with the quiet period

Soft tissue and muscle response to pulsed vibrations.

The data were only able to resolve responses in power dissipation in 17 cases for the pulsed-vibration experiments. Reduced major-axis regression analysis showed that there were significant correlations in the power dissipation between the mean response to the pulsed vibrations and the mean response to the continuous vibration for all four soft tissue groups, with the coefficients of determination $0.96 < r^2 < 0.99$. The maximum power dissipated in the pulsed case was 19–35% of that for the maximum power dissipated in the continuous vibration case. Increases in soft tissue power dissipation thus occurred in response to pulsed vibrations that were close to the natural frequency in a similar manner to the responses to continuous vibration. The results from the pulsed-vibration trials thus support the hypothesis that when the frequency of the input force is the same as the natural frequency of the soft tissue packages there is an increase the vibration power dissipated by that tissue.

The natural frequencies (NF) of the soft tissues during the pulsed-vibration trials showed a broad response to the pulse duration of the test impacts, with peaks occurring at input frequencies between 17.1 and 29.1 Hz (Table 3). The maximum NF (NF_{max}) were between 14.6 and 44.9% greater than the NF at the 10-Hz input frequencies. The NF increased at a lower rate than the increases in input frequency between the 10-Hz input frequency and the input frequency at which NF_{max} occurred. When NF_{max} occurred, it was smaller than the input frequency for all four soft tissue masses. The results from the continuous-vibration trials thus do not support the hypothesis that when the frequency of the input force is the same as the natural frequency of the soft tissue packages there is an increase in the natural frequency of that tissue.

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Table 3.

The natural free vibration frequencies NF following each pulsed impact

Postdisplacement peaks of EMG intensity occurred ~80 ms after each onset of the platform displacement during the pulsed-vibration tests. These postdisplacement peaks in myoelectric intensity occurred predominantly at the low (25–75 Hz) EMG frequency band. A typical postdisplacement peak is shown in Fig. 6, A and B, for *impact 1* from a 3-s burst; however, the traces from the subsequent pulses (2–7) show similar postdisplacement activities. After the first pulse, the body responded to the subsequent *pulses 2–7* with altered muscle activity immediately before each pulsed platform displacement. Figure 6, C and D, shows the relative change in EMG activities for *pulses 2–7* compared with the EMG intensity for *pulse 1*. The response to the *pulses 2–7* consisted of an increase in the myoelectric activity at the high (150–300 Hz)-EMG-frequency band, and this increase occurred within the 50-ms period before the table displacement.

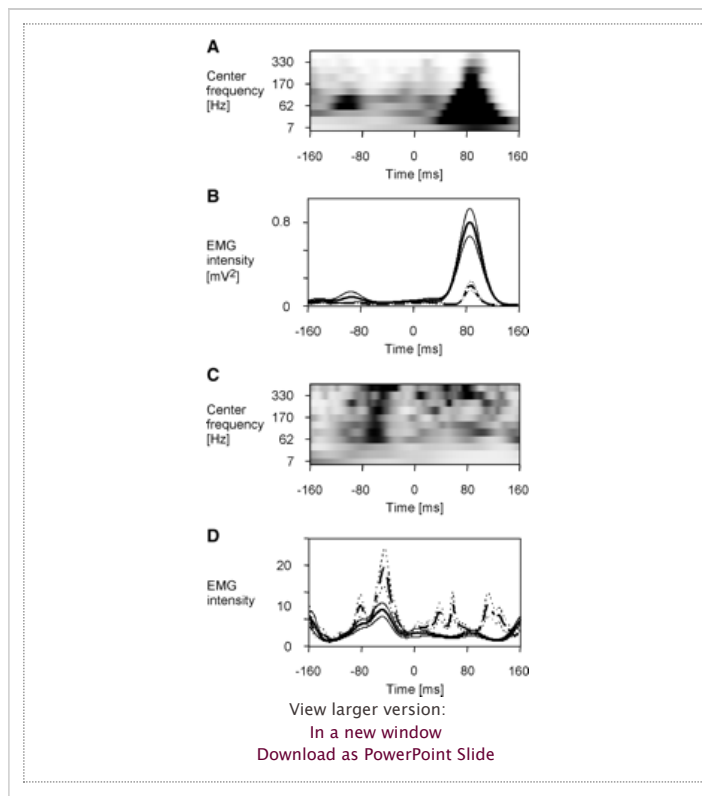


Fig. 6.

A: mean EMG intensity from the medial gastrocnemius for 1 subject for the initial impacts for the 5 trials at a 13.1-Hz pulse frequency. B: intensities for the high (150–300 Hz; dashed line)- and low (25–75 Hz; solid line)-EMG-frequency bands (mean, thick line \pm SE, thin lines; $n = 5$) for these initial impacts. C: mean EMG intensity for the subsequent 6 impacts for the 5 trials ($n = 30$) shown as their ratio to the mean EMG for the initial impacts. D: intensities for the high (150–300 Hz; dashed line)- and low (25–75 Hz; solid line)-EMG-frequency bands shown as their ratio to the mean EMG intensity at the respective frequency band for the initial impacts (mean \pm SE; $n = 30$). In all graphs, the onset of each pulse occurred at a time of 0 ms.

During the pulsed-vibration tests, there was an increase in the total EMG intensity in the 50-ms preactivation phase relative to the quiet EMG intensity. Specific increases in intensity occurred in the high-EMG-frequency band and decreases in the intensity occurred at the low-EMG-frequency band (Table 4). ANOVA for the tibialis anterior, medial gastrocnemius, and rectus femoris showed that these relative changes in total, high-EMG-frequency band, and low-EMG-frequency band intensities were significantly different. The increase in total EMG intensity in the preactivation phase was significant for the medial gastrocnemius and biceps femoris, and the increase in intensity at the high-EMG-frequency band was significant for all four muscles tested. The EMG intensity in the preactivation phase showed a response to the pulse duration of the impacts for a total of 24 cases out of the 80 possible subject-muscle combinations. The majority of cases, in which responses did not occur, resulted in the greatest EMG intensity occurring at the 10-Hz input signal. The total EMG intensities when the tissues were exposed to pulses at a 13.1-Hz input frequency ($n = 20$) were 1.2 ± 0.1 , 1.3 ± 0.1 , 2.2 ± 0.6 , and 2.1 ± 0.7 times the intensity of quiet EMG for the tibialis anterior, medial gastrocnemius, rectus femoris, and biceps femoris, respectively. Responses to the pulsed vibrations thus occur as increases in the total intensity of the 50-ms preactivation EMG activity and in particular an increase in the intensity at the high-EMG-frequency band. The results from the pulsed-vibration trials thus support the hypothesis that when the frequency of the input force is close to the natural frequency of the soft tissue masses there is an increase in the muscle activity within that tissue.

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Table 4.

The EMG intensity during the 50-ms pre-activation periods for the pulsed impacts relative to the EMG intensity during the quiet period

DISCUSSION

Peaks in the EMG intensity occurred when the vibration input frequency was close to the natural frequency of each respective soft tissue (Fig. 5). However, the response of the power dissipation and the natural frequencies of the soft tissues were not equivalent (Figs. 5 and 6; Table 4). The response to the vibrations during this experiment consisted of a sharp peak in the power-dissipation curve, which corresponded to an increase in the damping of the vibration when the input frequency was close to the natural frequency of the soft tissues. The natural frequencies of the soft tissues, on the other hand, showed much broader and smaller peaks, which did not follow the changes in the input frequency. It would appear from these results that the muscle tuning reaction to the vibration input was primarily one of an increase in the vibration damping, and the changes in frequency may have been a consequence of the altered muscle activity. Similar results have been shown for vibration damping in the horse leg, where digital flexor muscles are implicated in damping high-frequency vibrations while only causing a minor change in the frequency characteristics of the limb (37).

Limitations to the experimental design.

The experimental procedure was physically demanding for the subjects. For this reason only athletic persons were chosen as test subjects. Nevertheless, one subject showed visible muscle tremors that were presumably the result of fatigue. The results from this subject were excluded from further analysis. To minimize fatigue-related effects, the order in which the vibration stimuli were presented was randomized into a repeated-block design. If muscle fatigue occurred toward the end of the experiment, then this randomized design should remove possible bias from the results.

The amplitude of the vibrations was varied between test frequencies so that the resultant powers of the input vibrations were the same across all frequencies. This design was chosen so that the power dissipated owing to the vibrations could be compared across the range of inputs. However, measurements of the platform displacements using an accelerometer showed that the displacement at the 10-Hz input frequency was greater than required, resulting in a greater input power. For this reason, if a subject were to show the greatest power dissipation in response to the 10-Hz input, we could not be sure whether this reflects the greater power in the input or a greater response of the body. However, every time there was a peak in the response at frequencies greater than 10 Hz, we can be sure that this is a response of the soft tissues of the lower extremity to the input vibration. It is possible that subjects who did not show a response at frequencies greater than 10 Hz may have responded to inputs of 10 Hz or less.

The power dissipated by the soft tissues was calculated according to the assumption that the soft tissue moved as one rigid body. In some cases the resonance spectra for the soft tissues (Fig. 4) showed that there were a number of distinct resonant frequencies. This indicates that the soft tissues vibrated as a continuum mass rather than a rigid body. The power dissipated from the soft tissues was calculated across all frequencies from the measured vibration and therefore accounts for the power dissipated at all distinct frequencies. However, waves of vibration will travel within a flexible structure in addition to its gross motion. Damping of these waves involves additional power dissipation that cannot be accounted for by using this experimental design. The values presented from this study may thus be underestimations of the actual power dissipated by the soft tissues. To account for power dissipation from waves within the tissue, future experiments would require acceleration measurements from multiple sites across each soft tissue.

Mechanisms for muscle tuning.

The body relies on several mechanisms to regulate shock transmission through the body in response to impact forces: bone, cartilage, synovial fluids, soft tissues, joint kinematics, and muscular activity. Of these mechanisms, the body has immediate control over the joint kinematics and the muscle activity. The purpose of this study was to test damping mechanisms that can be altered by the body and thus that can be actively tuned to the excitation conditions. Impact energy can be dissipated from the knee and ankle joints because of joint compliance (14, 19), and this compliance can be altered by changes in joint angle and the forces of muscles crossing the joint. The vibration characteristics of the soft tissues change with joint angle as a result of changes in both muscle force and passive tension in structures such as the skin and connective tissue (31). To maintain joint compliance as constant as possible, the knee and ankle joint angles were controlled before each set of vibrations, and the subjects were required to stand with a static posture. Therefore, the predominant active process that changed in response to the presented vibrations was the muscle activity that changed the vibration characteristics of the soft tissues.

The results from this study demonstrate that the soft tissues of the lower extremities vibrate in a damped manner after exposure to pulsed or continuous vibrations. In order for the soft tissues to vibrate in such a manner they must have viscoelastic properties. Two major components within the muscle-tendon units that contribute to the stiffness of the system are the tendon and the attached cross bridges within the muscle (22, 26). Mechanical energy can be stored and returned from the elastic structures of the tendon and the attached cross bridges during soft tissue vibrations. Damping of the vibrations results in a net dissipation of mechanical energy. Damping may occur at the structural or material levels of the soft tissues. The muscle structure changes during force production as more cross bridges are attached, and the damping coefficients of the soft tissues increase with muscle force (31). Energy is absorbed during high-frequency vibration of activated muscle (11) that is not absorbed by muscle in rigor (17). Thus energy absorption by the muscle during vibrations can be attributed to the detachment and cycling of cross bridges. Such dissipation of mechanical work by the muscle appears as vibration damping in these experiments.

The power dissipated by a muscle during vibration depends on the amplitude of the vibration and the kinetics and the number of attached cross bridges. Cross-bridge models have predicted that the digital flexor muscles in the horse can dissipate 2 J per cycle during a 10-Hz vibration (37), which equates to 20 W/kg muscle. In this study, the muscle mass-specific peak power dissipations (Fig. 5) were 1.56 ± 0.16 , 2.29 ± 0.16 , 2.29 ± 0.16 , and 2.30 ± 0.11 W/kg for the tibialis anterior, triceps surae, quadriceps, and hamstring muscles, respectively. In this study, the lower extremity muscles were not fully activated, and so lower values for power dissipation would be expected than for the horse model in which it was assumed that power dissipation occurred from all cross bridges. The power dissipated in this study is at a level that can be explained by cross-bridge kinetics within the muscle.

Muscle response to vibration.

Bursts of muscle activity occurred immediately before the onset of platform displacement during the pulsed-vibration experiments. These bursts of muscle activity did not occur before the first burst of vibration of each set, but they occurred for the subsequent bursts 2-7 (Fig. 6, C and D). When a muscle is activated, it takes time before the muscle force is fully developed, and for isometric twitches in mammalian slow muscle this time is in the range 58-110 ms (6). Because of the time delay between the onset of muscle activation and the development of muscle force, the muscles must be activated before an expected action occurs. In this study, muscle preactivation occurred as an anticipatory response to the pulsed impacts. Similar muscle preactivations have been observed in response to cyclic heel-strike impacts from a human pendulum experiment (36) and before landing from falls of different heights (28). The EMG intensity during the 50-ms preactivation period before each burst of vibration occurred predominantly in the high (150-300 Hz)-EMG-frequency band (Table 4). Myoelectric signals within this high-frequency band have been shown to be characteristic of fast motor unit activity (33, 35). It is, therefore, likely that the muscle activity required for the vibration-tuning task in the preactivation period of this study was from predominantly the faster motor units of the lower extremity muscles.

If the contractile element remained isometric during the vibrations, then it could not perform or dissipate any mechanical work. Small changes in

muscle strain, however, would allow the contractile element to dissipate power by cyclically generating force while it was stretched and relaxing that force while it shortened. Such a mechanism would only be possible if the muscle could modulate its force at the same frequency as the vibration frequency. For example, the period of a 13.1-Hz cycle of vibration is 76 ms, and the muscle must be able to perform a contraction and relaxation cycle within this time if it is to dissipate power from such an oscillation. Mammalian slow-twitch fibers have slow contraction-relaxation kinetics and slow rates of myosin ATPase activity (1) with, for instance, contraction times for the cat gastrocnemius muscle ranging between 58 and 110 ms (6). Such long contraction kinetics would reduce the effectiveness of slow-twitch muscle fibers from dissipating power from the vibration stimuli in this experiment. However, fast-twitch muscle fibers can have twitch contraction times as short as 20 ms (6) and so would be better suited to the dissipation of the vibration power. This prediction supports the observations from this study that the muscle tuning response to the pulsed impacts occurs as an increase in the EMG intensity in the high-EMG-frequency band, which likely corresponds to the fast motor unit activity.

A burst of muscle activity occurred immediately after each impact during the pulsed vibrations. The peak EMG intensity in these postimpact events occurred ~80 ms after each onset of the table movement, with the initial increase in EMG occurring in as little as 50 ms (Fig. 6, A and B). These bursts of muscle activity typically occurred after every impact and were likely due to stretch reflexes. These bursts of muscle activity predominated in the low (25–75 Hz)-EMG-frequency band, which likely represents the activity of predominantly slower motor units (33). Thus the bursts of muscle activity after each impact may not generate significant force until at least 100 ms after the onset of the impact. The soft tissue vibrations following the pulsed impacts had little more than one or two cycles before they were substantially damped (Fig. 3), and there was significant damping even within the first cycle of each impact. Therefore, it is unlikely that the bursts of muscle activity that occurred after each pulse of vibration would have contributed significantly to the damping of the vibration caused by each pulse of vibration.

The muscle response to the continuous-vibration trials occurred as a general increase in the muscle activity (Table 2). The data cannot discriminate whether increases in EMG intensity occurred preferentially in the high (150–300 Hz)- or low (25–75 Hz)-frequency bands. Similarly, because of the continuous nature of the vibrations, the EMG signal from stretch reflexes cannot be discriminated from that for anticipatory, feed-forward responses. Reflex muscle contraction, the tonic vibration reflex, can be evoked by using continuous mechanical vibration (15). The tonic vibration reflex is mainly attributable to the muscle spindle Ia fibers that are able to respond to vibration frequencies of up to 200 Hz (4–5, 27). However, it has not clearly been demonstrated whether the pathways involved in the tonic vibration reflex are monosynaptic or polysynaptic. Motor unit firing patterns are phase-locked to the vibration frequency, although at higher excitation rates the motor units fire at a subharmonic of the excitation frequency (3,16). The motor unit discharge rate during tonic vibration reflexes in the triceps surae reaches 10 Hz, so at higher excitation frequencies not every cycle of vibration elicits a motor unit response (3). Dissipation of vibration power requires that the muscle performs net negative work, i.e., that it produces more force during lengthening than during shortening. Therefore, if power is to be dissipated, the phasic muscle activation during the tonic vibration reflex must satisfy the requirements that the muscle generates force at the correct time relative to the vibration.

Soft tissue vibrations during walking and running.

Impact forces during heel-toe running typically have a maximum between 10 and 30 ms (7, 23) and a major frequency component between 10 and 20 Hz. This frequency range spans the natural frequencies of the soft tissues measured in this study, which were ~15 Hz. Thus there is potential for vibrations in the soft tissues to occur as a result from the impact forces. Shoe material hardness alters the loading rate of the impact force during running (12, 18, 20,25) and thus may alter the tuning requirement for each soft tissue. Indeed, it has been shown that the muscle activity required for tuning the tissues to the impact force changes when the loading rate of the impact force was altered on a pendulum experiment (36) and also for overground running (34).

The results from this present study showed that the mechanical properties of the soft tissues in the lower extremities were tuned in response to the frequency content of each vibration input. The experimental protocol was designed to mimic the frequencies of actual impact forces that occur during running. However, the experiment consisted of a static task and not the dynamic movement of a locomotor activity. It remains to be seen whether similar changes in frequency and damping occur during running. The results from this experiment also show that the muscle activity patterns in the lower extremity are modified as a response to changes in the excitation frequency of input signals (e.g., impact forces). Such changes can be achieved with the material properties of a shoe midsole (12, 18, 20, 25). However, further work needs to be done to identify the natural frequencies that occur in the soft tissues at heel strike during walking and running.

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