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Fatigue-Related Loading Imbalance on the Shank in Running: A Possible Factor in Stress Fractures

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Abstract—In previous reports we have shown that in long distance running the impact acceleration on the shank increases with progressing fatigue. The aim of the present study was to test whether, in parallel to this increase, an imbalance in the activities between the ankle plantar and dorsi flexor muscles develops. The tests were made on fourteen subjects during 30 min treadmill running above their anaerobic thresholds. Respiratory data were collected to determine the anaerobic threshold speed and to indicate the progressively developing metabolic fatigue. Surface electromyogram (EMG) was monitored to indicate the changing activity of the shank muscles. In the tibialis anterior the average integrated EMG (iEMG) and the mean power frequency (MPF) significantly decreased from the beginning to the end of running. In the gastrocnemius iEMG did not change, while MPF increased during the course of running. The impact acceleration, measured by means of an accelerometer attached to the tibial tuberosity, significantly increased during the course of running. It was concluded that, with developing fatigue, an imbalance in the contraction of the shank muscles develops in parallel to an increase in shank shock acceleration. The combination of these two changes may hamper the loading balance on the tibia since the bone becomes exposed to excessive bending stresses and to higher risk of stress injury. © *2000 Biomedical Engineering Society.*
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Keywords—Impact acceleration, Tibial injury, Bone load, Tibialis anterior, Gastrocnemius.

INTRODUCTION

Fatigue, or stress, fractures occur in bones in response to repetitive stresses over multiple cycles, when the body's ability to adapt is exceeded.[3,5] In athletes 72% of the stress fractures were reported in running, 60% of which in long and middle distance running.[14] Of all the stress fractures in athletes, 50% were reported to be in the tibia, between the middle and distal thirds of the shaft.[20] An important factor which affects the incidence of bone stress injury, is exposure to abrupt changes in the bone loading,[3] and consequent alteration in the strain distribution[36] with insufficient recovery periods.[25] Other factors include footwear, terrain, and intensity of activity or training.[3] Clinical and experimental evidence suggests that the stress injury takes place at the site in which the maximum tensile stress due to bending is present.[7,16] This is explained by the fact that the fracture (or ultimate) stress is lower in tension than in compression[35] and that cracks tend to open in tension and close in compression. While the mechanical process of material fatigue is well determined,[26] fatigue failure of bone in life involves also complex physiological and neuromuscular responses.[27] For instance, contraction of muscles on the tensile side of a bending bone acts to limit the tensile strains there, helping to protect the bone from fracture risk.[2,21] It has been hypothesized that muscles also act as shock absorbers and that muscle fatigue can reduce the dampening effect and accelerate the initiation of stress fractures.[3,6,16,27] Recent studies have shown that, as a result of muscle fatigue, there is an increase in the strain rate (i.e., rate of strain development) in the tibia, rather than in the maximal strain,[12] suggesting that loading of the tibia during running becomes more impulsive as fatigue progresses.

Information of the impulsive loading on the bone in long-distance running can be obtained noninvasively either by means of the foot-ground reactive forces[9] or, directly, by measuring the transient accelerations on the shank caused by impact. By attaching an accelerometer externally on the tibial tuberosity, it has been shown that the impact acceleration increases with developing of muscle fatigue.[18,19,31,32] Fatigue can be either global or local in nature. Global or metabolic fatigue is associated with the development of metabolic acidosis following an endurance exercise and is accompanied by a decrease in the end-tidal carbon dioxide pressure ($PETCO_2$).[34] In long-distance running metabolic fatigue is reached when the running speed exceeds the anaerobic threshold.[34] Local fatigue in a muscle takes place as a result of an intensive activity of this muscle and is reflected by certain changes in its electromyogram (EMG) signal in either the time or frequency domains.[10]

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One important question is whether, as a result of fatigue, an imbalance between the activities of the plantar and dorsi flexor muscles of the ankle develops. Such an imbalance would compromise the protective action provided by the muscles to the shank.[2,21] The few published works on fatigue of the muscles of the leg have dealt mainly with short-distance running.[22] In long-distance running, studies can be found on fatigue of the quadriceps[8,18] but not of the ankle flexors. Thus, to address the earlier question, information on the possible development of local fatigue of the ankle flexors in long-distance running is of great interest. The aim of this study was, therefore, to test the hypothesis that with the progressing of muscle fatigue in long-distance running, an imbalance in the activities between the ankle plantar and dorsi flexor muscles develops in parallel to the increase in the shank impact acceleration.

METHODS

Subjects and General Procedure

Fourteen male subjects of 24.2±3.7 years of age, height 175.5±5.9 cm, leg length 90.0±3.0 cm, and body mass of 73.2±8.3 kg volunteered to participate in this study. The subjects, from the student population of the Technion, were recreational runners who, during the three months prior to the tests, have been regularly running for 8–10 km per week at a speed of 12 km/h approximately. The subjects were in an excellent state of health and, prior to the experiment underwent a general medical examination including resting electrocardiogram (ECG) and blood pressure (BP). The pretest ECG and BP were normal for all subjects. The subjects had no previous histories of any of the following: obesity, muscle weakness or injury, bone disease or injury, neurological diseases, drug consumption, or therapy. Each subject provided informal consent according to the local ethical committee's guidelines of the Technion.

To assure uniformity of the testing conditions, all subjects were provided with similar plain running sneakers, with no anatomical support for the foot's arch. Additionally, and as will be described later in the article, kinematic measurements were made to verify that the running style was uniform between the runners and that the pattern of running compared well with that reported in the literature.

Respiratory Data and Running Protocol

During running, the subject breathed through a mouthpiece attached to a turbine device. The respired gas was continuously sampled by a SensorMedics 4400 metabolic cart (Alpha Technologies, Inc., Laguna Hills, CA) for breath-by-breath determination of the gas exchange and ventilatory variables. The instrument was calibrated before every test using a standard 3 l syringe and precision reference gases.

Exercise values for oxygen consumption ($\dot{V}O_2$), minute ventilation ($\dot{V}E$), carbon dioxide production ($\dot{V}CO_2$), $PETCO_2$, ventilatory equivalent for oxygen ($\dot{V}E/\dot{V}O_2$), and ventilatory equivalent for carbon dioxide ($\dot{V}E/\dot{V}CO_2$) were calculated as an average of the breath-by-breath data during a time span of 30 s. $PETCO_2$ was used as a measure for the assessment of global fatigue due to the development of metabolic acidosis as it is considered an established and reliable measure for indicating both the anaerobic threshold (AT) and global fatigue.[34] It is also more sensitive than the changes in lactic acid during 30 min running.[15]

The running tests were performed on a treadmill (Quinton Q55, Seattle, WA) to allow repetitive monitoring of the data. Prior to each running test, the subjects underwent a 15 min warming-up running at an individually selected and comfortable speed to get used to the treadmill and the measuring equipment. One week prior to the experiment, a pretest was conducted for each subject to determine the AT using the following procedure. The subject was exposed to an incremental load by increasing the running speed on the treadmill from an initial 8 to a maximum of 15 km/h. The speed increments were of 1 km/h every 2 min and were given until $PETCO_2$ reached the decline phase. The decline phase was confirmed if it persisted for at least 1 min. AT was then determined as the point of initial increase of $\dot{V}E/\dot{V}O_2$ and $\dot{V}E/\dot{V}CO_2$, which just precedes the initial decline of $PETCO_2$.[34] The running test was performed for a duration of 30 min at a steady speed exceeding the AT level of each subject by 5%. In a previous study, we have shown that 30 min running at a speed exceeding the AT is a sufficient time to induce general fatigue.[31]

Heel-Strike Induced Impact Accelerations

A light-weight (4.2 g) accelerometer (Kistler Piezo-Beam, type 8634B50, Kistler, Winterthur, Switzerland), connected to a coupler (Kistler Piezotron, type 5122) was used. It was externally attached by a metal holder (1.4 g) above the tibial tuberosity of the right leg of each subject and was aligned with the longitudinal axis of the tibia to provide the axial component of the shank acceleration. The accelerometer was pressed onto the skin in closest position to the bony prominence of the tibial tuberosity by means of an elastic belt around the shank. The tension of the belt as measured by a spring dynamometer was approximately 50 N, which corresponds to an accelerometer preload of about 25 N. This was well above the 14 N level we found in which the acceleration trace for a given impact force became insensitive to the accelerometer attachment force, thus ensuring reproducibility of the measurements.[17]

The signals from the accelerometer were fed to the PC-based data acquisition system at a sampling rate of 1667 Hz per channel and stored for off-line processing. A high sampling rate was required to determine accurately the levels and timings of the spike acceleration resulting from foot strike. The acceleration data were acquired every 5 min for a time span of 20 s. Thus, information about 27 foot strikes was provided for each time span on the measured foot. In order to exclude possible running modifications, the subjects were not aware of when exactly the data were acquired. Based on the known running speed and the timing of the first foot strike, the developed software automatically detected the occurrences of the foot strikes and acceleration amplitudes. Since there was a possibility of unexpected errors (e.g., subject accidentally stumbled or misplaced the foot), all results were shown on the monitor and confirmed by an operator. The criterion for the elimination of data due to such errors was a deviation of more than three standard deviations from the mean. The acceleration signal was analyzed both in the time[18,31,32] and frequency domains.[19,28,32] In the time domain, the impact acceleration was defined as the maximal amplitude of the accelerometer transient at foot strike.

In the frequency domain, fast Fourier transform of the stance phase of the acceleration signal was carried out to determine the power spectral density (PSD).[19] PSD represents the amount of power within the signal per unit frequency. An eighth-order cascaded, 40 Hz low-pass Butterworth filter was used to remove the noise from the accelerometer signals. Apart from the PSD versus frequency plots, the median frequency (MDF) was reported to significantly shift towards the higher frequencies in fatigue compared to non fatigue condition.[19,32]

EMG Monitoring

The myoelectric activity of the tibialis anterior and gastrocnemius muscles was monitored during running. These are considered to be major dorsal and plantar flexors of the ankle,[1] respectively, and have been reported as functionally antagonists.[23,37] Two pairs of small bipolar disposable Ag/AgCl snap surface electrodes (10 mm diameter, Promedico Ltd.) were used and the signals were routed to an eight-channel surface electrode system (Atlas Research Ltd.). The amplifiers, powered by a PC, were fully electrically isolated to comply with the UL 544 safety standards.[30]

The EMG data were collected at a sampling rate of 1667 Hz per channel every 5 min for a time span of 20 s. Since the EMG and acceleration data had to be collected simultaneously, an electrical trigger was used to synchronize between the two sets of data. Processing of the integrated EMG (iEMG) and of the mean power frequency (MPF) was carried out in a similar way as earlier described.[18]

Kinematics of the Segments of the Lower Limb

Videography was used to confirm uniformity of the running pattern. However, due to its low sampling frequency (50 frames/s) it could not provide accurate information on the foot strike with the ground. On the other hand, it did supply information on the running style as well as on the displacement and the low-frequency motion of the subject. Data from five markers on the right leg were collected in the sagittal plane using a NV-M3000EN Panasonic camera (50 frames/s). Digitization, time histories, and smoothing of the data were accomplished by using Ariel Performance Analysis System (APAS) software and a fourth-order Butterworth digital filter (zero-lag) with a cutoff frequency of 10 Hz. An external trigger was used to synchronize between the accelerometer, EMG, and video data.

Statistical Analysis

Running data at each of the 5th, 10th, 15th, 20th, 25th, and 30th min of running were compared to those of the beginning of running. These data included $PETCO_2$, shank impact acceleration, and EMG of the tibialis anterior and gastrocnemius muscles. The possible effects of the variances in other variables, such as fitness level and anthropometrics were considered noninfluential. Differences were tested using Analysis of Variance (ANOVA) with posthoc Scheffe's test. Least squares linear fits were made to the data versus running time. Statistical significance was determined at $P<0.05$.

RESULTS

Running Style and Speed

The average of the individual running speeds for all the subjects tested was 12.71 (SD=0.68) km/h. All the runners had a heel-toe running style. Figure 1 presents a typical output of the shank acceleration, and of the iEMG of the gastrocnemius and tibialis anterior muscles in the 1st min (nonfatigue) and in the 30th min (fatigue). A stick diagram of the leg is presented in the lower part of the figure, showing one running cycle of the right leg. Kinematic results in the nonfatigue condition were found to compare well with published data from tests under similar conditions: average knee angle at cushioning flexion 45.5 (SD=3.0) deg, compared to 43.9 (SD=3.6) deg;[4] stride rate 1.41 (SD=0.05) l/s, compared to 1.41 (SD=0.09) 1/s.[8]

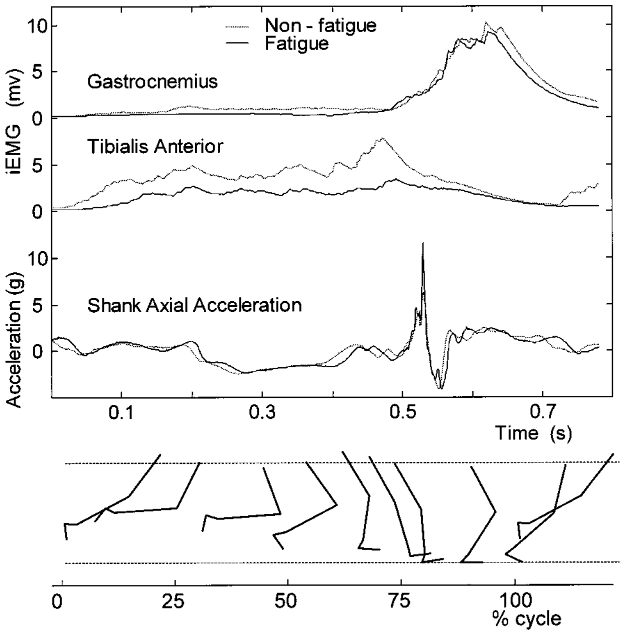

FIGURE 1. Typical one-stride output for one subject of the shank axial acceleration and of the iEMG of the gastrocnemius and the tibialis anterior in the 1st min (nonfatigue, dotted line) and in the 30th min (fatigue, solid line) of running. A stick diagram of the leg is presented in the lower part of the figure, showing one running cycle of the right leg.

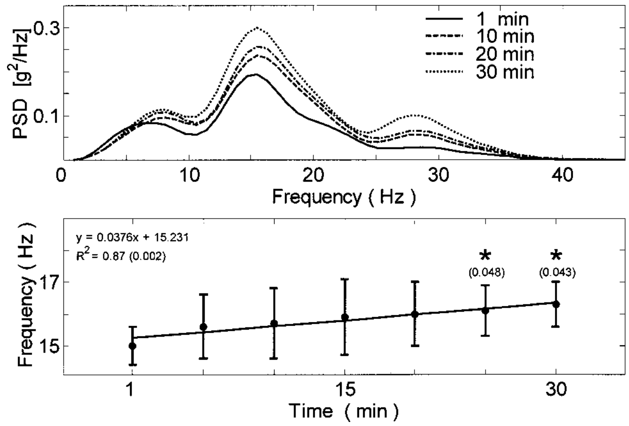

FIGURE 3. Average for all 14 subjects of the PSD profiles of the shank accelerometer traces during the stance phase of running. Four time stages of a total of 30 min running are demonstrated (top). The MDF is shown at the bottom part of the figure. Vertical bars represent standard deviations and asterisks represent statistically significant differences (P in parentheses) compared to the values of the 1st min of running. Linear regressions show an increasing trend of the MDF.

$PETCO_2$ *and Impact Acceleration*

Summarized values of the average $PETCO_2$, and impact acceleration on the shank are presented in Fig 2. $PETCO_2$ decreased significantly from the 30th min compared to the 1st min ($P=0.045$), indicating that global fatigue developed. The impact acceleration at heel strike increased significantly from the 15th min, compared to the 1st min ($P \leqslant 0.048$).

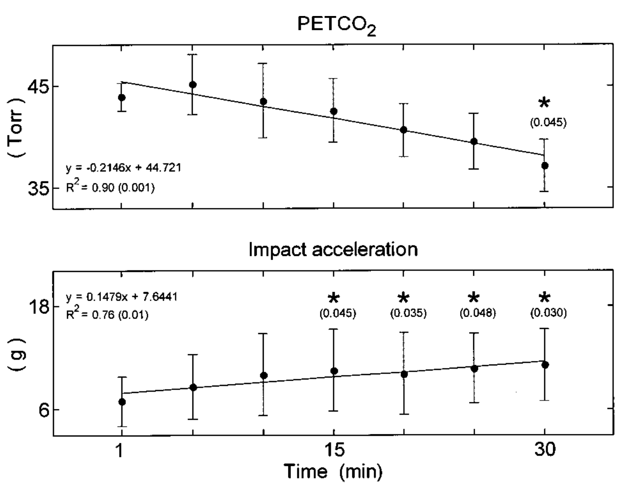

FIGURE 2. Average for all 14 subjects of $PETCO_2$ (top) and impact acceleration on the shank (bottom). Vertical bars represent standard deviations and asterisks represent statistically significant differences (P in parentheses) compared to the values of the 1st min of running. Linear regressions show a decreasing trend for $PETCO_2$ and an increasing trend for the impact acceleration.

Figure 3 presents the results of the spectral analysis of the accelerometer signal. The top part of the figure shows the average of the PSD of the accelerometer traces during the contact phase of running at four time stages. In the first minute of running, the PSD demonstrates a two-peak pattern, in accordance with previous reports.[19,28] The impact peak (12–20 Hz zone), higher than the active peak (4–9 Hz zone), is associated with the high-frequency content of the foot-ground impact and corresponds to the initial 15% of the stance phase. As fatigue develops, in the later stages of running, an additional peak within the 25–35 Hz range level becomes noticeable, in parallel to the increase in the peak of the impact region. The possibility that this increase was an artifact is ruled out since the resonance frequency of a 4 g skin-mounted accelerometer is well above 35 Hz.[29] The changes in the average peak frequencies from the first to the 30th min of running for both the active and the impact frequency regions were not significant. Nevertheless, the average MDF of the PSD acceleration signal in the shank significantly increased from the 25th min of running and onwards ($P \leqslant 0.048$) as compared to the beginning of running (bottom part of Fig. 3).

EMG

Averages of the iEMG data of the gastrocnemius and tibialis anterior muscles are presented in Fig. 4. Compared to the 5th min, the change in the gastrocnemius iEMG was insignificant as fatigue developed. On the

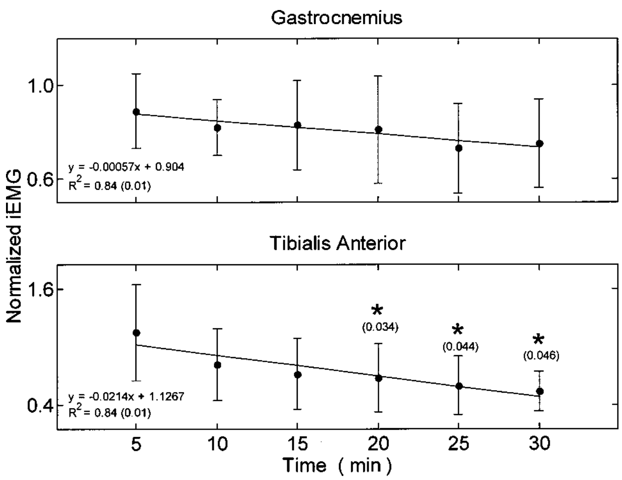

FIGURE 4. Average for all 14 subjects of iEMG for the gastrocnemius (top) and tibialis anterior (bottom) during 30 min running. Vertical bars represent standard deviations and asterisks represent statistically significant differences (P in parentheses) compared to the values of the 1st min of running. Linear regressions disclose decreasing trends of the iEMGs of the two muscles.

other hand, the tibialis anterior iEMG significantly decreased from the 20th min onwards compared to the 5th min of running ($P \leqslant 0.046$). The MPF of the EMG data of the gastrocnemius and the tibialis anterior muscles are presented in Fig. 5. MPF of the gastrocnemius significantly increased from the 1st min to the 30th min ($P = 0.049$). MPF of the tibialis anterior significantly decreased from the 20th min and onwards compared to the 1st min of running ($P \leqslant 0.048$).

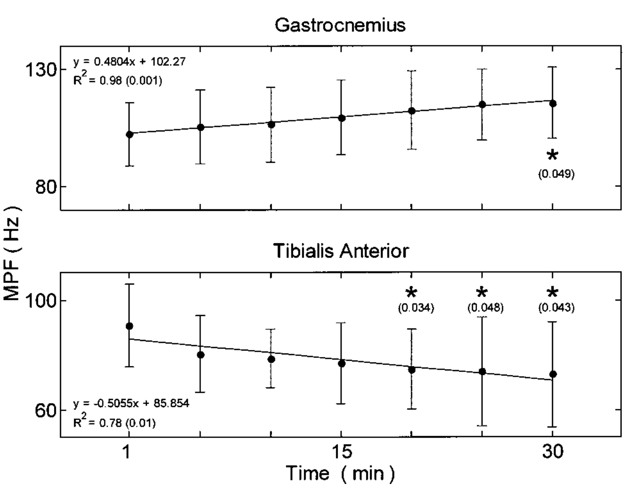

FIGURE 5. Average for all 14 subjects of MPF for the gastrocnemius (top) and tibialis anterior (bottom). Vertical bars represent standard deviations and asterisks represent statistically significant differences (P in parentheses) compared to the values of the 1st min of running. Linear regressions demonstrate an increasing trend for the gastrocnemius muscle and a decreasing trend for the tibialis anterior.

DISCUSSION

Stress fractures in long bones of the lower limbs are believed to originate from repetitive and/or excessive loading, such as may take place in long-distance running at a speed exceeding the anaerobic threshold. In the present study the average running distance per test was 6.35 km (30 min of running at the average speed of 12.7 km/h) in agreement with the definition of ''long distance''.[33] We have measured and analyzed the following: respiratory data and muscle EMG to monitor global and local fatigues, respectively; videography to confirm uniformity of the testing conditions and of the kinematics and accelerometry, to provide quantitative information on loading of the shank. Accelerometry is an advantageous method due to its being noninvasive. Its major drawback, however, is that it is not directly related to bone stress, as in the case of strain or strain rate measurements which are invasive.[12,36] We have addressed two major fatigue-related factors taking part in exposing the shank to stress fractures risk. One is increased intensity and median frequency (MDF) of the impact acceleration on the shank accompanying the decline in $PETCO_2$, the latter expressing metabolic fatigue.[34] The second is the increasing imbalance between the ankle's antagonistic flexor muscles resulting from changes in the activities of these muscles. While the first of these issues has been treated in previous studies,[18,19,31,32] no reports were found in the literature on the second.

The question whether the disturbance in activation between the agonistic and antagonist ankle muscles combines with the increased impact acceleration on the shank is an important issue due to the following reason. Muscles have an important role in bone loading, particularly bending.[2,21] When purely bent, one surface of the bone is subject to compression and the opposite surface to tension. Since bone is weaker in tension than compression,[35] it should be of interest to protect the bone from excessive tensile stresses.[13] Co-contraction of antagonistic muscles do help in providing that protection by (a) compound bending, i.e., converting nonaxial bending stresses into more axial and compressive stresses, therefore lowering the tensile stresses on the bone;[2,21] (b) stabilizing the lower leg at heel strike while loading occurs;[11] and (c) serving as effective shock absorbers to lessen the impact on the shank due to the initial heel contact.[16] Thus, when imbalance between the muscles develops and the muscles that span the tensile surface of the bone become less active than those of the opposite side, the result is a decrease in the protection abilities of the muscles.

The results obtained in this study revealed an increased MDF of the acceleration signal that indicated that during running the heel strike became more impulsive as general fatigue developed. Impact intensity also increased. As previously suggested, this may result in an increased fracture risk due to the muscles' inability to dissipate and attenuate the heel strike induced impact acceleration.[16] Imbalance between the muscles was found in this study to develop simultaneously with impact acceleration thus exposing the bone to an enhanced risk of stress fractures, confirming the hypothesis behind this study.

The development of loading imbalance was demonstrated by the myoelectric signals of the gastrocnemius and tibialis anterior muscles. The iEMG of the gastrocnemius muscle did not change suggesting a maintained activity of this muscle. This situation can be possible due to an enhanced firing rate of the working motor units, as reflected by the increase in the MPF, as was the case in our results. In the tibialis anterior muscle, however, both MPF and iEMG decreased substantially. The resulting reduction in the number of active motor units (reflected by the iEMG decrease) and in the motor unit firing rate (reflected by both decreases in MPF and iEMG) indicates that activity of this muscle is reduced due to fatigue.[10] It should be pointed out that, due to its relatively high rate of sustained activity in the running cycle, the tibialis anterior muscle is susceptible to overload fatigue.[24]

Thus, the mechanical consequence of fatigue in long-distance running is two fold: enhanced impact acceleration due to global fatigue and muscle activity imbalance due to local fatigue before and during foot contact, resulting in the development of excessive tibial bending stresses and higher risk of stress injury.

CONCLUSION

Fatigue in long-distance running at a speed exceeding the anaerobic threshold involves a gradually increasing impact loading on the shank and an imbalance in contractions of the muscles acting on the shank. The combination of these two conditions may hamper the loading balance on the tibia since the bone becomes exposed to higher bending stresses and higher risk of stress injury.

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