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Acute and Subacute Changes in the Ultrasound Measurements of the Calcaneus Following Intense Exercise

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Abstract. The amount of exercise necessary to cause bone structural change in humans is unknown. We examined whether a single bout of intense exercise *in vivo* leads to acute and subacute changes in the physical properties of bone as measured by ultrasound. It was hypothesized that structural changes such as accumulation of fatigue microdamage would result in a decrease in velocity of sound (VOS) and broadband ultrasound attenuation (BUA) across the calcaneus. We performed a prospective cohort study in 111 (97 M, 14 F) entrants of the 1996 Melbourne marathon (42.3 km) and 28 (10 M, 18 F) nonrunning controls. Runners had a mean (SD) age of 45.3 ± 11.4 years (range 20–75), had completed 15.2 ± 17.3 prior marathons (0–88), and had been running regularly for 14.2 ± 9.2 years (0.25–50). An ultrasound densitometer (Cuba Clinical, McCue) was used to measure VOS and BUA across the right calcaneus. Runners were tested on three occasions: 1-3 days prior to, immediately after (<2 hours), and 5-6 days following the marathon. Seventy-three (66%) runners presented for all three measurements. Controls were tested on three occasions with the same time intervals as the runners. BUA values in the runners were significantly elevated by 5.0% immediately after the marathon but returned to baseline levels by the third test session ($P = 0.0001$). Changes in BUA values in the controls were not significant and all were less than 0.7% ($P = 0.88$). Age was a significant independent predictor of the BUA change between test 1 and test 2 in the runners ($\beta = 0.2094$; SE = 0.0917; $P = 0.03$). VOS measurements were not significantly different across the three testing sessions in both the runners ($P = 0.07$) and the controls ($P = 0.33$). Therefore, ultrasound measurements of BUA and VOS did not detect evidence of lasting structural change in the calcaneus following a marathon.

Key words: Exercise — Bone microdamage — Ground reaction force — Ultrasound.

During weight-bearing physical activity, forces are generated within the skeletal system as a result of impact with the ground and muscular contraction [1, 2]. Vertical ground reaction forces (GRF) have been shown to vary from 2.5 times body weight during running [3] but up to 12 times body weight during jumping and landing activities [4, 5]. Transient impulse forces associated with GRFs are then propagated up the lower limb, undergoing attenuation as they pass toward the head [6, 7]. The ability of the skeleton to withstand such loads without significant structural damage is related to, among other factors, the capacity of bone to remodel according to its functional requirements. However, accumulation of microdamage can occur if loading is excessive. In athletes, such a sequence is ultimately responsible for the clinical entity of a stress fracture.

In vitro studies have shown that bone specimens subjected to cyclic fluctuating loads undergo gradual mechanical failure [8, 9] consistent with the development of microscopic cracks within bone. Animal models have revealed the existence of bone structural damage following repetitive loading using physiological strains *in vivo* [10, 11]. Thus, microdamage may be a normal phenomenon in humans.

The amount of loading needed to elicit structural damage is unclear although it is likely that a threshold level exists. Significant microdamage was produced in dog forelimbs subjected to three-point bending at 1500 or 2500 microstains for 10,000 cycles, but not when subjected to lower strain or fewer cycles [12, 13]. The threshold may be approximately 2000 microstrain, the upper range of human physiological values [14]. Therefore, the intense training of many athletes might be expected to result in microdamage at maximally stressed bone sites. Measurement of ultrasound (US) parameters may provide a useful, noninvasive, nonionizing means of measuring the accumulation of bone structural changes in exercising individuals.

Ultrasound has been used as a tool for estimating the biomechanical competence of bone [15]. This is related to the fact that mechanical properties of bone alter the shape, intensity, and speed of the propagating sound wave. Commercially available US devices mainly measure velocity of sound (VOS) and/or broadband ultrasound attenuation (BUA). VOS has a quantitative relationship to the stiffness and mass density of bone and although no comparable

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physical model exists for BUA, there is evidence to show that it is also related to these and other bone properties [16–18]. Clinically, ultrasound measures have been found to correlate moderately with bone density values obtained by X-ray absorptiometry techniques [19], to discriminate between individuals with and without a history of osteoporotic fracture [20] and to detect changes in bone properties following intervention [21].

Rubin et al. [22] used transcutaneous US to assess acute changes in the physical properties of the tibia and patella following a marathon. The results were contrary to what the authors hypothesized, with significant increases in US velocity measured immediately after the period of intense weight-bearing activity. The mechanism for this unexpected, acute increase was not fully elucidated and lack of subacute follow-up does not allow one to evaluate whether the changes were transient. Furthermore, a control group was not included to assess the temporal stability of measurements over a similar time frame.

Therefore, based on the questions raised by the study of Rubin et al. [22], we aimed to assess the acute and subacute changes in US measurements of the calcaneus following a marathon. We reasoned that if structural changes were produced in bone as a result of intense exercise, alterations in US bone properties would be detected at both the acute and subacute phases. The calcaneus was chosen as it is subjected to high forces due to impact with the ground during running. Over the course of a marathon, the heel is likely to contact the ground around 30,000 times.

Materials and Methods

Subjects

A prospective, cohort study was performed on 111 (97 M, 14 F) entrants in the 1996 Melbourne Marathon (42.3 km) who volunteered for this study. This number represents 7.2% of the total registrants. Runners were recruited via flyers included in the race package mailed to all registrants and via advertisements in running club newsletters. Subjects had a mean (SD) age of 45.3 ± 11.4 years (range 20-75), a mean height of 174.4 ± 11.4 cm (range 150-193), and a mean weight of 71.5 ± 10.2 kg (range 46-95). Subjects commenced running training at 29.4 ± 11.0 years of age (range 12-57), had been running regularly for 14.2 ± 9.2 years (range 0.25-50) and had completed 15.2 ± 17.3 prior marathons (range 0-88). In the month preceding the present marathon, the runners trained a mean of 6.3 ± 2.3 hours per week (range 2-12), running 66.4 ± 19.2 km per week (range 20-120). Of the runners, 16 ± 14.4% had a history of stress fracture at some location in the lower limb.

Twenty-eight subjects (10 M, 18 F) were recruited from University staff and students to serve as nonrunning controls in order to assess the stability of measurements in normally active individuals across the same three time intervals as the runners. The controls were a convenience sample of volunteers who responded to verbal and written advertisements. They had a mean (SD) age of 33.0 ± 12.2 years (range 19-56), a mean height of 169.6 ± 9.2 cm (range 151-192) and a mean weight of 68.0 ± 12.1 kg (range 53-100).

Approval for this project was obtained from The University of Melbourne Human Research Ethics Committee and The Royal Melbourne Hospital Clinical Research and Ethics Committee. All subjects gave written informed consent prior to commencing the project.

Transmission Ultrasound Measurements

A portable US (Cuba Clinical V2.1 software, McCue, Hampshire, UK) was used to measure VOS in m/seconds and BUA in dB/MHz across the right calcaneus, a cancellous bone site. This machine has a center frequency of 1.0 MHz and an acoustic frequency of 0.28 Wcm^{-2}. Machine calibration was checked daily prior to testing using a phantom. For subject measurements, skin over the heel was cleansed with an alcohol swab. A liberal layer of coupling gel was then applied to either side of the heel to ensure optimal contact between the skin and transducer.

Ultrasound measurements were made on three occasions in all subjects. For the runners, measurements were made (1) 1–3 days prior to the marathon, (2) immediately following the marathon, and (3) 5–6 days after the marathon. The runners were asked not to train for at least 4 hours prior to the 1st and 3rd testing occasions. For the controls, three measurements were performed with the same time intervals as the runners. The controls were asked not to perform any vigorous, weight-bearing exercise for at least 4 hours prior to testing.

Ultrasonometry *in vitro* precision was determined on a calibrated phantom (a homogenous cylinder of plexiglass with established VOS and BUA) from 17 measurements taken over the testing duration (20 days). The *in vitro* coefficient of variation (CV) was 0.6% for VOS and 1.1% for BUA. Short-term *in vivo* precision was evaluated from three sequential measurements, with repositioning between each in 20 normal, healthy subjects ranging in age from 25 to 56 years. The mean CV was 0.48% for VOS (95% CI: 0.33–0.63%) and 2.96% for BUA (95% CI:2.22–3.71%).

Anthropometric Measurements

Anthropometric techniques were used to obtain height, weight, maximal calf girth, and tibial width based on procedures outlined by Ross and Marfell-Jones [23]. Tibial width was measured with calipers at the junction of the middle and distal thirds of the right tibia.

Statistical Analysis

Comparison of VOS and BUA over the three time intervals was made separately in the runners and the controls using one-way repeated measures analysis of variance (ANOVA) followed by post hoc Scheffe F tests. Two-tailed tests were used and P values were set at 0.05.

Predictors of change in BUA and VOS from test 1 to test 2 in the runners (before the marathon and immediately after the marathon) were sought univariately using height, weight, baseline US measurements, years of running training, age commenced running, number of marathons, sex, stress fracture history, race time, current training volume, calf girth, and tibial width. Significant univariate predictors were then included in a forward, stepwise, multiple linear regression. The relationship between baseline VOS and baseline BUA was assessed in the runners and the controls using univariate correlations.

Results

Acute and Subacute Changes in US Measurements Following a Marathon

Seventy-three (66%) runners presented for US measurements on all three testing occasions. On the second testing occasion, runners were tested within 2 hours of completion of the marathon. The runners had significantly lower VOS and BUA values than the controls at all three time points ($P < 0.05$).

Results showed that BUA measurements were significantly elevated immediately after the marathon but returned to prerace levels by the 3rd testing session ($P = 0.0001$). The mean size of the elevation was 5.0% Contrary to the

Table 1. Mean (SD) for velocity of sound (VOS) and broadband ultrasound attenuation (BUA) measurements on the three testing occasions in runners and controls

US measurements	Runners (n = 73)	Controls (n = 28)
VOS (m/s)		
Test 1	1669.712 ± 41.931	1710.179 ± 51.670
Test 2	1675.182 ± 40.025	1715.429 ± 47.341
Test 3	1672.924 ± 39.950	1714.571 ± 46.775
BUA (dB/MHz)		
Test 1	87.694 ± 17.169	95.821 ± 22.909
Test 2	90.195 ± 16.758[a]	95.107 ± 21.979
Test 3	87.815 ± 16.345	95.250 ± 19.763

[a] Significantly different from Test 1 and Test 3; Scheffe F tests; $P < 0.05$

runners, BUA values in the controls remained essentially unchanged over the three tests ($P = 0.89$) with the largest change being a 0.7% decrease in BUA at the 2nd test session. The results for BUA in the runners and controls are shown in Table 1 and Figure 1.

Although VOS measurements in runners showed a marginal 0.3% increase immediately following the marathon, changes in VOS across the testing intervals were not significant ($P = 0.07$). In the controls, VOS values also increased by a mean of 0.3% between the 1st and 2nd testing occasions and returned to baseline levels by the 3rd testing occasion, but these variations were not statistically significant ($P = 0.34$). The power of the study to detect a change of approximately 1% between the first and second testing occasions was 99% in both runners and controls [24]. The results for VOS are shown in Table 1 and Figure 2.

Predictors of Acute Changes in US Measurements Following a Marathon

Significant univariate predictors of the change in BUA immediately following the marathon in the runners were age ($r = 0.23$, $P = 0.04$); years of running training ($r = 0.25$, $P = 0.03$); and baseline BUA ($r = 0.23$; $P = 0.05$). In a forward, stepwise, multiple regression, only age was a significant independent predictor of the acute change in BUA ($\beta = 0.2094$; SE = 0.0917; $P = 0.03$; $r = 0.26$). For every 10-year increase in age, there was an approximate 2.4% increase in BUA measurements immediately following the marathon. However, the strength of the relationship was low with only 7% of the variance in change in BUA able to be explained by age. The relationship between age and acute change in BUA measurements in runners is shown in Figure 3. None of the variables was able to predict a change in VOS between test 1 and test 2 in the runners.

Relationship Between Baseline VOS and BUA

The relationship between baseline BUA and VOS measurements is shown for runners and controls in Figure 4. There was a significant moderate-to-high correlation between VOS and BUA for the runners ($r = 0.79$, $P < 0.0001$) and for the controls ($r = 0.72$, $P < 0.0001$).

Discussion

We found that immediately following a period of intense weight-bearing exercise, BUA values at the calcaneus increased significantly but returned to baseline levels after 5-6 days. This acute increase was not apparent in nonrunning controls. Conversely, VOS measurements were not influenced by exercise.

The transient increase in BUA measurements does not appear to support the hypothesis that an intense bout of weight-bearing exercise leads to accumulation of bone microdamage. This concurs with the conclusions drawn by Rubin et al. [22] upon finding acute increases in VOS at both the tibia and patella. If substantial microdamage was present, any possible influences on bone US measurements would be expected to be in the direction of a decrease in values and not an increase. Also, since microdamage is normally repaired by the remodeling process which occurs over approximately 3 months, any change in US values due to microdamage should still be evident in the subacute phase. Furthermore, VOS values remained relatively stable across the testing occasions. If microdamage was present, it theoretically should alter both BUA and VOS. However, since the sensitivity of US to detect microdamage *in vivo* is unknown, it is possible that structural damage did occur but not sufficiently to alter subacute measurements.

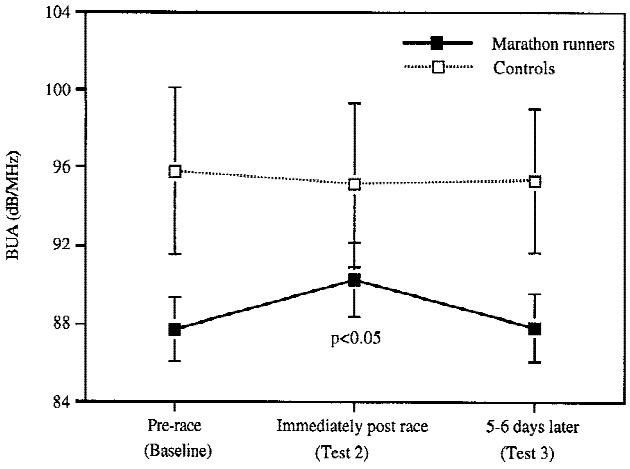

Fig. 1. Mean (SE) of BUA in runners and controls for the three testing occasions.

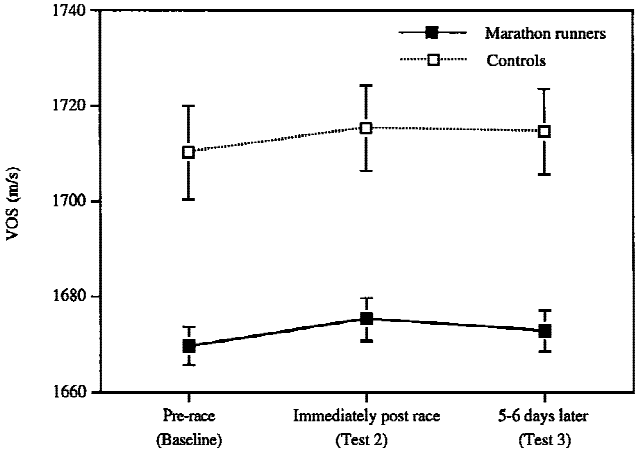

Fig. 2. Mean (SE) of VOS in runners and controls for the three testing occasions.

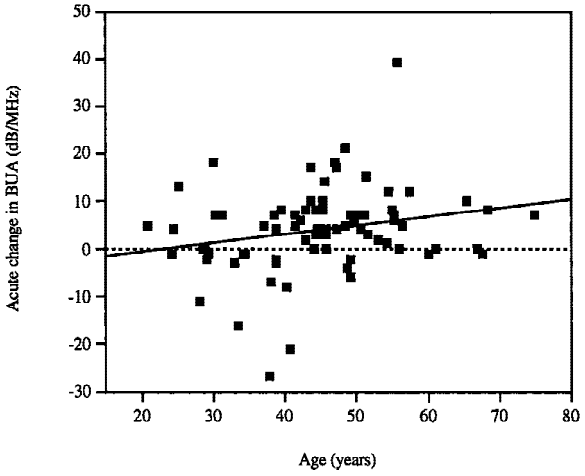

Fig. 3. Relationship between age and acute change in BUA in runners.

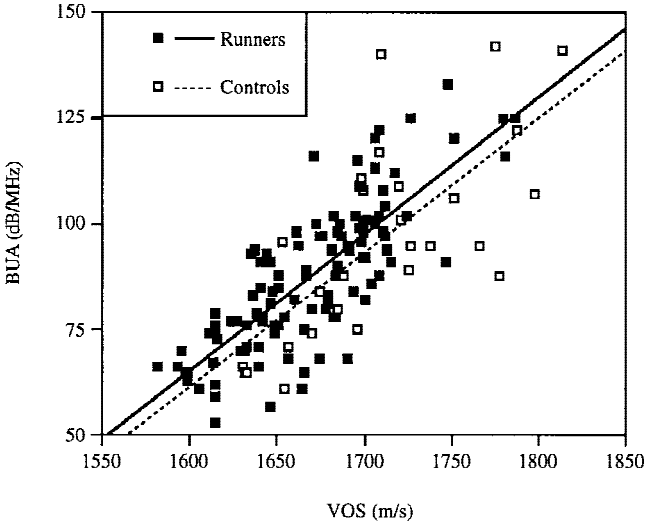

Fig. 4. Scatterplot of the correlation between VOS and BUA in runners and controls.

Our results cannot be generalized to other bones in the lower extremity. It is possible that microdamage might have occurred in the tibia as it is subjected to high bending forces during running [2] and is the most common site for stress fractures in running athletes [25, 26]. In addition, the results cannot be generalized to nonconditioned individuals. The majority of the runners had been training for many years and had competed in a number of previous marathons. Since bone responds to the loads applied, it is likely that the skeleton of the runners had adapted to vigorous exercise and hence could withstand repetitive loading during the marathon without structural damage. Marathon running may select out those individuals with bones strong enough to cope with such loading. It is possible that in a group of sedentary individuals subjected to an equivalent period of intense weight-bearing exercise, there may be bone microdamage.

A number of factors may influence bone US measurements and hence provide possible explanations for the results found in our study. Though it appears unlikely that internal defects such as microfractures occurred during the marathon, transient alterations at the molecular level in both the organic and inorganic phase of bone cannot be excluded. For example, changes in US measurements may arise from proteoglycan reorientation or reorganization of other matrix macromolecules, changes in the mineralization, or crystalline property of bone or slippage at cement lines. Such possibilities have not been investigated.

Other factors that may influence US measurements include soft tissue and microcirculation changes. For example, Bonfield and Tully [27] found that US velocity was temperature dependent with a linear inverse relationship between the two from 21 to 44°C. However, bone temperature might be expected to be increased rather than decreased following the marathon. Bone hydration may also play a role as *in vitro* studies have shown higher velocities in dry compared with wet bone [28]. Similarly, soft tissue edema or dehydration may have occurred after the exercise bout. Since Johansen and Stone [29] reported that the presence of edema caused a mean reduction in both BUA and VOS, this is a less likely explanation for our elevated measurements post-race.

The only independent predictor of the change in BUA between test 1 and test 2 in runners was age, with larger BUA changes associated with increasing age. This relationship was independent of other factors such as years of running training, baseline BUA, race finishing time, or stress fracture history. The mechanism for this relationship is speculative. One possible explanation is that if the increases in BUA are due to acute exercise-related physiological changes, then these may be amplified in older runners. Tissue edema related to disruption of subcutaneous collagen tissue or soft tissue microhemorrhage following the marathon might be greater in older runners with more degenerate connective tissues. Similarly, transient changes in the organic or inorganic phase of bone may be influenced by age. However, it must be noted that the relationship between age and acute BUA changes was weak, accounting for only 7% of the variance.

At baseline, the controls had significantly greater values for VOS and BUA than the runners. Since the groups were not matched for age, this finding is most likely related to the younger age of the controls where the mean age was 12 years less than the runners. As with measurements of bone density, US measurements also show a negative correlation with age [30]. However, differences in US measurements between groups might reflect a detrimental effect of marathon running on bone density. Although athletes generally have higher bone density than less active controls [31], there have been reports of significantly lower bone density in both male and female distance runners [32–34]. Explanations for these findings are unclear but may include sex hormone disturbances, increased cortisol levels, or dietary deficiencies.

Baseline VOS and BUA measurements were moderately to highly correlated. The lack of perfect agreement may reflect the higher error associated with BUA values. Alternatively, it may suggest that the two parameters are measuring different properties of bone. In an *in vitro* study of trabecular bone, Gluer et al. [35] found that all US parameters were significantly associated with bone structure independent of bone mineral density. However, though VOS was largely influenced by trabecular separation, BUA was additionally influenced by trabecular connectivity. Further research is needed to clarify the exact relationship of US parameters and bone properties.

In conclusion, this study found a transient increase in

BUA values and no change in VOS at the calcaneus following a period of repetitive loading *in vivo*. These results do not support the accumulation of lasting structural changes at this skeletal site. Instead, the results are likely to reflect temporary soft tissue or bony changes associated with intense exercise.

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