Bone Mass, External Loads, and Stress Fracture in Female Runners

Susan K. Grimston, Jack R. Engsberg, Reinhard Kloiber, and David A. Hanley

Increased incidence of stress fracture has been reported for amenorrheic runners, while some studies have reported decreased spinal bone mass in amenorrheic runners. Based on results from these studies, one tends to associate decreased spinal bone mass with an increased risk of stress fracture. The present study compared regional bone mass and external loads during running between six female runners reporting a history of stress fracture (seven tibial and three femoral neck) and eight female runners with no history of stress fracture. Dual photon absorptiometry measures indicated significantly greater spinal (L2-L4) and femoral neck bone mineral density in stress fracture subjects \(p<0.05\) but no differences between groups for tibial bone density. Normalized forces recorded from Kistler force plates indicated significantly greater vertical propulsive, maximal medial, lateral, and posterior forces for stress fracture subjects during running \(p<0.05\).

In the past decade the number of women engaging in long-distance running has increased dramatically (Speroff, 1982). With this increase has been a concurrent increase in the number of women developing stress fractures. Results of extensive questionnaire/survey studies have indicated that female runners with athletic amenorrhea (cessation of menses associated with athletic training) demonstrate a higher incidence of stress fracture than their regularly menstruating counterparts (Barrow & Saha, 1986; Clement, Taunton, Smart, & McNicol, 1981; Lloyd et al., 1986). In addition, researchers have repeatedly demonstrated that amenorrheic long-distance runners generally have a lower spinal bone mineral density (BMD) than do regularly menstruating runners and sedentary women (Cook et al., 1987; Drinkwater et al., 1984; Grimston, Sanborn, Miller, & Huffer, 1990a; Grimston, Engsberg, Kloiber, & Hanley, 1990b; Marcus et al., 1985; Nelson et al., 1986).

Despite the fact that bone mass was not measured in the runners responding to questionnaires about stress fracture incidence, the association has been drawn

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between low spinal bone mass and an increased risk for stress fracture (Linnell, Stager, Blue, Oyster, & Robertshaw, 1984; Martin & Bailey, 1987). When investigators have measured spinal bone mass in subjects with medically diagnosed stress fracture, no direct association was found between low BMD (osteopenia) and stress fracture incidence (Milgrom et al., 1989).

However, in a recent study (Myburgh, Hutchins, Fataar, Hough, & Noakes, 1990), 25 athletes with a history of stress fracture were compared to 25 similar athletes without stress fracture. The athletes with stress fracture history were shown to have significantly lower spinal and femoral neck bone density, lower current dietary calcium intake, and current menstrual irregularity. Bone mineral density of bones distal to the proximal femur and external loading kinetics were not reported in their study.

Investigators measuring BMD of regions in the proximal femur in runners have generally found normal to high bone mass, irrespective of the runners’ menstrual status and/or spinal bone mass (Dalen & Olsson, 1974; Drinkwater, Bruemner, & Chestnut, 1990; Grimston et al., 1990a; Grimston et al., 1990b; Nilsson & Westlin, 1971). The influence of repetitive impact loading on the lower extremity bones of runners has been offered as an explanation for these findings, with reference made to the principles underlying Wolff’s law (Wolff, 1892).

The most common site for stress fracture in runners is the tibial diaphysis, with fractures of the metatarsals, femoral neck, and pubic ramus also reported (Clement et al., 1981; Hershman & Mailly, 1990; Markey, 1987; Matheson et al., 1987; McBryde, 1975; Orava, 1980; Orava, Puranen & Ala-Ketole, 1978; Sullivan, Warren, Pavlov, & Kelmar, 1984). Tibial bone density and/or external loading kinetics have not been measured in previous studies of stress fracture in runners to determine whether these differ between runners with and without stress fracture history. The complexity of stress fracture etiology suggests the need for further study.

The relationships among low spinal BMD, stress fracture, and the effects of repetitive mechanical loading in female runners have yet to be established. The magnitude of impact forces and bending moments, and the repetition with which a long-distance runner experiences these loads, are likely of considerable importance in this regard. The purpose of this study was to compare the external loading patterns and regional bone mass measures of female runners with and without a history of stress fracture. Variables reflecting training as well as menstrual and calcium intake history were also considered.

**Methods**

The runners recruited for this investigation were participants in an ongoing investigation into the etiology of osteopenia in female runners. Six runners reporting a history of stress fracture (SF), seven tibial diaphysis and three femoral neck fractures, and eight runners with no history of stress fracture (NSF) volunteered as subjects. Five SF and five NSF subjects were currently menstruating regularly (11–12 menses/yr). One SF and three NSF subjects had secondary amenorrhea (0–3 menses/yr), which could be associated with their running. All were injury free at the time of the investigation and had resumed their regular training distance (average 52 km/wk). All subjects gave written consent to participate in the
study after procedures had been explained to them, and in accordance with the requirements of the University of Calgary Conjoint Medical Ethics Committee.

Regional bone mass was measured at three sites: the lumbar vertebrae (L2–L4), the femoral neck (FN), and the medial diaphysis of the tibia (T), using dual photon absorptiometry (BMC-LAB 22a, Novo Diagnostics Systems). Results were expressed as the total bone mineral density in grams of hydroxyapatite per square centimeter of tissue. The accuracy of measures using this procedure in our laboratory is 2–3%.

Four trials (two for each foot) of force plate data for each subject were collected using a Kistler force plate, collecting at 1,000 Hz and interfaced with a SUN computer. Subjects wore a standard shoe for each trial and were instructed to run at a standard speed of 4.0±0.4 m/sec. The standard shoes were used to prevent any differences in shoe design contributing to differences in loading kinetics. A successful trial was defined by the following three criteria: correct speed, no change in gait pattern in the approach to the force plate, and foot landing fully on the force plate. Force data (i.e., vertical, anterior/posterior, and medial/lateral forces) were normalized by dividing by body weight. Two peak force values in the vertical, and peak forces in the medial, lateral, anterior, and posterior directions were determined from the force time curves.

Subjects responded to questionnaires about training, menstrual, and dietary history. The subsequent derivation of training, menstrual, and calcium indices have been described previously (Grimston et al., 1990a; Grimston et al., 1990b). In brief, the training index (TI) quantified the age that training began relative to the age at menarche; the menstrual index (MI) quantified the average number of menstrual episodes per year for all the years since menarche; and the calcium index (CI) reflected the average daily intake of dietary calcium as a function of the variability in calcium intake, during the period 12–23 years of age. Although there are obvious limitations with retrospective questionnaires, the autobiographical approach used in our protocols have been shown to promote reliable and accurate recall by subjects (Babbie, 1989; Bradburn, Rips, & Shevell, 1987).

Student t tests for independent groups were used to determine any significant differences between variables. Analysis of variance with age as a covariate was used in comparing regional bone mass density, due to the age range of the subjects (16–32 years) and the known effect of age on bone mineral density (Aloia, 1989; Block et al., 1989; Gallagher & Hedlund, 1989; Riggs et al., 1986). All references to significant differences imply a probability at or below p<0.05.

**Results**

With the exception of age, the runners were similar in their physical measures and current running regimes (Table 1). Historical indices indicated that the stress fracture group had begun long-distance running at an age significantly closer to their age at menarche than the non-stress-fracture subjects had, as reflected in the training index (TI). Since the stress fracture group was significantly younger than the non-stress-fracture group (26.9 vs. 32.8 yrs), age was used as a covariate in further statistical analyses.

We conducted a covariate analysis on age since our runners fell within the linear, premenopausal portion of a curve defining the relationship between bone mineral density and age (Block et al., 1989). A covariate analysis between two
variables implies a linear relationship between the two variables (Sokal & Rohlf, 1981). Cross-sectional studies have tested the capacity of linear and various polynomial functions to define the relationship between age and bone density, with variable results (Block et al., 1989; Buchanan, Myers, Lloyd, & Greer, 1988; Gallagher & Hedlund, 1989; Riggs et al., 1986). An extensive study of 538 women tested linear, cubic single-phase, and two-phase linear regressions to define the relationship between age and bone mineral density. Results showed the lowest coefficient of variation for the two-phase regression of bone density on age, using a premenopausal (20–50 yrs) linear segment and a postmenopausal exponential segment (Block et al., 1989).

Covariate analysis on age showed significant differences in spinal and femoral bone mass, with values significantly higher in the stress fracture group (Table 2). There were no significant differences in tibial bone density between groups (p>0.05).

The four amenorrheic runners in the study all had lower bone mass in the spine than all other runners tested (mean BMD L2–L4 = 0.77±0.07 gHA/cm²). In our small cohort, amenorrheic runners did not demonstrate an increased incidence of stress fracture, with only one of four reporting previous fracture compared to five of ten regularly menstruating runners reporting previous fracture. Kinetic analysis of running gait indicated significantly greater vertical impact and propulsive forces, and maximal medial, lateral, and posterior forces in stress fracture subjects (Table 3).

**Discussion**

Investigations of stress fracture incidence in long-distance runners have largely been dominated by two approaches: (a) determination of spinal bone mass in amenorrheic runners and (b) questionnaire surveys of large running populations.
Table 2
Comparison of Mean Values Based on One-Way ANOVA
(mean ± standard error of the mean)

<table>
<thead>
<tr>
<th>Measure</th>
<th>Non stress fracture (n=8)</th>
<th>Stress fracture (n=6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Regional bone mass (gHA/cm²)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lumbar vertebrae (L2–L4)</td>
<td>0.85±0.05</td>
<td>0.92±0.05</td>
</tr>
<tr>
<td>Femoral neck</td>
<td>0.79±0.03</td>
<td>0.85±0.04</td>
</tr>
<tr>
<td>Tibial diaphysis</td>
<td>17.02±0.75</td>
<td>18.67±1.08</td>
</tr>
<tr>
<td>Kinetic analysis (N/body wt)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vertical impact force</td>
<td>1.85±0.05</td>
<td>2.09±0.09*</td>
</tr>
<tr>
<td>Vertical propulsive force</td>
<td>2.51±0.04</td>
<td>2.68±0.03*</td>
</tr>
<tr>
<td>Maximum anterior force</td>
<td>0.50±0.01</td>
<td>0.50±0.01</td>
</tr>
<tr>
<td>Maximum posterior force</td>
<td>0.39±0.01</td>
<td>0.45±0.01*</td>
</tr>
<tr>
<td>Maximum medial force</td>
<td>0.13±0.01</td>
<td>0.19±0.02*</td>
</tr>
<tr>
<td>Maximum lateral force</td>
<td>0.11±0.01</td>
<td>0.18±0.02*</td>
</tr>
</tbody>
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*Significantly higher than non stress fracture; p<0.05.

Table 3
Comparison of Means Using Age as a Covariate
(mean ± standard error of the mean)

<table>
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<th>Measure</th>
<th>Non stress fracture (n=8)</th>
<th>Stress fracture (n=6)</th>
</tr>
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<tr>
<td>Lumbar BMD</td>
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*Significantly higher than non stress fracture; p<0.05.

Based on results from these two methodologically distinct approaches, an association has been drawn between low spinal bone mass (osteopenia) and increased stress fracture incidence.

Some investigators studying bone mass in amenorrheic runners have reported stress fracture incidence (Marcus et al., 1985; Nelson et al., 1986). However, measurement of bone mass in amenorrheic runners reporting stress fractures in these studies has been limited in number and generally restricted to measurements at the lumbar vertebrae. Thus the cohort size from which to base a direct association between osteopenia and stress fracture incidence must be considered small and somewhat nonrepresentative of the broader population experiencing stress fracture, which includes male long-distance runners.
A recent study of bone mineral density and stress fracture acknowledged the fact that stress fracture etiology may be far more complex than previously suggested (Myburgh et al., 1990). These investigators found significantly lower bone density in the spine, femoral neck, Ward Triangle, and trochanter of the proximal femur; however, tibial bone mineral density was not measured. Within the stress fracture group they identified a few individuals with very low BMD (less than 90% of their age-related spine density), while the mean for the stress fracture group was 96% of the age-related spine density.

The apparent paradox in results between the present study and those of Myburgh and associates (1990) suggests that the population of runners with stress fracture may be heterogeneous in terms of spinal bone density. Their stress fracture group was also reported to have significantly lower current calcium intake and a lower oral contraceptive use, both of which may be significant determinants of bone density (Myburgh et al., 1990).

Additional factors reported as being associated with stress fracture incidence include training parameters, for example the quantity, intensity, and duration of training. Our results indicate no significant differences between groups in the total number of years training or in the average weekly training distances. These results concur with the findings of Myburgh and associates, who in fact used a case control study design to eliminate differences in these variables (Myburgh et al., 1990).

There were significant differences between groups for training index (Table I), reflecting an earlier commencement of training with respect to age at menarche for the stress fracture subjects (Table 1). Given that peak cortical bone density occurs at the end of the fourth decade of life (Johnston et al., 1985; Riggs, et al., 1986), it is possible that beginning long-distance running during the second decade of life may place repeated mechanical loads on bones that lack the structural maturity to adapt adequately.

This speculation can be supported by results from exercise studies using young animal models. These studies demonstrated that exercise training has a positive influence on the development of bone quantity but negative effects on the biomechanical (quality) properties of growing bones (Hou, Salem, Zernicke, & Barnard, in press; Woo et al., 1981). The possible relationship between training age and skeletal maturity would merit further study.

The results of the present investigation indicate that stress fracture incidence may be associated with significantly greater spinal and femoral neck BMD. However, there were no differences in tibial bone mass between groups. Our findings conflict with the suggestion that decreased spinal bone mass is associated with an increased risk of stress fracture, in particular the recent findings of Myburgh and associates (1990).

There are several possible explanations for the different results between the present study and those of Myburgh and associates. The most obvious cause for discrepancy is the small subject number in the present investigation. However, it is not clear from the Myburgh study how many of the 16 stress fracture subjects who participated in distance running were female, nor is it clear the locations of the stress fractures for the female runners. Runners in the present study were all female and, with the exception of one subject, all had experienced stress fracture of the tibial diaphysis.

Of all the subjects studied by Myburgh et al. (1990), there were only four
reported tibial stress fractures and no information as to whether these fractures were incurred by the female runners or the other athletes. Lacking this information, it is difficult to draw direct comparisons between the relatively restricted sample in the present investigation to comparable stress fracture athletes studied by Myburgh et al. (1990). Although female distance runners with a history of tibial stress fracture may not be unique in having significantly greater spinal and femoral bone densities than other stress fracture athletes, there are no data to the contrary at this time.

A second explanation for differences between previous findings and those of the present study could be the fact that possible differences in external loading kinetics between stress fracture and non-stress-fracture runners have not previously been evaluated. Runners generating greater external loads might develop significantly greater bone mineral densities than runners generating lower external loads, regardless of stress fracture incidence. This merits further study, particularly to determine whether greater external loading kinetics predispose runners to stress fracture despite normal to high bone mineral densities.

The most significant biomechanical factor associated with tibial stress fracture in humans has been the bending strength of the tibia about the anterior/posterior (AP) axis of bending, with movements occurring in the mediolateral (ML) direction (Milgrom et al., 1989). In this regard, the tibial area moment of inertia (i.e., the distribution of tibial bone area in relationship to an axis of bending) demonstrates a significant relationship to stress fracture incidence. The significantly greater vertical, medial, and lateral forces experienced by our stress fracture subjects would result in a significantly greater bending moment about the AP axis of bending in the tibia (Figure 1). This greater bending moment may be associated with the seven tibial fractures reported in our subjects.

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**Ground Reaction Force**

![Diagrammatic representation of the increased bending moment about the long axis of the tibia in stress fracture subjects due to greater vertical and medial forces during running.](image)
Future studies that include measurements of tibial morphology would allow for testing of the possible relationships suggested by results from this preliminary study. The effect of these greater forces and moments on the structural integrity of the femoral neck, and thus their possible association with the three femoral neck stress fractures reported, cannot be readily assessed from these data.

The greater external loads experienced by our stress fracture subjects, however, may explain the significantly greater spinal and femoral bone mass of these athletes, and thus the differences between our results and those of previous investigators (Myburgh et al., 1990). Bone tissue, particularly trabecular bone, responds to mechanical strain through an increase in bone mass with increased mechanical load (Brewer, Meyer, Keele, Upton, & Hagan, 1983; Dalsky et al., 1986; Lanyon, 1986; Rubin & Lanyon, 1986). The significantly higher measures found for our stress fracture subjects might therefore be predicted based on kinetic analysis of running gait. Determining the cause or causes of these greater external loads would require at least an extensive three-dimensional kinematic analysis of each subject's running gait.

The results of this study illustrate the complexity of the relationship between bone mass and stress fracture incidence in female runners. In our small group of subjects, spinal and femoral bone mineral density were significantly greater in runners with a history of stress fracture. Stress fracture subjects also experienced significantly greater external loads than non-stress-fracture subjects. The question of whether these greater forces are a cause or a consequence of runners having suffered stress fracture cannot be addressed by the results of this investigation.

References


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