Risk factors for stress fracture in female endurance athletes: a cross-sectional study

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ABSTRACT
Objective: To identify psychological and physiological correlates of stress fracture in female endurance athletes.
Design: A cross-sectional design was used with a history of stress fractures and potential risk factors assessed at one visit.
Methods: Female-endurance athletes (58 runners and 12 triathletes) aged 26.0±7.4 years completed questionnaires on stress fracture history, menstrual history, athletic training, eating psychopathology and exercise cognitions. Bone mineral density, body fat content and lower leg lean tissue mass (LLLTM) were assessed using dual-x-ray absorptiometry. Variables were compared between athletes with a history of stress fracture (SF) and those without (controls; C) using χ², analysis of variance and Mann-Whitney U tests.
Results: Nineteen (27%) athletes had previously been clinically diagnosed with SFs. The prevalence of current a/oligomenorrhea and past amenorrhoea was higher in SF than C (p=0.008 and p=0.035, respectively). SF recorded higher global scores on the eating disorder examination questionnaire (p=0.049) and compulsive exercise test (p=0.006) and had higher LLLTM (p=0.029) compared to C. These findings persisted with weight and height as covariates. In multivariate logistic regression, compulsive exercise, amenorrhoea and LLLTM were significant independent predictors of SF history (p=0.006, 0.009 and 0.035, respectively).
Conclusions: Eating psychopathology was associated with increased risk of SF in endurance athletes, but this may be mediated by menstrual dysfunction and compulsive exercise. Compulsive exercise, as well as amenorrhoea, is independently related to SF risk.

INTRODUCTION
Stress fractures (SFs) occur when microdamage caused by repetitive mechanical load exceeds the biological capacity of the bone.1 SFs are the most common overuse injury in athletes, interrupting training and prematurely ending sporting careers.2 Over 40% of athletes report a history of SF according to some previous research, although prevalence differs markedly between studies.3–5 Risk factors for SF have been studied in military recruits commencing physically demanding physical training programmes. The risk factors identified have included irregular menses; low bone mineral density (BMD); low lean mass, lower muscle size and strength; poor skeletal alignment (leg length discrepancy); narrow tibial cross-sectional area and low physical fitness compared to those who did not SF.6–9 While these studies have improved our understanding of SF risk,9,10 there is a need to examine risk factors for SFs in female endurance athletes. In this study, we examined associations between eating psychopathology, exercise psychopathology, and SF history in female endurance athletes.
understanding of the aetiology of SF, these risk factors may not translate well to athletes, who are accustomed to training and have high levels of fitness. In female athletes, previously identified risk factors for SF include small calf circumference, late age at menarche, menstrual dysfunction (both past and current), low bone density, current disordered eating and abnormal gait. However, findings have often been inconsistent (e.g., low bone density is associated with increased risk in some studies but not others).

Female endurance athletes have particularly high prevalence of the female athlete triad, which involves menstrual dysfunction, low BMD and dietary restriction/energy deprivation. All these may be risk factors for SFs, but previous studies on their role in athletic populations have had small sample sizes and yielded conflicting findings. Increased awareness of the female athlete triad in recent decades may have affected the management of female athletes, perhaps to the extent that the associations reported in older studies may no longer persist. The current high prevalence (1–62%) of disordered eating among female athletes could further increase SF risk, as restrictive eating patterns were associated with increased risk in some of the previous studies. Increased awareness of the female athlete triad in recent decades may have affected the management of female athletes, perhaps to the extent that the associations reported in older studies may no longer persist. The current high prevalence (1–62%) of disordered eating among female athletes could further increase SF risk, as restrictive eating patterns were associated with increased risk in some of the previous studies.

### Study design

A cross-sectional design was used with a history of SFs and potential risk factors assessed at one visit. Questionnaires were completed to assess SF history, eating psychopathology, dietary intakes, menstrual dysfunction, contraceptive use and training history. A 4-day diet diary was provided for subsequent completion and return. BMD, bone mineral content (BMC), geometric properties and body composition were measured using dual-energy x-ray absorptiometry (DXA) and anthropometric measures were taken using standard techniques. The study received ethical approval from the National Research Ethics Service and Loughborough University Ethics Committee. All athletes gave written informed consent.

### Participants

Seventy female endurance athletes aged 18–45 years were recruited through the English Institute of Sport, UK athletics, British triathlon and UK-registered running and triathlon clubs, via letters, posters, group meetings and word-of-mouth. Athletes were required to be competing at international, national or county level and training approximately 8–10 (runners) or 15–20 h/week (triathletes) in events from 800 m to the marathon or triathlon. Athletes were excluded from the study if they had not been trained in the last 6 months and if they were currently pregnant or lactating or had been in the last 12 months.

### Injury history

History of SF was assessed by a questionnaire. These were defined as a fracture/reaction clinically diagnosed by a sports physician and confirmed with a positive diagnosis on x-ray, CT or MRI. For each SF, athletes recorded the age when the SF occurred, the anatomical location, time of the year and the method of diagnosis.

### Menstrual function

Participants were classified as secondary amenorrhoeic (less than 4 menses per year) oligomenorrhoeic (4–9 periods/year; periods occurring at intervals greater than 35 days after onset of menses) or eumenorrhoeic (10 or more periods per year) based on their menstrual function in the last 12 months. For analysis purposes, athletes who were classified with menstrual dysfunction were combined into one group (a/oligomenorrhoea, 0–9 periods/year) and compared with the eumenorrhoeic athletes. Age at menarche, number of periods in the preceding 12 months, duration of a/oligomenorrhoea since menarche and current use of hormonal contraception (HC) were assessed by a questionnaire.

### Eating psychopathology and compulsive exercise cognitions

Athletes completed the Eating Disorder Examination Questionnaire (EDE-Q) and the Compulsive Exercise Test (CET). The EDE-Q is a self-reported version of the EDE, the current gold standard for measuring eating-disordered psychopathology. The EDE-Q is a 36-item questionnaire comprising four subscales (restraint, eating concern, shape concern and weight concern), a global score and a specific section of diagnostic questions related to bingeing/purging. Each item on the questionnaire is scored from 0 to 6 with high scores representative of concerns with eating behaviours and attitudes. The CET is a 24-item self-reported questionnaire scored from 0 to 5 with high scores representative of compulsive exercise traits. It is designed to assess four domains of compulsive exercise (compulsivity, affect regulation, prediction and potential risk factors assessed at one visit. Questionnaires were completed to assess SF history, eating psychopathology, dietary intakes, menstrual dysfunction, contraceptive use and training history. A 4-day diet diary was provided for subsequent completion and return. BMD, bone mineral content (BMC), geometric properties and body composition were measured using dual-energy x-ray absorptiometry (DXA) and anthropometric measures were taken using standard techniques. The study received ethical approval from the National Research Ethics Service and Loughborough University Ethics Committee. All athletes gave written informed consent.

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weight-driven and shape-driven exercise and behavioural rigidity.\textsuperscript{17} The CET has not previously been used in competitive athletes but has been validated in a comparable group of young female exercisers.\textsuperscript{17}

**Dietary behaviours**
Participants completed a food frequency questionnaire (European Prospective Investigation into Cancer; EPIC FFQ)\textsuperscript{28,29} and a 4-day diet diary. The EPIC FFQ provides general information of the number of portions consumed of a wide range of foods over the past 12 months. The 4-day food diary was completed by the participants over 3 week days and 1 weekend. The FFQ and the 4-day diet diary were coded and entered into a computerised software package (CompEat Pro, V.5.8) to determine energy, macronutrient and micronutrient intakes.

**Bone and body composition**
DXA was used to measure BMD and BMC of the total body, lumbar spine, femoral neck and radius (Lunar Prodigy, GE Healthcare, Madison, Wisconsin, USA v.12.2). Bone geometry at the femoral neck was estimated using Lunar Advanced Hip Structural Analysis (AHA) algorithms to determine the cross-sectional moment of inertia (CSMI), cross-sectional area (CSA), minimal femoral neck width and section modulus (Z). All scans were conducted on the dominant side as this is reported to be the most prevalent side for injury.\textsuperscript{13}

**Anthropometric measures**
Height and body mass were measured prior to the DXA measurements using standard protocols, via a stadiometer (Holtain Ltd, Pembrokeshire) and beam balance scale (Herbert and Sons Ltd, London), respectively. Body composition (mass of fat, lean and bone) was assessed from a total body DXA scan. The mass of bone-free lean tissue in the lower leg (LLLTM; defined as the region inferior to the knee joint space) was also determined from the DXA scan, as a measure related to calf muscle mass. Athletes were blinded to the body composition results until the end of the study to prevent any possible bias when answering questionnaires related to eating psychopathology. Circumferences of the dominant thigh and calf were measured with a tape measure held horizontally at the midpoint between the inguinal crease and the patella for the thigh, and midpoint between the proximal tibia border and the medial malleolus for the calf.\textsuperscript{30}

**Statistical analysis**
All statistical analyses were performed using SPSS V.18.0 (SPSS, Chicago, Illinois, USA). The Kolmogorov-Smirnov test was used to assess normal distribution. Athletes were classified into two groups: those with a history of SF and a control (C) group. Potential risk factors were compared between groups using analysis of variance (ANOVA) and Mann-Whitney U tests. $\chi^2$ Tests were used to determine whether frequencies of categorical variables differed between SF and C groups. Discriminators of SF identified as statistically significant in univariate analyses along with theory-driven variables weight (as a measure for body size) and training duration were entered into a logistic regression to determine their independent contribution to SF/reaction injury. The first model (model A) examined the contribution of a/oligomenorrhea. Subsequent models (models B, C and D) examined the contributions of CET and EDE-Q independent of a/oligomenorrhea and/or each other. The final model (model E) includes all potential discriminators of SF to determine which ones are independent of each other. For this analysis, eating psychopathology and compulsive exercise variables were recoded into two subgroups using a median split. Statistical significance was considered at the 5% probability level ($p<0.05$).

**RESULTS**
Seventy female athletes (58 runners and 12 triathletes) were recruited, of whom 36% were at of national or international standard. A high prevalence of menstrual dysfunction was evident, with 42.9% of athletes categorised as a/oligomenorrhoeic ($\leq$9 periods/year) and 57.1% eumenorrhoeic ($

The SF group scored higher on the global scores for past amenorrhoea and past amenorrhoea were significantly higher in the SF than in the C group ($\chi^2$ test $p=0.008$ and 0.035, respectively; table 1) although age at menarche did not differ significantly between groups. Dominant LLLTM was significantly higher in the SF than in the C group ($p=0.029$); this finding persisted after adjustment for weight (kg) and training duration (hours/week) ($p=0.035$). Calf circumference measurements, bone measurements, energy, macronutrient intakes and calcium intakes did not differ between groups (table 1).

The SF group scored higher on the global scores for both the EDE-Q and CET Global score ($p=0.049$, $p=0.006$ respectively, table 2) and had a higher behaviour rigidity score on the CET ($p=0.038$) than controls (table 2). There were no significant differences in global scores for either, EDE-Q ($p=0.216$) and CET ($p=0.216$) between the athletes at the national/international level and subelite level.

The variables that differed according to SF in the univariate analysis, (current a/oligomenorrhoea, LLLTM,
EDE-Q scores and CET scores) were entered into a multiple logistic regression adjusted for weight (kg) and training duration (hours/week). Amenorrhea was associated with 4.7-fold greater odds of SF relative to eumenorrhea (p=0.009, table 3, model A). This finding persisted when current HC was entered into the regression model (p=0.012, B=1.506) and when CET, EDE-Q and/or LLLTM were added to the model (table 3, models B–E). CET score and LLLTM, but not EDE-Q scores contributed to SF risk independent of amenorrhea. Athletes with CET scores above the median had 7.1-fold greater odds of past SF than those below the median (p=0.006, B=1.956; table 3 models B and D). Each additional SD of LLLTM was associated with 2.3-fold greater odds of fracture (p=0.035, B=0.843). Findings for all models were similar when not adjusted for weight and training duration.

**DISCUSSION**

Eating psychopathology, compulsive exercise, menstrual dysfunction and LLLTM were associated with increased SF risk in endurance athletes. The association of eating psychopathology was not independent of menstrual function, suggesting that the influence of disordered eating on SF is mediated through amenorrhea. Compulsion to exercise and LLLTM, as well as

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**Table 1  Characteristics of athletes according to stress fracture (SF) history: mean (SD)**

| Characteristics                              | SF group (n=19) | Control (C) group (n=51) |
|----------------------------------------------|----------------|-------------------------|
| Age (years)                                  | 25.6 (6.4)     | 26.1 (7.8)              |
| Height (m)                                   | 1.67 (0.05)    | 1.67 (0.05)             |
| Weight (kg)                                  | 56.2 (4.8)     | 54.9 (6.2)              |
| Body fat (%)                                 | 16.6 (4.7)     | 17.4 (6.4)              |
| Lower leg lean tissue mass (kg)              | 2.61 (0.03)    | 2.46 (0.02)             |
| Calf circumference (cm)                      | 32.0 (2.3)     | 31.2 (1.80)             |
| Time since stress fracture (years; median (IQR)) | 2.0 (3.0)     | –                       |
| Training                                     |                |                         |
| Time since commencing training (years)       | 7.4 (4.3)      | 7.8 (6.4)               |
| Age at commencing competition (years)        | 18.3 (7.0)     | 18.4 (8.5)              |
| Weekly training duration (hours/week)        | 14.2 (4.3)     | 12.1 (4.3)              |
| Bone mineral density (g/cm²)                 |                |                         |
| Total body                                   | 1.160 (0.070)  | 1.151 (0.064)           |
| Femoral neck                                 | 1.068 (0.122)  | 1.089 (0.107)           |
| Lumbar spine                                 | 1.144 (0.157)  | 1.119 (0.099)           |
| Bone mineral content (g)                     |                |                         |
| Total body                                   | 2482 (305)     | 2425 (298)              |
| Femoral neck                                 | 4.9 (0.4)      | 5.1 (0.7)               |
| Lumbar spine                                 | 60.5 (12.2)    | 60.2 (9.9)              |
| Bone geometry                                |                |                         |
| CSA (mm²)                                     | 154 (17)       | 157 (21)                |
| Minimal femoral neck width (mm)              | 28.9 (2.6)     | 28.4 (2.1)              |
| Section modulus (mm³)                        | 677 (117)      | 674 (121)               |
| Menstrual history                            |                |                         |
| Age at Menarche (years)                      | 13.9 (1.7)     | 14.1 (2.1)              |
| History of amenorrhea†                       | 15 (78.9%)     | 26 (51.0%)              |
| Current a/oligomenorrhea†                    | 13 (68.4%)     | 17 (33.3%)              |
| Current eumenorrhea†                         | 6 (31.6%)      | 34 (66.7%)              |
| Hormonal contraception                       |                |                         |
| Current hormonal contraception users†        | 4 (21.1%)      | 17 (33.3%)              |
| Current hormonal contraception use in a/oligomenorrhea (HCA)† | 4 (21.1%) | 3 (64.7%) |
| Four-day diet analysis                       | SF (n=17)      | C (n=44)                |
| Energy intake (kcal)                         | 1862 (688)     | 1875 (365)              |
| CHO (%)                                      | 55.2 (5.3)     | 56.6 (5.3)              |
| Protein (%)                                  | 14.8 (2.9)     | 16.2 (3.3)              |
| Fat (%)                                      | 30.0 (4.9)     | 27.2 (5.3)              |
| Calcium (mg)                                 | 887 (377)      | 944 (277)               |

*Significant difference between stress fracture and control groups: p<0.05.
†Reported as the number of people and percentage.
menstrual dysfunction, were independently related to SF risk and may play a role in SF aetiology.

In this study, 27% of female athletes had a history of SF. This is similar to the prevalence of 31% in a recent study in female cross-country runners, but substantially lower than the 50% in a classic study in endurance athletes. Differences in findings could be explained by factors such as: diagnostic techniques, athlete age, training intensity and volume and potential recruitment bias. It is also possible that findings from earlier studies, and increased awareness of female athlete triad, have led to improvements in athlete management contributing to lower prevalence of SF in more recent studies. In our study, as in a recent study in distance runners, the most common site was the metatarsals, while in earlier studies more SFs occurred at the tibia in distance runners and track and field athletes, although metatarsal and tarsal SFs were more common in sprinters and jumpers. Metatarsal SF prevalence is associated with changes in training surface, training volume, footwear and running biomechanics, so these factors may contribute to differences in SF reaction locations.

Current menstrual dysfunction was prevalent in this sample of athletes, with 43% of athletes reporting current amenorrhea or oligomenorrhea. Both current and past menstrual dysfunction were significantly more frequent in athletes with a history of SF, consistent with earlier findings. Furthermore, current menstrual dysfunction was an independent predictor of SF history, with a/oligomenorrheic athletes having 4.3 times greater odds of SF compared with those who were currently eumenorrheic. This is consistent with the 6-fold greater odds of SF in athletes with menstrual dysfunction reported by Bennell et al. The endurance

### Table 2 Eating disorder examination and compulsive exercise test questionnaire responses according to stress fracture history: median (IQR)

| Characteristics            | Stress fracture group (N=19) | Control (C) group (N=51) |
|----------------------------|-----------------------------|--------------------------|
| EDE-Q                      |                             |                          |
| Restraint                  | 1.6 (0.6–3.2)               | 1.0 (0.4–1.8)            |
| Eating concerns            | 0.6 (0.2–1.4)               | 0.2 (0.2–0.6)            |
| Shape concerns             | 1.9 (0.9–3.6)               | 1.3 (0.5–2.0)            |
| Weight concerns            | 1.2 (0.6–3.0)               | 1.0 (0.4–1.8)            |
| Global scores              | 1.7 (0.6–2.7)               | 0.9 (0.5–1.6)*           |
| CET                        |                             |                          |
| Compulsive exercise        | 3.4 (2.6–3.9)               | 2.8 (1.9–3.5)            |
| Shape and weight           | 2.4 (1.8–3.4)               | 2.0 (1.6–2.6)            |
| Mood regulation            | 4.0 (3.4–5.0)               | 3.8 (3.0–4.4)            |
| Lack of exercise enjoyment | 0.7 (0.0–1.3)               | 0.3 (0.0–1.0)            |
| Behaviour rigidity         | 3.7 (3.0–4.0)               | 3.0 (2.7–3.7)*           |
| Global scores              | 2.9 (2.4–3.1)               | 2.4 (2.1–2.8)*           |

*Significant difference between stress fracture and control groups: Mann Whitney-U test \( p<0.05 \)

EDE-Q, Eating Disorder Examination Questionnaire; CET, Compulsive Exercise Test.

### Table 3 Multivariate logistic regression models of stress fracture history; odds ratio (95% CI)

| Model A | Model B | Model C | Model D | Model E |
|---------|---------|---------|---------|---------|
| Constant | −3.9 | −3.2 | −3.2 | −3.2 |
| Cox & Snell \( R^2 \) | 0.137 | 0.245 | 0.149 | 0.248 |
| Variables | | | | |
| Current/oligomenorrhea† | 4.7 (1.5 to 15.0)* | 6.6 (1.9 to 38.2)* |
| Global CET (median to <median) | 7.1 (1.9 to 27.1)** |
| Global EDE-Q (median to <median) | 4.1 (1.4 to 14.6)* |
| Lower leg lean tissue mass (SD) | 0.7 (0.2 to 2.8) |

Logistic regression with stress fracture history as the dependent variable. \( p<0.05 \)

†Eumenorrhea as the reference group.

CET, Compulsive Exercise Test. The global CET and EDEQ variables represent a comparison with a median split. Models are adjusted for age (years), height (cm), weight (kg), and training duration (hours/week).
athletes had an average menarcheal age of 14 years, compared with 12.3 years in a broader population indicating later menarche occurs in endurance athletes, as has previously been reported in gymnasts. Age at menarche was not associated with an increased risk of SF, although associations have been reported in some 12-15 although not all 20 previous studies. As the results did not identify significant differences in either age at menarche or current BMI, it seems most likely that secondary amenorrhea rather than primary amenorrhea or delayed menarche was associated with SF.

Interestingly, although athletes with SF had a higher prevalence of amenorrhea, they had a lower prevalence of hormonal contraceptive use, although this difference did not reach statistical significance. It is possible that amenorrhea not countered by HC use is associated with greater SF risk; furthermore, amenorrheic athletes are possibly less likely to comply with HC use in the fear of gaining weight. 40 Alternatively it is possible that having a history of SF influences HC-prescribing strategies. Given the increased awareness of the female athlete triad, clinical physicians presented with an amenorrheic athlete with a SF may attempt to determine the cause of, and resolve, the amenorrhea rather than prescribing HC, although past research has shown a trend towards a reduction in SF when HC was prescribed. 11-13 20 40 Further studies on the role of HC use in SF are needed.

Athletes with a history of SF scored higher on the EDE-Q than athletes with no SF history, thus demonstrating a potential relationship with eating psychopathology. This finding supports earlier work which used a less robust measure of eating psychopathology. 9 However, unlike previous studies 9 our findings did not show eating psychopathology to be a discriminator of SF history independent of a/oligomenorrhea. The relationship between eating psychopathology and SF may therefore be mediated through menstrual dysfunction. Future intervention studies would be necessary to confirm whether treatment of disordered eating can reduce SF risk.

A novel finding from the present study is that athletes with high (above median) CET scores had moderately (7.3 times) greater odds of SF, perhaps indicating a greater tendency to train through discomfort. Compulsive exercise predicted SF independent of a/oligomenorrhea and eating psychopathology. It is therefore important to examine compulsive exercise, as well as menstrual status, when identifying athletes at risk of SF.

Lower calf circumference has been reported to increase the risk of SF in track and field athletes independent of all other factors 13 and this finding is assumed to be mediated by the lower leg muscle mass. In this study, calf circumference did not differ according to SF history, but LLLTM measured by DXA was positively related with SF, with athletes who had a greater lower leg lean and running more being more likely to have had an SF, independent of other risk factors. Due to the retrospective nature of this study, however, it is not possible to determine if greater LLLTM is a cause or effect of SF history. Future studies should further examine the potential role of lower leg musclearity in SF aetiology using more robust measures than calf circumference, for instance DXA or MRI.

Factors not significantly related to SF in this study included dietary intakes, BMD, bone geometry and calf circumference. Low dietary calcium intake was a significant predictor of SF in some, 20 but not all 15 previous research. Findings may differ depending on measurement tools and differences in dietary intakes, although the mean calcium intake was above the recommended values in all these studies. Similarly, low BMD was associated with SF risk in some 15 13 20 but not all previous studies. 2

Strengths of this study include the assessment of CET. Our findings that compulsive attitudes towards exercise and eating psychopathology are associated with SF risk highlight that those treating and coaching endurance athletes should consider attitudes to exercise and eating behaviours when assessing SF risk. Future research should examine whether interventions that address these features can reduce SF risk. The major limitation of this study is imposed by the experimental design. Due to the cross-sectional nature of this study, it is not possible to determine whether risk factors are a cause or effect of the SF as they have been measured after the outcome occurred. Also, as the study was not prospective we could not determine relative risks. It should be noted that as the prevalence of SF is relatively high, ORs may be substantially greater than relative risks. Further limitations include potential recruitment bias, self-reporting of the history of SF and the ability to recall data. However, the occurrence of a SF during an athlete’s career is usually an event of such significance that it is unlikely to be misreported. Athletes were measured at variable intervals following SF during which time their behaviours, and other risk factors, may have changed. Differences in SF site and prevalence between recent and classic studies suggest that changes in athlete management have occurred (perhaps following findings from earlier studies), so there is a need to conduct further prospective studies in contemporary female endurance athletes.

In conclusion, disordered eating was associated with an increased risk of SF, but this may be mediated through menstrual dysfunction. Compulsive exercise cognitions, as well as amenorrhea, were independently associated with SF risk and may have a role in SF aetiology.
REFERENCES

1. Schaffler MB, Radin EL, Burr DB. Long term fatigue behavior of compact bone at low strain magnitude and rate. Bone 1990;11:321–8.

2. Bennell KL, Malcolm SA, Thomas SA, et al. Risk-factors for stress-fractions in female track and field athletes—a retrospective analysis. Clin J Sport Med 1995;5:229–35.

3. Bennell KL, Malcolm SA, Thomas SA, et al. The incidence and distribution of stress fractures in competitive track and field athletes—a twelve-month prospective study. Am J Sports Med 1996;24:211–17.

4. Nativ A, Puffer JC, Casper J, et al. Stress fracture risk factors, incidence and distribution: a 3 year prospective study in collegiate runners. Med Sci Sports Exerc 2000;32(5 Suppl):S347.

5. Snyder RA, Koester MC, Dunn WR. Epidemiology of stress fractures. Clin Sports Med 2005;24:37–52.

6. Milgrom C, Giladi M, Simkin A. Analysis of the biomechanical mechanisms of tibial stress fractures among Israeli infantry recruits. J Bone Joint Surg Am 2000;82A:1255–64.

7. Beck TJ, Ruff CB, Shaffer RA, et al. Stress fracture in military recruits: gender differences in muscle and bone susceptibility factors. Bone 2000;27:437–44.

8. Beck T. Measuring the structural strength of bone with dual-energy x-ray absorptiometry: principals, technical limitations, and future possibilities. Osteoporos Int 2003;14(Suppl 5):S81–8.

9. Giladi M, Milgrom C, Simkin A, et al. Stress fractures—identifiable risk factors. Am J Sports Med 1991;19:647–52.

10. Carbon R, Sambrook PN, Deakin V, et al. Bone-density of elite female athletes with stress-fractures. Med J Australia 1990;153:373–6.

11. Myburgh KH, Hutchins J, Fataar AB, et al. Bone-density of elite female athletes with stress-fractures. Med J Australia 1990;153:373–6.

12. Myburgh KH, Matheson GO, Clement DB, McKenzie DC, et al. Stress fractures in track and field athletics. J Sci Med Sport 2001;4:186–93.

13. Bennell KL, Malcolm SA, Thomas SA, et al. Risk factors for stress fractures in track and field athletes—a twelve-month prospective study. Am J Sports Med 1996;24:211–17.

14. Nativ A, Puffer JC, Casper J, et al. Stress fracture risk factors, incidence and distribution: a 3 year prospective study in collegiate runners. Med Sci Sports Exerc 2000;32(5 Suppl):S347.

15. Snyder RA, Koester MC, Dunn WR. Epidemiology of stress fractures. Clin Sports Med 2005;24:37–52.

16. Milgrom C, Giladi M, Simkin A. Analysis of the biomechanical mechanisms of tibial stress fractures among Israeli infantry recruits. J Bone Joint Surg Am 2000;82A:1255–64.

17. Beck TJ, Ruff CB, Shaffer RA, et al. Stress fracture in military recruits: gender differences in muscle and bone susceptibility factors. Bone 2000;27:437–44.

18. Beck T. Measuring the structural strength of bone with dual-energy x-ray absorptiometry: principals, technical limitations, and future possibilities. Osteoporos Int 2003;14(Suppl 5):S81–8.

19. Milgrom C, Simkin A, et al. Stress fractures—identifiable risk factors. Am J Sports Med 1991;19:647–52.

20. Carbon R, Sambrook PN, Deakin V, et al. Bone-density of elite female athletes with stress-fractures. Med J Australia 1990;153:373–6.

21. Bennell KL, Malcolm SA, Thomas SA, et al. Risk factors for stress fractures in female track and field athletes—a retrospective analysis. Clin J Sport Med 1995;5:229–35.

22. Davis C, Kennedy SH, Ravelski E, et al. The role of physical activity in the development and maintenance of eating disorders. Psychol Med 1994;24:957–67.

23. Snow-Harter GM. Bone health and prevention of osteoporosis in active and athletic women. Clin J Sports Med 1994;13:389–404.

24. Nattiv A, Lewis B, et al. Low bone mineral density at axial and appendicular sites in amenorrheic athletes. Med Sci Sports Exerc 1993;25:1197–202.

25. Barrow GW, Saha S. Menstrual irregularity and stress fractures in collegiate female distance runners. Am J Sports Med 1988;16:209–16.

26. Fairburn CG, Beglin SJ. Assessment of eating disorders—interview of self-reporting questionnaire. Int J Eat Disord 1994;16:363–70.

27. Welsh AA. The CAFE computer program for nutritional analysis of the EPIC-Norfolk food frequency questionnaire and identification of extreme nutrient values. J Hum Nutr Diet 2005;18:99–116.

28. Chaytor K, Cuthbertson K, Bouchard C, et al. Circumferences. Anthropometric standardization reference manual. Champaign, IL: Human Kinetics, 1988: 39–54.

29. Arendt E, Agel J, Heikes C, et al. Stress injuries to bone in college athletes—a retrospective review of experience at a single institution. Am J Sports Med 2003;31:959–68.

30. Benazzo F, Barnabei G, Ferrario A, et al. Stress fractures in track and field athletics. J Sports Traumatol 1992;14:51–65.

31. Bennell KL, Brukner PD. Epidemiology and site specificity of stress fractures. Clin Sports Med 1997;16:179–95.

32. Matheson GO, Clement DB, McKenzie DC, et al. Stress fractures in athletes—a study of 320 cases. Am J Sports Med 1987;15:46–58.

33. Crossley K, Bennell KL, Wrigley T, et al. Ground reaction forces, bone characteristics, and tibial stress fracture in male runners. Med Sci Sports Exerc 1999;31:1088–93.

34. Grimston SK, Zernicke RF. Exercise-related stress responses in bone. J Appl Biomech 1993;9:2–14.

35. Warren MP. Amenorrhea in endurance runners. J Clin Endocrinol Metab 1992;75:1393–7.

36. Malina R, Bouhoutsos C. Characteristics of young athletes. Growth, maturation, and physical activity. Champaign, IL: Human Kinetics, 1991: 443–63.

37. Carbon RJ. Exercise, amenorrhea and the skeleton. Brit Med Bull 1992:48:546–60.

38. Cobb KL, Bachrach LK, Sowers M, et al. The effect of oral contraceptives on bone mass and stress fractures in female runners. Med Sci Sports Exerc 2007;39:1464–73.