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# Risk factors for stress fracture in female endurance athletes: a retrospective cohort study

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## Article Summary

### Article Focus

1. To identify the factors related to stress fracture in female endurance athletes,

2. To evaluate associations of eating and exercise behaviours with stress fracture and to determine whether associations are independent of previously recognised risk factors.

### Key Points

1. Compulsive exercise and lower leg lean tissue mass, as well as amenorrhoea, are independently related to stress fracture risk

2. Eating disorder psychopathology was associated with increased risk of stress fracture in endurance athletes, but this may be mediated by menstrual dysfunction and compulsive exercise.

### Strengths and Limitations

1. Strength; the assessment of the Compulsive Exercise test (CET) and a more robust measure of eating psychopathology determined that compulsive exercise and eating psychopathology are associated with stress fracture risk.

2. Limitation, potential recruitment bias, self-reporting of history of stress fracture and the ability to recall data.

3. Limitation; measurements at variable intervals following stress fracture during which time athlete behaviours, and other risk factors, may have changed.

**Abstract**

**Objective:** To identify psychological and physiological correlates of stress fracture in female endurance athletes.

**Design:** A retrospective cohort design was used with 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 chi-squared, ANOVA and Mann Whitney U tests.

**Results:** Nineteen (27%) athletes had previously been clinically diagnosed with stress fractures. The prevalence of current a/oligomenorrhoea 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 stress fracture history (p=0.009, 0.005 and 0.37 respectively).

**Conclusion:** Eating psychopathology was associated with increased risk of stress fracture in endurance athletes, but this may be mediated by menstrual dysfunction and compulsive exercise. Compulsive exercise, as well as amenorrhoea, are independently related to stress fracture risk.

## Introduction

Stress fractures occur when microdamage caused by repetitive mechanical load exceeds the biological capacity of the bone (1). Stress fractures are the most common overuse injury in athletes, interrupting training and prematurely ending sporting careers (2). Over 40% of athletes report a history of stress fracture according to some previous research, although prevalence differs markedly between studies (3-5).

Risk factors for stress fracture 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 stress fracture (6-9). Whilst these studies have improved our understanding of the aetiology of stress fracture, these risk factors may not translate well to athletes, who are accustomed to training and have high levels of fitness (2). In female athletes, previously identified risk factors for stress fracture include small calf circumference, late age at menarche, menstrual dysfunction (both past and current), low bone density, current disordered eating, and abnormal gait (2, 5, 10-15). However, findings have often been inconsistent (e.g. low bone density is associated with increased risk in some studies but not others) (5).

Female endurance athletes have particularly high prevalence of the female athlete triad, which involves menstrual dysfunction, low bone mineral density and dietary restriction/energy deprivation (16). All of these may be risk factors for stress fractures (12, 13, 15) but previous studies on their role in athletic populations have had small sample sizes and yielded conflicting findings (5, 17). Increased awareness of the female athlete triad in recent decades may have affected management of the female athlete, perhaps to the extent that the associations reported in older studies may no longer persist. The current high prevalence (1-

62%) (18) of disordered eating among female athletes could further increase stress fracture risk, as restrictive eating patterns were associated with increased risk in some of the previous studies (2, 19, 20).. Furthermore, restrictive eating patterns may be accompanied by compulsive exercise behaviours (21, 22), but the role of compulsive exercise has not been examined. Eating behaviours may have an independent effect on stress fracture risk, or this may be mediated through related exercise behaviours, menstrual dysfunction or low bone density. It is important to determine whether eating behaviours have an independent effect as this would demonstrate that resolving menstrual dysfunction and low bone density would not entirely resolve the increased risk of stress fracture.

There is thus a need for further research on the role of eating and exercise behaviours in stress fracture risk, using multivariate models to determine whether risk factors act independently. Therefore, this study aimed to identify the factors related to stress fracture in female endurance athletes, to evaluate associations of eating and exercise behaviours with stress fracture and to determine whether associations are independent of previously recognised risk factors.

## Methods

### Study design

A retrospective cohort design was used with history of stress fractures and potential risk factors assessed at one visit. Questionnaires were completed to assess stress fracture history, eating psychopathology, dietary intakes, menstrual dysfunction, contraceptive use and training history. A four day diet diary was provided for subsequent completion and return. Bone mineral density (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 (NRES) 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 either be competing at international, national, or county level or training approximately 8-10 hours per week (runners) or 15-20 hours per week (triathletes) in events from 800m to the marathon or triathlon. Athletes were excluded from the study if they had not currently trained in the last six months and if they were currently pregnant or lactating or had been in the last 12-months.

**Measurements**

**Injury history**

History of stress fractures/stress reactions (repetitive stress injuries on bone) was assessed by 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 stress fracture/reaction, athletes recorded the age when the stress fracture/reaction occurred, the anatomical location, time of year, and method of diagnosis.

**Menstrual function**

Participants were classified as amenorrheic (0-3 periods per year) oligomenorrhoeic (4-9 periods per year) or eumenorrhoeic ($\geq$ 10 periods per year) based on their menstrual function in the last 12-months (23). Age at menarche, number of periods in the preceding 12 months, duration of a/oligomenorrhoea since menarche, and current use of hormonal contraception (HC) was assessed by questionnaire.

**Eating psychopathology and compulsive exercise cognitions**

Athletes completed the Eating Disorder Examination Questionnaire (EDE-Q) (23) and the Compulsive Exercise Test (CET) (17). The EDE-Q is a self reported version of the Eating Disorder Examination (EDE), the current gold standard for measuring eating disordered psychopathology (23). The EDE-Q is a 36-item questionnaire comprising four subscales (restraint, eating concern, shape concern, weight concern), a global score and a specific section of diagnostic questions related to bingeing/purging. Each item on the questionnaire is scored from 0-6 with high scores representative of concerns with eating behaviours and attitudes. The CET is a 24-item self reported questionnaire scored from 0-5 with high scores representative of compulsive exercise traits. It is designed to assess four domains of compulsive exercise (compulsivity, affect regulation, weight and shape driven exercise and behavioural rigidity) (17). The CET has not previously been used in competitive athletes but has been validated in a comparable group of young female exercisers (17).

**Dietary behaviours**

Participants completed a food frequency questionnaire (European Prospective Investigation into Cancer; EPIC FFQ) (24, 25) and a four 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 four day food diary was completed by the participants over three week days and one weekend. The FFQ and the 4-day diet diary were coded and entered into a computerized software package (CompEat Pro, version 5.8) to determine energy, macro- and micro-nutrient 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, WI, U.S.A version 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 (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 (26).

**Statistical analysis**

All statistical analyses were performed using SPSS18.0 (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-squared tests were used to determine whether frequencies of categorical variables differed between SF and C groups. Discriminators of stress fracture identified as statistically significant in univariate analyses were entered into a logistic regression to determine their independent contribution to stress fracture/reaction injury. For this analysis, eating psychopathology and compulsive exercise variables were re-coded into two subgroups using a median split. Statistical significance was considered at the 5% probability level ($p < 0.05$).

## Results

70 female athletes (58 runners and 12 triathletes) were recruited, of whom 36% were at national or international standard. A high prevalence of menstrual dysfunction was evident, with 42.9% of athletes categorized as a/oligomenorrhoeic ($\leq$ 9 periods/year) and 57.1% eumenorrhoeic ($\geq$ 10 periods a year). Of the 70 athletes, 21 (30%; 4 a/oligomenorrhoeic and 17 eumenorrhoeic) were currently using hormonal contraceptives (HC).

Nineteen athletes (27%) reported having experienced a total of 24 stress fractures. Common stress fracture sites were the metatarsals (46%), tibia or fibula (38%), calcaneus (13%), and the femur (4%). The mean (SD) age at the first stress fracture was 21.4 (1.4) years, with stress fractures diagnosed on average 3.2 years from the start of training. Age, height, body mass and body fat did not differ between SF and C groups ($P > 0.05$) (Table 1).

Prevalence of current a/oligomenorrhoea and past amenorrhoea were significantly higher in the SF than C group (chi-squared test p=0.008 and 0.035 respectively; Table 1) although age at menarche did not differ significantly between groups. Dominant lower leg lean tissue mass was significantly higher in the SF than C group (p=0.029), and this finding persisted after adjustment for height and weight. Calf circumference measurements, bone measurements, energy, macronutrient intakes and calcium intakes did not differ between groups (Table 1).

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

| **Characteristics** | **Stress Fracture (SF) group (n = 19)** | **Control (C) group (n = 51)** |
|---|---|---|
| Age (yrs) | 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) |
| ***Training*** | | |
| Time since commencing training (yrs) | 7.4 (4.3) | 7.8 (6.4) |
| Age at commencing competition (yrs) | 18.3 (7.0) | 18.4 (8.5) |
| Weekly training duration (hrs/week) | 14.2 (4.3) | 12.1 (4.3) |
| ***Bone Mineral Density (g/cm$^2$)*** | | |
| 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 (mm$^2$) | 154 (17) | 157 (21) |
| Minimal femoral neck width (mm) | 28.9 (2.6) | 28.4 (2.1) |
| Section Modulus (mm$^3$) | 677 (117) | 674 (121) |
| ***Menstrual History*** | | |
| Age at Menarche ** | 13.9 (1.7) | 14.1(2.1) |
| History of amenorrhoea ** | 15 (78.9%) | 26 (51.0%) |
| Current a/oligomenorrhoea** | 13 (68.4%) | 17 (33.3%) |
| Current eumenorrhoea ** | 6 (31.6%) | 34 (66.7%) |
| ***Hormonal Contraception*** | | |
| Current hormonal contraception users (HC) ** | 4 (21.1%) | 17 (33.3%) |
| Current hormonal contraception use in a/oligomenorrhoea (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

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).

**Table 2: Eating disorder examination and compulsive exercise test questionnaire responses according to stress fracture history: median (inter-quartile range).**

| Characteristics | Stress Fracture (SF) 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 $P < 0.05$

The variables that differed according to SF in the univariate analysis, (current a/oligomenorrhoea, lower leg lean tissue mass, EDE-Q scores, and CET scores) were entered into a multiple logistic regression. A/oligomenorrhoea was associated with 4.3 fold greater odds of stress fracture relative to eumenorrhoea (*p=0.011,* Table 3, model A). This finding persisted when current HC was entered into the regression model *(p=0.014, B=1.425)* and when CET, EDE-Q and/or lower leg lean tissue were added to the model (Table 3). CET score and lower leg lean tissue mass, but not EDE-Q scores contributed to stress fracture risk independently of a/oligomenorrhoea. Athletes with CET scores above the median had 5.5 fold greater odds of past stress fracture than those below the median (*p=0.010, B=1.700;* Table 3 models B and D). Each additional kg of lower leg lean mass was associated with 9.7 fold greater odds of fracture (p=0.37, B=2.274) and this persisted with inclusion of other risk factors (Table 3 model E).

**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 (B)** | -1.7 | -4.6 | -2.5 | -4.4 | -11.8 |
| *Cox & Snell* **$R^2$** | 0.095 | 0.190 | 0.107 | 0.191 | 0.253 |
| ***Variables*** | | | | | |
| Current a/oligomenorrhoea [1] | 4.3 (1.4-13.4) ** | 5.8 (1.7-20.3) ** | 4.0 (1.3-12.6) * | 6.2 (1.7-22.5) ** | 6.6 (1.7-26.1) ** |
| Global CET | | 5.5 (1.5-19.8) * | | 6.1 (1.4-26.1) * | 7.4 (1.5-35.7) * |
| Global EDE-Q | | | 1.7 (0.6-5.4) | 0.8 (0.2-3.0) | 0.8 (0.2-3.5) |
| Lower leg lean tissue mass (kg) | | | | | 16.4 (1.5-182.3) * |

Logistic regression with stress fracture history as the dependent variable. * *P< 0.05,* ***P <0.01,*

[1]=Eumenorrhoea as the reference group, CET=Compulsive Exercise Test, EDE-Q= Eating Disorder Examination-Questionnaire.

## Discussion

Eating psychopathology, compulsive exercise, menstrual dysfunction and lower leg lean tissue mass were associated with increased stress fracture risk in endurance athletes. The association of eating psychopathology was not independent of menstrual function, suggesting that the influence of disordered eating on stress fracture is mediated through a/oligomenorrhoea. Compulsion to exercise and lower leg lean tissue mass, as well as menstrual dysfunction, were independently related to stress fracture risk and may play a role in stress fracture aetiology.

In this study, 27% of female athletes had a history of stress fracture. This is similar to the prevalence of 31% in a recent study in female cross-country runners (20), but substantially lower than the 50% in a classic study in endurance athletes (2). 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 stress fracture in more recent studies. In our study, as in a recent study in distance runners (27), the most common site was the metatarsals, whilst in earlier studies more stress fractures occurred at the tibia in distance runners (2, 3, 28) and track and field athletes (2, 3, 29-31), although metatarsal and tarsal stress fractures were more common in sprinters and jumpers (28). Metatarsal SF prevalence is associated with changes in training surface, training volume, footwear and running biomechanics (32), so these factors may explain the differences in stress fracture/reaction locations.

Current menstrual dysfunction was prevalent in this sample of athletes, with 43% of athletes reporting current amenorrhoea or oligomenorrhoea. Both current and past menstrual dysfunction were significantly more frequent in athletes with a history of stress fracture, consistent with earlier findings (2, 10, 11, 33, 34). Furthermore, current menstrual dysfunction was an independent predictor of stress fracture history, with a/oligomenorrhoeic athletes 4.3 times more likely to have a history of stress fracture compared with those who were currently eumenorrhoeic. This is consistent with the 6-fold greater risk of stress fracture in athletes with menstrual dysfunction reported by Bennell et al, (2). The endurance athletes had an average menarcheal age of 14.0 years, compared to 12.3 years in a broader population indicating later menarche occurs in endurance athletes, as has previously been reported in gymnasts (35). Age at menarche was not associated with an increased risk of stress fracture, although associations have been reported in some (2, 13, 36) although not all (11, 20) previous studies. As the results did not identify significant differences in either age at menarche or current BMD, it seems most likely that secondary amenorrhoea rather than primary amenorrhoea or delayed menarche was associated with stress fracture.

Athletes with a history of stress fracture scored higher on the EDE-Q than athletes with no stress fracture history, thus demonstrating a potential relationship with eating psychopathology. This finding supports earlier work which used a less robust measure of eating psychopathology (2). However, unlike previous studies (2) our findings did not show eating psychopathology to be a discriminator of stress fracture history independent of a/oligomenorrhoea. The relationship between eating psychopathology and stress fracture may therefore be mediated through menstrual dysfunction. Future intervention studies would be necessary to confirm whether treatment of disordered eating can reduce stress fracture risk.

A novel finding from the present study is that athletes with high (above median) CET scores had substantially (7.4 times) greater odds of stress fracture, perhaps indicating a greater tendency to train through discomfort. Compulsive exercise predicted stress fracture independently of a/oligomenorrhoea and eating psychopathology. It is therefore important to examine compulsive exercise, as well as menstrual status, when identifying athletes at risk for stress fracture.

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

Factors not significantly related to stress fracture in this study included dietary intakes, bone mineral density, bone geometry and calf circumference. Low dietary calcium intake was a significant predictor of stress fracture in some (20), but not all (13) previous research. Findings may differ depending on measurement tools and differences in dietary intakes, although mean calcium intake was above recommended values in all these studies. Similarly, low BMD was associated with SF risk in some (13, 15, 20) but not all previous studies (2, 10).

Strengths of this study include the assessment of CET. Our findings that compulsive attitudes towards exercise and eating psychopathology are associated with stress fracture risk highlight that those treating and coaching endurance athletes should consider attitudes to exercise and eating behaviours when assessing stress fracture risk. Future research should examine whether interventions that address these features can reduce stress fracture risk. The retrospective nature of this study incurs some limitations, such as potential recruitment bias, self reporting of history of stress fracture and the ability to recall data. However, the occurrence of a stress fracture during an athlete's career is usually event of such significance that it is unlikely to be misreported. Athletes were measured at variable intervals following stress fracture during which time their behaviours, and other risk factors, may have changed. Differences in stress fracture 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 increased risk of stress fracture, but this may be mediated through menstrual dysfunction. Compulsive exercise cognitions, as well as amenorrhoea, were independently associated with stress fracture risk and may have a role in stress fracture aetiology.

**Conflicts of Interest**

There are no perceived conflicts of interest associated with the research.

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**Contributorship Statement**

Contributors: RLD and KBW were responsible for the study design, data collection, statistical analysis, interpretation, reporting, and manuscript preparation and are the guarantors. NP, CM, GS and NC contributed to the study design, interpretation, and manuscript preparation.

**Data Sharing Statement**

There is no additional data available

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

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

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## Article Summary

### Article Focus

1. To identify the factors related to stress fracture in female endurance athletes,

2. To evaluate associations of eating and exercise behaviours with stress fracture and to determine whether associations are independent of previously recognised risk factors.

### Key Points

1. Compulsive exercise and lower leg lean tissue mass, as well as amenorrhoea, are independently related to stress fracture risk

2. Eating disorder psychopathology was associated with increased risk of stress fracture in endurance athletes, but this may be mediated by menstrual dysfunction and compulsive exercise.

### Strengths and Limitations

1. Strength; the assessment of the Compulsive Exercise test (CET) and a more robust measure of eating psychopathology determined that compulsive exercise and eating psychopathology are associated with stress fracture risk.

2. Limitation, potential recruitment bias, self-reporting of history of stress fracture and the ability to recall data.

3. Limitation; measurements at variable intervals following stress fracture during which time athlete behaviours, and other risk factors, may have changed.

**Abstract**

**Objective:** To identify psychological and physiological correlates of stress fracture in female endurance athletes.

**Design:** A cross-sectional design was used with 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 chi-squared, ANOVA and Mann Whitney U tests.

**Results:** Nineteen (27%) athletes had previously been clinically diagnosed with stress fractures. The prevalence of current a/oligomenorrhoea 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 stress fracture history ($p=0.006$, 0.009 and 0.035 respectively).

**Conclusion:** Eating psychopathology was associated with increased risk of stress fracture in endurance athletes, but this may be mediated by menstrual dysfunction and compulsive exercise. Compulsive exercise, as well as amenorrhoea, are independently related to stress fracture risk.

## Introduction

Stress fractures occur when microdamage caused by repetitive mechanical load exceeds the biological capacity of the bone (1). Stress fractures are the most common overuse injury in athletes, interrupting training and prematurely ending sporting careers (2). Over 40% of athletes report a history of stress fracture according to some previous research, although prevalence differs markedly between studies (3-5).

Risk factors for stress fracture 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 stress fracture (6-9). Whilst these studies have improved our understanding of the aetiology of stress fracture, these risk factors may not translate well to athletes, who are accustomed to training and have high levels of fitness (2). In female athletes, previously identified risk factors for stress fracture include small calf circumference, late age at menarche, menstrual dysfunction (both past and current), low bone density, current disordered eating, and abnormal gait (2, 5, 10-15). However, findings have often been inconsistent (e.g. low bone density is associated with increased risk in some studies but not others) (5).

Female endurance athletes have particularly high prevalence of the female athlete triad, which involves menstrual dysfunction, low bone mineral density and dietary restriction/energy deprivation (16). All of these may be risk factors for stress fractures (12, 13, 15) but previous studies on their role in athletic populations have had small sample sizes and yielded conflicting findings (5, 17). Increased awareness of the female athlete triad in recent decades may have affected management of the female athlete, perhaps to the extent that the associations reported in older studies may no longer persist. The current high prevalence (1-

62%) (18) of disordered eating among female athletes could further increase stress fracture risk, as restrictive eating patterns were associated with increased risk in some of the previous studies (2, 19, 20). Furthermore, restrictive eating patterns may be accompanied by compulsive exercise behaviours (21, 22), but the role of compulsive exercise has not been examined. Eating behaviours may have an independent effect on stress fracture risk, or this may be mediated through related exercise behaviours, menstrual dysfunction or low bone density. It is important to determine whether eating behaviours have an independent effect as this would demonstrate that resolving menstrual dysfunction and low bone density would not entirely resolve the increased risk of stress fracture.

There is thus a need for further research on the role of eating and exercise behaviours in stress fracture risk, using multivariate models to determine whether risk factors act independently. Therefore, this study aimed to identify the factors related to stress fracture in female endurance athletes, to evaluate associations of eating and exercise behaviours with stress fracture and to determine whether associations are independent of previously recognised risk factors.

**Methods**

**Study design**

A cross-sectional design was used with history of stress fractures and potential risk factors assessed at one visit. Questionnaires were completed to assess stress fracture history, eating psychopathology, dietary intakes, menstrual dysfunction, contraceptive use and training history. A four day diet diary was provided for subsequent completion and return. Bone mineral density (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 (NRES) 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 either, be competing at international, national, or county level and training approximately 8-10 hours per week (runners) or 15-20 hours per week (triathletes) in events from 800m to the marathon or triathlon. Athletes were excluded from the study if they had not currently trained in the last six months and if they were currently pregnant or lactating or had been in the last 12-months.

**Measurements**

**Injury history**

History of stress fracture was assessed by 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 stress fracture, athletes recorded the age when the stress fracture occurred, the anatomical location, time of year, and method of diagnosis.

**Menstrual function**

Participants were classified as secondary amenorrhoeic (less than 4 menses per year) oligomenorrhoeic (4-9 periods per 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 (23). For analysis purposes athletes who were classified with menstrual dysfunction were combined into one group (a/oligomenorrhoea, 0-9 periods per year) (24-26) 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 questionnaire.

**Eating psychopathology and compulsive exercise cognitions**

Athletes completed the Eating Disorder Examination Questionnaire (EDE-Q) (27) and the Compulsive Exercise Test (CET) (17). The EDE-Q is a self reported version of the Eating Disorder Examination (EDE), the current gold standard for measuring eating disordered psychopathology (27). The EDE-Q is a 36-item questionnaire comprising four subscales (restraint, eating concern, shape concern, weight concern), a global score and a specific section of diagnostic questions related to bingeing/purging. Each item on the questionnaire is scored from 0-6 with high scores representative of concerns with eating behaviours and attitudes. The CET is a 24-item self reported questionnaire scored from 0-5 with high scores representative of compulsive exercise traits. It is designed to assess four domains of compulsive exercise (compulsivity, affect regulation, weight and shape driven exercise and behavioural rigidity) (17). The CET has not previously been used in competitive athletes but has been validated in a comparable group of young female exercisers (17).

**Dietary behaviours**

Participants completed a food frequency questionnaire (European Prospective Investigation into Cancer; EPIC FFQ) (28, 29) and a four 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 four day food diary was completed by the participants over three week days and one weekend. The FFQ and the 4-day diet diary were coded and entered into a computerized software package (CompEat Pro, version 5.8) to determine energy, macro- and micro-nutrient 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, WI, U.S.A version 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 (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 (30).

**Statistical analysis**

All statistical analyses were performed using SPSS18.0 (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 stress fracture (SF) and a control (C) group. Potential risk factors were compared between groups using analysis of variance (ANOVA) and Mann-Whitney U-Tests. Chi-squared tests were used to determine whether frequencies of categorical variables differed between SF and C groups. Discriminators of stress fracture 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 stress fracture/reaction injury. The first model (model A), examined the contribution of a/oligomenorrhoea. Subsequent models (Model B, C, and D) examined the contributions of CET and EDE-Q independent of a/oligomenorrhoea and/or each other. The final model (Model E) includes all potential discriminators of stress fracture to determine which are independent of each other. For this analysis, eating psychopathology and compulsive exercise variables were re-coded into two subgroups using a median split. Statistical significance was considered at the 5% probability level ($p < 0.05$).

## Results

70 female athletes (58 runners and 12 triathletes) were recruited, of whom 36% were at national or international standard. A high prevalence of menstrual dysfunction was evident, with 42.9% of athletes categorized as a/oligomenorrhoeic ($\leq$ 9 periods/year) and 57.1% eumenorrhoeic ($\geq$ 10 periods a year). Of the 70 athletes, 21 (30%; 4 a/oligomenorrhoeic and 17 eumenorrhoeic) were currently using hormonal contraceptives (HC).

Nineteen athletes (27%; 24% at the national/international level and 29% at the sub-elite level) reported having experienced a total of 24 stress fractures. Common stress fracture sites were the metatarsals (46%), tibia or fibula (38%), calcaneus (13%) and the femur (4%). The mean (SD) age at the first stress fracture was 21.4 (1.4) years, with stress fractures diagnosed at a median (IQR) of 3.0 (6.8) years from the start of training. Age, height, body mass and body fat did not differ between SF and C groups ($P > 0.05$; Table 1).

Prevalence of current a/oligomenorrhoea and past amenorrhoea were significantly higher in the SF than C group (chi-squared 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 C group (p=0.029); this finding persisted after adjustment for weight and training duration (hrs/week) (p=0.035). Calf circumference measurements, bone measurements, energy, macronutrient intakes and calcium intakes did not differ between groups (Table 1).

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

| Characteristics | Stress Fracture (SF) group (n = 19) | Control (C) group (n = 51) |
|---|---|---|
| Age (yrs) | 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 (yrs) (median (IQR) | 2.0 (3.0) | - |
| ***Training*** | | |
| Time since commencing training (yrs) | 7.4 (4.3) | 7.8 (6.4) |
| Age at commencing competition (yrs) | 18.3 (7.0) | 18.4 (8.5) |
| Weekly training duration (hrs/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 ($mm^2$) | 154 (17) | 157 (21) |
| Minimal femoral neck width (mm) | 28.9 (2.6) | 28.4 (2.1) |
| Section Modulus ($mm^3$) | 677 (117) | 674 (121) |
| ***Menstrual History*** | | |
| Age at Menarche (years) | 13.9 (1.7) | 14.1 (2.1) |
| History of amenorrhoea ** | 15 (78.9%) | 26 (51.0%) |
| Current a/oligomenorrhoea** | 13 (68.4%) | 17 (33.3%) |
| Current eumenorrhoea ** | 6 (31.6%) | 34 (66.7%) |
| ***Hormonal Contraception*** | | |
| Current hormonal contraception users (HC) ** | 4 (21.1%) | 17 (33.3%) |
| Current hormonal contraception use in a/oligomenorrhoea (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

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 sub-elite level.

**Table 2: Eating disorder examination and compulsive exercise test questionnaire responses according to stress fracture history: median (inter-quartile range).**

| Characteristics | Stress Fracture (SF) 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 $P < 0.05$

The variables that differed according to SF in the univariate analysis, (current a/oligomenorrhoea, lower leg lean tissue mass, EDE-Q scores, and CET scores) were entered into a multiple logistic regression adjusted for weight (kg) and training duration (hrs). A/oligomenorrhoea was associated with 4.7 fold greater odds of stress fracture relative to eumenorrhoea (*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 stress fracture risk independently of a/oligomenorrhoea. Athletes with CET scores above the median had 7.1 fold greater odds of past stress fracture 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.

**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 (B)** | -3.9 | -7.6 | -4.7 | -7.6 | -3.2 |
| *Cox & Snell* $\mathbf{R^2}$ | 0.137 | 0.245 | 0.149 | 0.248 | 0.253 |
| ***Variables*** | | | | | |
| Current a/oligomenorrhoea [1] | 4.7 (1.5-15.0) ** | 7.1 (1.9-27.1) ** | 4.5 (1.4-14.6) * | 6.6 (1.9-31.1) ** | 8.6 (1.9-38.2) ** |
| Global CET (>median to <median) | | 7.1 (1.8-28.4) ** | | 8.8 (1.7-44.2) ** | 10.2 (1.7-36.4) * |
| Global EDE-Q (>median to <median) | | | 1.8 (0.5-5.7) | 0.7 (0.2-2.8) | 0.7 (0.1-3.5) |
| Lower leg lean tissue mass (SD) | | | | | 2.3 (1.1-5.0)* |

Logistic regression with stress fracture history as the dependent variable. * *P< 0.05, **P <0.01,*
[1]=Eumenorrhoea as the reference group, CET=Compulsive Exercise Test, EDE-Q= Eating Disorder Examination-Questionnaire. The global CET and EDEQ variables represent a comparison with a median split. Models are adjusted for weight and training duration.

## Discussion

Eating psychopathology, compulsive exercise, menstrual dysfunction and lower leg lean tissue mass were associated with increased stress fracture risk in endurance athletes. The association of eating psychopathology was not independent of menstrual function, suggesting that the influence of disordered eating on stress fracture is mediated through a/oligomenorrhoea. Compulsion to exercise and lower leg lean tissue mass, as well as menstrual dysfunction, were independently related to stress fracture risk and may play a role in stress fracture aetiology.

In this study, 27% of female athletes had a history of stress fracture. This is similar to the prevalence of 31% in a recent study in female cross-country runners (20), but substantially lower than the 50% in a classic study in endurance athletes (2). 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 stress fracture in more recent studies. In our study, as in a recent study in distance runners (31), the most common site was the metatarsals, whilst in earlier studies more stress fractures occurred at the tibia in distance runners (2, 3, 32) and track and field athletes (2, 3, 26, 33, 34), although metatarsal and tarsal stress fractures were more common in sprinters and jumpers (32). Metatarsal SF prevalence is associated with changes in training surface, training volume, footwear and running biomechanics (35), so these factors may contribute to differences in stress fracture/reaction locations.

Current menstrual dysfunction was prevalent in this sample of athletes, with 43% of athletes reporting current amenorrhoea or oligomenorrhoea. Both current and past menstrual dysfunction were significantly more frequent in athletes with a history of stress fracture, consistent with earlier findings (2, 10, 11, 36, 37). Furthermore, current menstrual dysfunction was an independent predictor of stress fracture history, with a/oligomenorrhoeic athletes having 4.3 times greater odds of stress fracture compared with those who were currently eumenorrhoeic. This is consistent with the 6-fold greater odds of stress fracture in athletes with menstrual dysfunction reported by Bennell et al, (2). The endurance athletes had an average menarcheal age of 14.0 years, compared to 12.3 years in a broader population indicating later menarche occurs in endurance athletes, as has previously been reported in gymnasts (38). Age at menarche was not associated with an increased risk of stress fracture, although associations have been reported in some (2, 13, 39) although not all (11, 20) previous studies. As the results did not identify significant differences in either age at menarche or current BMD, it seems most likely that secondary amenorrhoea rather than primary amenorrhoea or delayed menarche was associated with stress fracture.

Interestingly, although athletes with stress fracture had a higher prevalence of amenorrhoea, they had a lower prevalence of hormonal contraceptive use, although this difference did not reach statistical significance. It is possible that amenorrhoea not countered by HC use is associated with greater stress fracture risk; furthermore amenorrhoeic 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 stress fracture influences HC prescribing strategies. Given the increased awareness of the female athlete triad, clinical physicians presented with an amenorrhoeic athlete with a stress fracture may attempt to determine the cause of, and resolve, the amenorrhoea rather than prescribing HC, although past research has shown a trend towards a reduction in stress fracture when HC was prescribed (11, 26, 40). Further studies of the role of HC use in stress fracture is needed.

Athletes with a history of stress fracture scored higher on the EDE-Q than athletes with no stress fracture history, thus demonstrating a potential relationship with eating psychopathology. This finding supports earlier work which used a less robust measure of eating psychopathology (2). However, unlike previous studies (2) our findings did not show eating psychopathology to be a discriminator of stress fracture history independent of a/oligomenorrhoea. The relationship between eating psychopathology and stress fracture may therefore be mediated through menstrual dysfunction. Future intervention studies would be necessary to confirm whether treatment of disordered eating can reduce stress fracture 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 stress fracture, perhaps indicating a greater tendency to train through discomfort. Compulsive exercise predicted stress fracture independently of a/oligomenorrhoea and eating psychopathology. It is therefore important to examine compulsive exercise, as well as menstrual status, when identifying athletes at risk for stress fracture.

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

Factors not significantly related to stress fracture in this study included dietary intakes, bone mineral density, bone geometry and calf circumference. Low dietary calcium intake was a significant predictor of stress fracture in some (20), but not all (13) previous research.

Findings may differ depending on measurement tools and differences in dietary intakes, although mean calcium intake was above recommended values in all these studies. Similarly, low BMD was associated with SF risk in some (13, 15, 20) but not all previous studies (2, 10).

Strengths of this study include the assessment of CET. Our findings that compulsive attitudes towards exercise and eating psychopathology are associated with stress fracture risk highlight that those treating and coaching endurance athletes should consider attitudes to exercise and eating behaviours when assessing stress fracture risk. Future research should examine whether interventions that address these features can reduce stress fracture 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 fractures are a cause or effect of the stress fracture 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 stress fractures is relatively high, odds ratios may be substantially greater than relative risks. Further limitations include potential recruitment bias, self-reporting of history of stress fracture and the ability to recall data. However, the occurrence of a stress fracture 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 stress fracture during which time their behaviours, and other risk factors, may have changed. Differences in stress fracture 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 increased risk of stress fracture, but this may be mediated through menstrual dysfunction. Compulsive exercise cognitions, as well as amenorrhoea, were independently associated with stress fracture risk and may have a role in stress fracture aetiology.

**CONFLICT OF INTERESTS**
There are no perceived conflicts of interest associated with the research.

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Contributors: RLD and KBW were responsible for the study design, data collection, statistical analysis, interpretation, reporting, and manuscript preparation and are the guarantors. NP, CM, GS and NC contributed to the study design, interpretation, and manuscript preparation.

**DATA SHARING**
There is no additional data available

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

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## Article Summary

### Article Focus

1. To identify the factors related to stress fracture in female endurance athletes,

2. To evaluate associations of eating and exercise behaviours with stress fracture and to determine whether associations are independent of previously recognised risk factors.

### Key Points

1. Compulsive exercise and lower leg lean tissue mass, as well as amenorrhoea, are independently related to stress fracture risk

2. Eating disorder psychopathology was associated with increased risk of stress fracture in endurance athletes, but this may be mediated by menstrual dysfunction and compulsive exercise.

### Strengths and Limitations

1. Strength; the assessment of the Compulsive Exercise test (CET) and a more robust measure of eating psychopathology determined that compulsive exercise and eating psychopathology are associated with stress fracture risk.

2. Limitation, potential recruitment bias, self-reporting of history of stress fracture and the ability to recall data.

3. Limitation; measurements at variable intervals following stress fracture during which time athlete behaviours, and other risk factors, may have changed.

## Abstract

**Objective:** To identify psychological and physiological correlates of stress fracture in female endurance athletes.

**Design:** A cross-sectional design was used with 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 chi-squared, ANOVA and Mann Whitney U tests.

**Results:** Nineteen (27%) athletes had previously been clinically diagnosed with stress fractures. The prevalence of current a/oligomenorrhoea 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 stress fracture history (p=0.006, 0.009 and 0.035 respectively).

**Conclusion:** Eating psychopathology was associated with increased risk of stress fracture in endurance athletes, but this may be mediated by menstrual dysfunction and compulsive exercise. Compulsive exercise, as well as amenorrhoea, are independently related to stress fracture risk.

## Introduction

Stress fractures occur when microdamage caused by repetitive mechanical load exceeds the biological capacity of the bone (1). Stress fractures are the most common overuse injury in athletes, interrupting training and prematurely ending sporting careers (2). Over 40% of athletes report a history of stress fracture according to some previous research, although prevalence differs markedly between studies (3-5).

Risk factors for stress fracture 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 stress fracture (6-9). Whilst these studies have improved our understanding of the aetiology of stress fracture, these risk factors may not translate well to athletes, who are accustomed to training and have high levels of fitness (2). In female athletes, previously identified risk factors for stress fracture include small calf circumference, late age at menarche, menstrual dysfunction (both past and current), low bone density, current disordered eating, and abnormal gait (2, 5, 10-15). However, findings have often been inconsistent (e.g. low bone density is associated with increased risk in some studies but not others) (5).

Female endurance athletes have particularly high prevalence of the female athlete triad, which involves menstrual dysfunction, low bone mineral density and dietary restriction/energy deprivation (16). All of these may be risk factors for stress fractures (12, 13, 15) but previous studies on their role in athletic populations have had small sample sizes and yielded conflicting findings (5, 17). Increased awareness of the female athlete triad in recent decades may have affected management of the female athlete, perhaps to the extent that the associations reported in older studies may no longer persist. The current high prevalence (1-

62%) (18) of disordered eating among female athletes could further increase stress fracture risk, as restrictive eating patterns were associated with increased risk in some of the previous studies (2, 19, 20). Furthermore, restrictive eating patterns may be accompanied by compulsive exercise behaviours (21, 22), but the role of compulsive exercise has not been examined. Eating behaviours may have an independent effect on stress fracture risk, or this may be mediated through related exercise behaviours, menstrual dysfunction or low bone density. It is important to determine whether eating behaviours have an independent effect as this would demonstrate that resolving menstrual dysfunction and low bone density would not entirely resolve the increased risk of stress fracture.

There is thus a need for further research on the role of eating and exercise behaviours in stress fracture risk, using multivariate models to determine whether risk factors act independently. Therefore, this study aimed to identify the factors related to stress fracture in female endurance athletes, to evaluate associations of eating and exercise behaviours with stress fracture and to determine whether associations are independent of previously recognised risk factors.

## Methods

### Study design

A cross-sectional design was used with history of stress fractures and potential risk factors assessed at one visit. Questionnaires were completed to assess stress fracture history, eating psychopathology, dietary intakes, menstrual dysfunction, contraceptive use and training history. A four day diet diary was provided for subsequent completion and return. Bone mineral density (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 (NRES) 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 either, be competing at international, national, or county level and training approximately 8-10 hours per week (runners) or 15-20 hours per week (triathletes) in events from 800m to the marathon or triathlon. Athletes were excluded from the study if they had not currently trained in the last six months and if they were currently pregnant or lactating or had been in the last 12-months.

**Measurements**

**Injury history**

History of stress fracture was assessed by 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 stress fracture, athletes recorded the age when the stress fracture occurred, the anatomical location, time of year, and method of diagnosis.

**Menstrual function**

Participants were classified as secondary amenorrhoeic (less than 4 menses per year) oligomenorrhoeic (4-9 periods per 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 (23). For analysis purposes athletes who were classified with menstrual dysfunction were combined into one group (a/oligomenorrhoea, 0-9 periods per year) (24-26) 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 questionnaire.

**Eating psychopathology and compulsive exercise cognitions**

Athletes completed the Eating Disorder Examination Questionnaire (EDE-Q) (27) and the Compulsive Exercise Test (CET) (17). The EDE-Q is a self reported version of the Eating Disorder Examination (EDE), the current gold standard for measuring eating disordered psychopathology (27). The EDE-Q is a 36-item questionnaire comprising four subscales (restraint, eating concern, shape concern, weight concern), a global score and a specific section of diagnostic questions related to bingeing/purging. Each item on the questionnaire is scored from 0-6 with high scores representative of concerns with eating behaviours and attitudes. The CET is a 24-item self reported questionnaire scored from 0-5 with high scores representative of compulsive exercise traits. It is designed to assess four domains of compulsive exercise (compulsivity, affect regulation, weight and shape driven exercise and behavioural rigidity) (17). The CET has not previously been used in competitive athletes but has been validated in a comparable group of young female exercisers (17).

**Dietary behaviours**

Participants completed a food frequency questionnaire (European Prospective Investigation into Cancer; EPIC FFQ) (28, 29) and a four 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 four day food diary was completed by the participants over three week days and one weekend. The FFQ and the 4-day diet diary were coded and entered into a computerized software package (CompEat Pro, version 5.8) to determine energy, macro- and micro-nutrient 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, WI, U.S.A version 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 (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 (30).

### Statistical analysis

All statistical analyses were performed using SPSS18.0 (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 stress fracture (SF) and a control (C) group. Potential risk factors were compared between groups using analysis of variance (ANOVA) and Mann-Whitney U-Tests. Chi-squared tests were used to determine whether frequencies of categorical variables differed between SF and C groups. Discriminators of stress fracture 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 stress fracture/reaction injury. The first model (model A), examined the contribution of a/oligomenorrhoea. Subsequent models (Model B, C, and D) examined the contributions of CET and EDE-Q independent of a/oligomenorrhoea and/or each other. The final model (Model E) includes all potential discriminators of stress fracture to determine which are independent of each other. For this analysis, eating psychopathology and compulsive exercise variables were re-coded into two subgroups using a median split. Statistical significance was considered at the 5% probability level ($p < 0.05$).

**Results**

70 female athletes (58 runners and 12 triathletes) were recruited, of whom 36% were at national or international standard. A high prevalence of menstrual dysfunction was evident, with 42.9% of athletes categorized as a/oligomenorrhoeic ($\leq$ 9 periods/year) and 57.1% eumenorrhoeic ($\geq$ 10 periods a year). Of the 70 athletes, 21 (30%; 4 a/oligomenorrhoeic and 17 eumenorrhoeic) were currently using hormonal contraceptives (HC).

Nineteen athletes (27%; 24% at the national/international level and 29% at the sub-elite level) reported having experienced a total of 24 stress fractures. Common stress fracture sites were the metatarsals (46%), tibia or fibula (38%), calcaneus (13%) and the femur (4%). The mean (SD) age at the first stress fracture was 21.4 (1.4) years, with stress fractures diagnosed at a median (IQR) of 3.0 (6.8) years from the start of training. Age, height, body mass and body fat did not differ between SF and C groups ($P > 0.05$; Table 1).

Prevalence of current a/oligomenorrhoea and past amenorrhoea were significantly higher in the SF than C group (chi-squared 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 C group (p=0.029); this finding persisted after adjustment for weight and training duration (hrs/week) (p=0.035). Calf circumference measurements, bone measurements, energy, macronutrient intakes and calcium intakes did not differ between groups (Table 1).

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

| **Characteristics** | **Stress Fracture (SF) group (n = 19)** | **Control (C) group (n = 51)** |
|---|---|---|
| Age (yrs) | 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 (yrs) (median (IQR) | 2.0 (3.0) | - |
| ***Training*** | | |
| Time since commencing training (yrs) | 7.4 (4.3) | 7.8 (6.4) |
| Age at commencing competition (yrs) | 18.3 (7.0) | 18.4 (8.5) |
| Weekly training duration (hrs/week) | 14.2 (4.3) | 12.1 (4.3) |
| ***Bone Mineral Density ($g/cm^2$)*** | | |
| 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 ($mm^2$) | 154 (17) | 157 (21) |
| Minimal femoral neck width (mm) | 28.9 (2.6) | 28.4 (2.1) |
| Section Modulus ($mm^3$) | 677 (117) | 674 (121) |
| ***Menstrual History*** | | |
| Age at Menarche (years) | 13.9 (1.7) | 14.1 (2.1) |
| History of amenorrhoea ** | 15 (78.9%) | 26 (51.0%) |
| Current a/oligomenorrhoea** | 13 (68.4%) | 17 (33.3%) |
| Current eumenorrhoea ** | 6 (31.6%) | 34 (66.7%) |
| ***Hormonal Contraception*** | | |
| Current hormonal contraception users (HC) ** | 4 (21.1%) | 17 (33.3%) |
| Current hormonal contraception use in a/oligomenorrhoea (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

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 sub-elite level.

**Table 2: Eating disorder examination and compulsive exercise test questionnaire responses according to stress fracture history: median (inter-quartile range).**

| Characteristics | Stress Fracture (SF) 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 $P < 0.05$

The variables that differed according to SF in the univariate analysis, (current a/oligomenorrhoea, lower leg lean tissue mass, EDE-Q scores, and CET scores) were entered into a multiple logistic regression adjusted for weight (kg) and training duration (hrs). A/oligomenorrhoea was associated with 4.7 fold greater odds of stress fracture relative to eumenorrhoea (*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 stress fracture risk independently of a/oligomenorrhoea. Athletes with CET scores above the median had 7.1 fold greater odds of past stress fracture 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.

**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 (B)** | -3.9 | -7.6 | -4.7 | -7.6 | -3.2 |
| *Cox & Snell* $\mathbf{R^2}$ | 0.137 | 0.245 | 0.149 | 0.248 | 0.253 |
| ***Variables*** | | | | | |
| Current a/oligomenorrhoea [1] | 4.7 (1.5-15.0) ** | 7.1 (1.9-27.1) ** | 4.5 (1.4-14.6) * | 6.6 (1.9-31.1) ** | 8.6 (1.9-38.2) ** |
| Global CET (>median to <median) | | 7.1 (1.8-28.4) ** | | 8.8 (1.7-44.2) ** | 10.2 (1.7-36.4) * |
| Global EDE-Q (>median to <median) | | | 1.8 (0.5-5.7) | 0.7 (0.2-2.8) | 0.7 (0.1-3.5) |
| Lower leg lean tissue mass (SD) | | | | | 2.3 (1.1-5.0)* |

Logistic regression with stress fracture history as the dependent variable. * *P< 0.05,* ***P <0.01,*

[1]=Eumenorrhoea as the reference group, CET=Compulsive Exercise Test, EDE-Q= Eating Disorder Examination-Questionnaire. The global CET and EDEQ variables represent a comparison with a median split. Models are adjusted for weight and training duration.

## Discussion

Eating psychopathology, compulsive exercise, menstrual dysfunction and lower leg lean tissue mass were associated with increased stress fracture risk in endurance athletes. The association of eating psychopathology was not independent of menstrual function, suggesting that the influence of disordered eating on stress fracture is mediated through a/oligomenorrhoea. Compulsion to exercise and lower leg lean tissue mass, as well as menstrual dysfunction, were independently related to stress fracture risk and may play a role in stress fracture aetiology.

In this study, 27% of female athletes had a history of stress fracture. This is similar to the prevalence of 31% in a recent study in female cross-country runners (20), but substantially lower than the 50% in a classic study in endurance athletes (2). 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 stress fracture in more recent studies. In our study, as in a recent study in distance runners (31), the most common site was the metatarsals, whilst in earlier studies more stress fractures occurred at the tibia in distance runners (2, 3, 32) and track and field athletes (2, 3, 26, 33, 34), although metatarsal and tarsal stress fractures were more common in sprinters and jumpers (32). Metatarsal SF prevalence is associated with changes in training surface, training volume, footwear and running biomechanics (35), so these factors may contribute to differences in stress fracture/reaction locations.

Current menstrual dysfunction was prevalent in this sample of athletes, with 43% of athletes reporting current amenorrhoea or oligomenorrhoea. Both current and past menstrual dysfunction were significantly more frequent in athletes with a history of stress fracture, consistent with earlier findings (2, 10, 11, 36, 37). Furthermore, current menstrual dysfunction was an independent predictor of stress fracture history, with a/oligomenorrhoeic athletes having 4.3 times greater odds of stress fracture compared with those who were currently eumenorrhoeic. This is consistent with the 6-fold greater odds of stress fracture in athletes with menstrual dysfunction reported by Bennell et al, (2). The endurance athletes had an average menarcheal age of 14.0 years, compared to 12.3 years in a broader population indicating later menarche occurs in endurance athletes, as has previously been reported in gymnasts (38). Age at menarche was not associated with an increased risk of stress fracture, although associations have been reported in some (2, 13, 39) although not all (11, 20) previous studies. As the results did not identify significant differences in either age at menarche or current BMD, it seems most likely that secondary amenorrhoea rather than primary amenorrhoea or delayed menarche was associated with stress fracture.

Interestingly, although athletes with stress fracture had a higher prevalence of amenorrhoea, they had a lower prevalence of hormonal contraceptive use, although this difference did not reach statistical significance. It is possible that amenorrhoea not countered by HC use is associated with greater stress fracture risk; furthermore amenorrhoeic 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 stress fracture influences HC prescribing strategies. Given the increased awareness of the female athlete triad, clinical physicians presented with an amenorrhoeic athlete with a stress fracture may attempt to determine the cause of, and resolve, the amenorrhoea rather than prescribing HC, although past research has shown a trend towards a reduction in stress fracture when HC was prescribed (11, 26, 40). Further studies of the role of HC use in stress fracture is needed.

Athletes with a history of stress fracture scored higher on the EDE-Q than athletes with no stress fracture history, thus demonstrating a potential relationship with eating psychopathology. This finding supports earlier work which used a less robust measure of eating psychopathology (2). However, unlike previous studies (2) our findings did not show eating psychopathology to be a discriminator of stress fracture history independent of a/oligomenorrhoea. The relationship between eating psychopathology and stress fracture may therefore be mediated through menstrual dysfunction. Future intervention studies would be necessary to confirm whether treatment of disordered eating can reduce stress fracture 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 stress fracture, perhaps indicating a greater tendency to train through discomfort. Compulsive exercise predicted stress fracture independently of a/oligomenorrhoea and eating psychopathology. It is therefore important to examine compulsive exercise, as well as menstrual status, when identifying athletes at risk for stress fracture.

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

Factors not significantly related to stress fracture in this study included dietary intakes, bone mineral density, bone geometry and calf circumference. Low dietary calcium intake was a significant predictor of stress fracture in some (20), but not all (13) previous research.

Findings may differ depending on measurement tools and differences in dietary intakes, although mean calcium intake was above recommended values in all these studies. Similarly, low BMD was associated with SF risk in some (13, 15, 20) but not all previous studies (2, 10).

Strengths of this study include the assessment of CET. Our findings that compulsive attitudes towards exercise and eating psychopathology are associated with stress fracture risk highlight that those treating and coaching endurance athletes should consider attitudes to exercise and eating behaviours when assessing stress fracture risk. Future research should examine whether interventions that address these features can reduce stress fracture 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 fractures are a cause or effect of the stress fracture 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 stress fractures is relatively high, odds ratios may be substantially greater than relative risks. Further limitations include potential recruitment bias, self-reporting of history of stress fracture and the ability to recall data. However, the occurrence of a stress fracture 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 stress fracture during which time their behaviours, and other risk factors, may have changed. Differences in stress fracture 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 increased risk of stress fracture, but this may be mediated through menstrual dysfunction. Compulsive exercise cognitions, as well as amenorrhoea, were independently associated with stress fracture risk and may have a role in stress fracture aetiology.

**Conflicts of Interest**

There are no perceived conflicts of interest associated with the research.

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**Instructions**: Listed below are a series of statements regarding exercise. Please read each statement carefully and circle the number that best indicates how true each statement is of you. Please answer all the questions as honestly as you can.

| Never true | Rarely true | Sometimes true | Often true | Usually true | Always true |
|---|---|---|---|---|---|
| **0** | **1** | **2** | **3** | **4** | **5** |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 1) | I feel happier and/or more positive after I exercise. | 0 | 1 | 2 | 3 | 4 | 5 |
| 2) | I exercise to improve my appearance. | 0 | 1 | 2 | 3 | 4 | 5 |
| 3) | I like my days to be organised and structured of which exercise is just one part. | 0 | 1 | 2 | 3 | 4 | 5 |
| 4) | I feel less anxious after I exercise. | 0 | 1 | 2 | 3 | 4 | 5 |
| 5) | I find exercise a chore. | 0 | 1 | 2 | 3 | 4 | 5 |
| 6) | If I feel I have eaten too much, I will do more exercise. | 0 | 1 | 2 | 3 | 4 | 5 |
| 7) | My weekly pattern of exercise is repetitive. | 0 | 1 | 2 | 3 | 4 | 5 |
| 8) | I do not exercise to be slim. | 0 | 1 | 2 | 3 | 4 | 5 |
| 9) | If I cannot exercise I feel low or depressed. | 0 | 1 | 2 | 3 | 4 | 5 |
| 10) | I feel extremely guilty if I miss an exercise session. | 0 | 1 | 2 | 3 | 4 | 5 |
| 11) | I usually continue to exercise despite injury or illness, unless I am very ill or too injured. | 0 | 1 | 2 | 3 | 4 | 5 |
| 12) | I enjoy exercising. | 0 | 1 | 2 | 3 | 4 | 5 |
| 13) | I exercise to burn calories and lose weight. | 0 | 1 | 2 | 3 | 4 | 5 |
| 14) | I feel less stressed and/or tense after I exercise. | 0 | 1 | 2 | 3 | 4 | 5 |
| 15) | If I miss an exercise session, I will try and make up for it when I next exercise. | 0 | 1 | 2 | 3 | 4 | 5 |
| 16) | If I cannot exercise I feel agitated and/or irritable. | 0 | 1 | 2 | 3 | 4 | 5 |
| 17) | Exercise improves my mood. | 0 | 1 | 2 | 3 | 4 | 5 |
| 18) | If I cannot exercise, I worry that I will gain weight. | 0 | 1 | 2 | 3 | 4 | 5 |
| 19) | I follow a set routine for my exercise sessions e.g. walk or run the same route, particular exercises, same amount of time, and so on. | 0 | 1 | 2 | 3 | 4 | 5 |
| 20) | If I cannot exercise I feel angry and/or frustrated. | 0 | 1 | 2 | 3 | 4 | 5 |
| 21) | I do not enjoy exercising. | 0 | 1 | 2 | 3 | 4 | 5 |
| 22) | I feel like I've let myself down if I miss an exercise session. | 0 | 1 | 2 | 3 | 4 | 5 |
| 23) | If I cannot exercise I feel anxious. | 0 | 1 | 2 | 3 | 4 | 5 |
| 24) | I feel less depressed or low after I exercise. | 0 | 1 | 2 | 3 | 4 | 5 |